20 Lecomte J, Buyses H, Taymans J, *et al*. Treatment of tendinitis and bursitis: a comparison of nimesulide and naproxen sodium in a double blind parallel trial. *Eur J Rheumatol Inflamm* 1994;**14**:29–32.
21 Dreiser RL, Ditisheim A, Charlot J, *et al*. A double blind, placebo controlled study of niflumic acid gel in the treatment of acute tendinitis. *Eur J Rheumatol Inflamm* 1991;**11**:38–45.
22 Khan KM, Visentini PJ, Kiss ZS, *et al*. Correlation of US and MR imaging with clinical outcome after open patellar tenotomy: prospective and retrospective studies. *Clinical Journal of Sports Medicine* 1999;**9**:129–37.
23 Panni A, Tartarone M, Maffuli N. Patellar tendinopathy in athletes. Outcome of nonoperative and operative management. *Am J Sports Med* 2000;**28**:392–7.
24 Testa V, Capasso G, Maffulli N, *et al*. Ultrasound guided percutaneous longitudinal tenotomy for the management of patellar tendinopathy. *Med Sci Sport Exercise* 1999;**31**:1509–15.
25 Cook J, Khan K, Kiss ZS. Patellar tendinitis: the significance of magnetic resonance imaging findings [letter]. *Am J Sports Med* 1999;**27**:831.
26 Cook JL, Khan KM, Harcourt PR, *et al*. Patellar tendon ultrasonography in asymptomatic active athletes reveals hypoechoic regions: a study of 320 tendons. *Clinical Journal of Sports Medicine* 1998;**8**:73–7.
27 Shalaby M, Almekinders LC. Patellar tendinitis: the significance of magnetic resonance imaging findings. *Am J Sports Med* 1999;**27**:345–9.
28 Cook J, Khan K, Kiss ZS, *et al*. Reproducibility and clinical utility of tendon palpation to detect patellar tendinopathy in young basketball players. *Br J Sports Med* 2001;**35**:65–9.
29 Cook JL, Khan K, Harcourt PR, *et al*. A cross-sectional study of 100 cases of jumper's knee managed conservatively and surgically. *Br J Sports Med* 1997;**31**:332–6.
30 Blazina M, Kerlan R, Jobe F, *et al*. Jumper's knee. *Orthop Clin North Am* 1973;**4**:665–78.
31 Curwin S, Stanish WD. *Tendinitis: its etiology and treatment*. Lexington: Collamore Press, 1984.
32 Kelly DW, Carter VS, Jobe FW, *et al*. Patellar and quadriceps ruptures-jumper's knee. *Am J Sports Med* 1984;**12**:375–80.
33 McCarty D, Gatter R, Phelps P. A dolorimeter for quantification of articular tenderness. *Arthritis and Rheumatology* 1965;**8**:551–9.
34 Colosimo AJ, Bassett FH. Jumper's knee: diagnosis and treatment. *Orthop Rev* 1990;**29**:139–49.

The last "oxygenless" ascent of Mt Everest

Our continued fascination with the element oxygen (O_2), first discovered by Joseph Priestley (1733–1804), is eminently justified, for without it we would simply not survive. Photosynthesis breathes life into what was 1000 million years ago considered to be a reductive atmosphere containing only 1–2% O_2. Contemporary estimates now suggest that the green plants on earth combine a total of 150 billion tons of carbon (from CO_2) with 25 billion tons of H_2 (from H_2O) to liberate 400 billion tons of O_2 each year, thus accounting for the present day atmospheric content of O_2 (20.9%), which has persisted for the last one tenth of the Earth's existence.[1]

Few are more acutely aware of the importance of the elixir of life than mountaineers when exposed to the innocuous effects of hypobaric hypoxia during ascent to terrestrial high altitude. Since the French mathematician Blaise Pascal (1623–1662) first showed that barometric pressure (and hence the inspired partial pressure of O_2 (P_{IO_2})) decreased with altitude, scientists have developed a fascination with the maximal altitude that humans could achieve without the unsporting assistance of supplemental O_2. The ensuing battle between scientific prediction and human performance soon blossomed into one of the most colourful sagas in the history of high altitude mountaineering and physiology.[2]

The aristocratic Italian climber, the Duke of Abruzzi, rocked the scientific world at the turn of the last century by completing an "oxygenless" ascent to 7500 m in the Karakoram despite ominous predictions by the British Alpine Club, " . . .21 500 ft (6553 m) is near the limit at which man ceases to be capable of slightest further exertion."[3] Naturally, it was not long before attention turned towards Mt Everest, which in 1892 had been declared the highest mountain in the world. Mountaineers were irrevocably drawn to the ultimate challenge, and, in 1924, Edward Norton (1884–1954) ascended to within 300 m of its summit without supplementary O_2, a remarkable feat eclipsed by the untimely deaths of George Mallory and Andrew Irvine some two days later.

These courageous attempts motivated a series of pioneering studies by Rodolfo Margaria (1901–1983) and Joseph Barcroft (1872–1947), who subsequently concluded that an oxygenless ascent of Mt Everest would not be possible.[4] However, the epochal ascent by Reinhold Messner and Peter Habeler in 1978 put paid to their gloomy predictions; the last 3% of Mt Everest had finally been conquered 54 years after Norton's initial bid, having claimed no fewer than 39 lives in the process. Messner's quote on reaching the top clearly establishes that the summit of Mt Everest was tantalisingly close to the limits of human tolerance, " . . .Now, after the hours of torment . . . I have nothing more to do than breathe . . .I am nothing more than a single, narrow, gasping lung, floating over the mists and the summits."[5] By 1986, Messner had confirmed his place in mountaineering history having climbed all 14 of the 8000 m giants without supplementary O_2.

The physiological significance of these achievements came to light a decade later when two landmark studies[6 7] measured the maximal oxygen uptake (V_{O_2MAX}) of healthy volunteers on the summit at a mere 1.07–1.17 litres/min or 15 ml/kg/min. These maximal energy expenditures are comparable to those typically observed in exercising patients with congestive heart failure, albeit 5.5 miles lower at sea level! Furthermore, the geographical location of Mt Everest at a latitude of 28°N results in a barometric pressure that is considerably higher than if it were located near one of the poles. The climatic idiosyncrasy of this "equatorial bulge" is fortunate in that, without it, a climber would not be able to summit without supplementary O_2.[8]

It is, however, somewhat discouraging to note that the physical challenge of climbing Mt Everest without supplementary O_2 is becoming more difficult as the summit appears to be increasing in height at a rate of 3 cm/year and steadily moving north eastward at 6 cm/year (Geological Society, personal communication). This is a consequence of it being located on the great fault system, which continues to push India under Nepal and China thus creating the Himalayas. These data required the initial measurement of the summit, which in itself has proved one of the most formidable geological challenges since the first attempt using vertical triangulation by James Nicolson during 1847–1849 (8840 m), which failed to take into account the deflection of the vertical produced by the Himalayan chain, the discrepancy between geoid and ellipsoid, and refraction of the atmosphere. The Chinese National Bureau of Surveying and Mapping and the Italian National Research Council have subsequently performed what is arguably the most accurate measurement of the summit using laser distance meters, theodolites, and satellite positioning systems. The value reported in 1992 was 8848.65 (0.35) m above sea level (inclusive of the snow on the summit measured as 2.55 m). These facts raise the irresistible question of how much longer an oxygenless ascent will remain humanly possible.

Figure 1 (based on the data presented in table 1) clearly illustrates that systemic O_2 transport is exquisitely sensitive

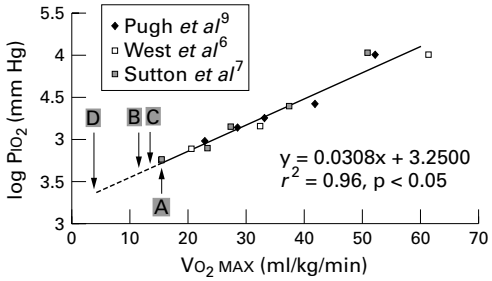

Figure 1 Decline in maximal oxygen uptake ($V_{O_2}MAX$) expressed relative to total body mass with a decrease in the inspired partial pressure of oxygen (P_{IO_2}). Data on the ordinate are log transformed and based on the individual data points presented in table 1. Point A illustrates existing data for $V_{O_2}MAX$ obtained during two "simulated" ascents of Mt Everest. Summit P_{IO_2} (42.5–43 mm Hg) was obtained (a) at a terrestrial altitude of 6300 m with subjects inspiring 14% O_2 during the American Medical Research Expedition to Everest[6] and (b) in a decompression chamber during Operation Everest II.[7] The regression line has been extrapolated to predict the corresponding P_{IO_2} values at: (a) 3.5 METs (assuming that 1 MET = 3.5 ml/kg/min), which equates to the lowest maximal value required for successful ascent of the last 100 m of Mt Everest during the summer months (barometric pressure about 251 mm Hg) assuming an average energy expenditure equivalent to 85% of a climber's $V_{O_2}MAX$ (point B); (b) 3.9 METs which is considered the lowest maximal value required for a winter ascent such as that achieved by Sherpa Ang Rita (point C) which is considerably more difficult despite a mere <1 mm Hg reduction in the P_{IO_2} compared to (a); (c) 1 MET which equates to a climber's basal metabolic rate (point D).

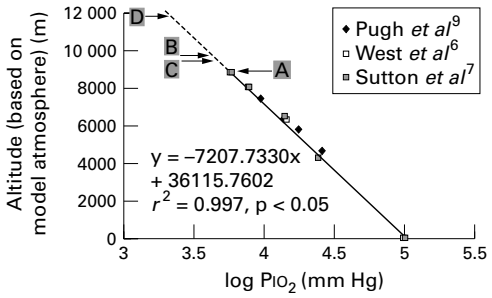

Figure 2 Inverse relation between altitude and the inspired partial pressure of oxygen (P_{IO_2}) based on the individual data points presented in table 1. Data on the abscissa are log transformed. Point A indicates the present altitude of Mt Everest measured at 8848.92 m. The regression line has been extrapolated to predict the corresponding altitudes at the respective P_{IO_2} values predicted in fig 1 (point B = 9972.7 m, point C = 9662.1 m, and point D = 11 913.9 m).

to a decline in P_{IO_2} primarily because of a decrease in alveolar P_{O_2} (P_{AO_2}). Extrapolation of this relation predicts the P_{IO_2} at a variety of energy expenditures ranging from 1 metabolic equivalent (MET) to 3.9 METS compared with the measured maximum of about 4.5 METS (assuming 1 MET = 3.5 ml/kg/min). Whole body $V_{O_2}MAX$ is expressed in relative terms and further reduced to METS to normalise for the unavoidable differences between studies in subject body mass, which is an undoubted confounding variable during attempts to fit pooled data.

The value of 3.5 METS (point B on fig 1) was selected because 3 METS is considered the minimum energy expenditure required for a safe ascent to the summit during the summer months assuming that the climber is working at about 85% of his maximum, which is not unreasonable (J S Milledge, personal communication). Messner's total body mass including equipment was reported to be 74 kg,[10] which at an ascent rate of 2 m/min during the last 100 m of the climb would have resulted in a power output close to 150 kg/m/min. According to the data of West *et al*[6] and the aforementioned assumption, this submaximal effort would have required a $V_{O_2}MAX$ of approximately 3.5 METS.

Table 1 Measured values of maximal oxygen uptake ($V_{O_2}MAX$) and inspired partial pressures of oxygen (P_{IO_2}) at various altitudes in three landmark investigations[6 7 9]

Study	*Sample size (n)*	*Altitude (m) Model atmosphere*	*P_{IO_2} (mm Hg)*	*$V_{O_2}MAX$ (ml/kg/min)*
Pugh *et al*[9]	3	Sea level	147.1	52.1
	3	4650	82.3	41.7
	3	5800	69.7	33.0
	3	6400	62.2	28.5
	1	7440	53.0	22.8*
West *et al*[6]	5	Sea level	147.1	61.3
	8	6300	63.7	32.4
	6	8050	48.5	20.6
	2	8848	42.5	15.3
Sutton *et al*[7]	8	Sea level	150.0	50.8
	8	4300	80.0	37.3
	7	6500	63.0	27.2
	5	8100	49.0	23.3
	5	8848	43.0	15.4

*One subject in the original paper was excluded from analysis because of infectious illness at the time of exercise testing.

Seasonal variations in barometric pressure at these extreme altitudes have a major impact on systemic O_2 transport and are thus of considerable physiological significance. Previous theoretical calculations have identified that a mere 4 mm Hg decrease in summit barometric pressure would reduce a climber's predicted $V_{O_2}MAX$ by about 10%.[8] Thus, point C on fig 1 refers to the predicted $V_{O_2}MAX$ of a climber based on the previous assumptions attempting to summit during the winter months, a remarkable feat that was achieved on 22 December 1987 by Sherpa Ang Rita. Retrospective analyses indicated that the summit pressure during this ascent was 247 mm Hg,[11] only 3–4 mm Hg lower than that observed during the American Medical Research Expedition to Everest (AMREE)[6] and Operation Everest II[7] (point A).

Incorporating this information into the linear function presented in fig 2 (based on the data summarised in table 1), we can identify the corresponding altitude at each of the respective P_{IO_2} values which equates to 9972.7 m (3.5 METS, point B), 9662.1 m (3.9 METS, point C), and 11 913.9 m (1 MET, point D).

Assuming a constant growth rate of 3 cm/year and present (2001) height of 8848.92 m, a climber with a $V_{O_2}MAX$ of 3.5 METS would therefore have to make his assault on the summit during the summer months before about 39 460 AD. A winter ascent would need to be launched before 29 107 AD. By about 104 167 AD, a climber's $V_{O_2}MAX$ on the summit would equate to his basal metabolic rate measured at sea level, emphasising the futility of any summit bid. Incidentally, these dates could be extended by another 85 years if the snow on the summit melted.

A climber with a sea level $V_{O_2}MAX$ that is comparatively higher than the values incorporated in fig 1 could further extend the theoretical timescale of an oxygenless ascent. Data obtained during AMREE[6] clearly indicate a slight leftward displacement of the $V_{O_2}MAX$ α P_{IO_2} curve, effected in part by a 24% higher sea level $V_{O_2}MAX$ when compared with the data obtained by Pugh *et al*.[9] Hypoxic chemosensitivity may also affect these predictions, with previous studies indicating that climbers with a "brisk" hypoxic ventilatory response are capable of reaching higher altitudes on the mountain.[12] However, the hyperventilatory responses observed at high altitude are energetically more demanding, accounting for 26% of an individual's $V_{O_2}MAX$ at 5050 m compared with 5.5% at sea level (assuming a mechanical efficiency of 5%).[13] Thus it would appear that the mechanical power of breathing may impair a climber's ability to perform external work on the mountain. The limits for reaching the theoretical summit are also affected by subtle changes in barometric pressure as the comparative example of Messner's summer ascent versus Sherpa

Ang Rita's winter ascent clearly indicates; a successful winter bid would have to occur about 10 millenia before a summer bid despite a mere 3–4 mm Hg difference in summit pressure. More pronounced seasonal changes in summit pressure, which can differ by up to as much as 11.5 mm Hg at Everest's present altitude, could further reduce the theoretical timescale of an oxygenless ascent.[14] Changes in the geological (summit growth rate) and climatic (F_{IO_2} and barometric pressure α altitude relation possibly as the result of global rewarming) assumptions on which these calculations are based have obvious implications for summit dates; whether the predictions are correct, only time will tell!

Despite its appeal, the risks inherent in an ascent to such extreme altitudes by "fair means" are quite considerable and can increase a climber's susceptibility to hypoxic brain damage, as studies showing clear evidence for residual impairment of central nervous system function after return to sea level[15] would seem to suggest. Furthermore, recent epidemiological data clearly indicate that an oxygenless ascent/descent of Mt Everest and K2 is associated with more fatalities[16] probably because of the proposed therapeutic benefits of supplemental O_2 as a means of reducing the incidence of "medical" (related to altitude illness) and "traumatic" (caused by accident as the result of misjudgement) deaths.[17] Thus, although high altitude mountaineers may find some reassurance that, at least for the next 40 millennia, an oxygenless ascent will have some chance of succeeding, the ethical implications of such a remarkable feat warrant due consideration. The physical and intellectual challenge posed by Everest lives on!

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1 Asimov I. *Asimov's new guide to science*. Revised ed. London: Penguin Group, 1984:191–231.
2 West JB. *High life: a history of high-altitude physiology and medicine*. New York: Oxford University Press, 1998.
3 Hinchcliff TW. *Over the sea and far away*. London: Longmans Green, 1876: 90–1.
4 Barcroft J, Douglas CG, Kendal LP, *et al*. Muscular exercise at low barometric pressures. *Arch Sci Biol Napoli* 1931;**16**:609–15.
5 Messner R. *Everest: expedition to the ultimate*. London: Kaye and Ward, 1979: 180.
6 West JB, Boyer SJ, Graber DJ, *et al*. Maximal exercise at extreme altitudes on Mount Everest. *J Appl Physiol* 1983;**55**:688–98.
7 Sutton JR, Reeves JT, Wagner PD, *et al*. Operation Everest II: oxygen transport during exercise at extreme simulated altitude. *J Appl Physiol* 1988;**64**: 1309–21.
8 West JB, Wagner PD. Predicted gas exchange on the summit of Mt. Everest. *Respir Physiol* 1980;**42**:1–16.
9 Pugh LGCE, Gill MB, Lahiri S, *et al*. Muscular exercise at great altitudes. *J Appl Physiol* 1964;**19**:431–40.
10 Pugh LGCE, Sutton JR. Everest then and now. In: Sutton JR, Houston CS, Jones NL, eds. *Hypoxia, exercise, and altitude: proceedings of the Third Banff International Hypoxia Symposium*. New York: AR Liss, 1983:415–28.
11 Ward MP, Milledge JS, West JB. *High altitude medicine and physiology*. London: Arnold, 2000:28.
12 Schoene RB, Lahiri S, Hackett PH, *et al*. Relationship of hypoxic ventilatory response to exercise performance on Mount Everest. *J Appl Physiol* 1984;**56**:1478–83.
13 Cibella F, Cuttitta G, Romano S, *et al*. Respiratory energetics during exercise at high altitude. *J Appl Physiol* 1999;**86**:1785–92.
14 West JB, Lahiri S, Maret KH, *et al*. Barometric pressures at extreme altitudes on Mt. Everest: physiological significance. *J Appl Physiol* 1983;**54**:1188–94.
15 Hornbein TF, Townes BD, Schoene RB, *et al*. The cost to the central nervous system of climbing to extremely high altitude. *N Engl J Med* 1989;**321**: 1714–19.
16 Huey RB, Eguskitza X. Supplemental oxygen and mountaineer death rates on Everest and K2. *JAMA* 2000;**284**:181.
17 Pollard A, Clarke C. Deaths during mountaineering at extreme altitude. *Lancet* 1988;**1**:1277.