

This document contains supplemental data and illustrations for a study performed about mortality on Everest. At the end of this document there is an informal outline of the content, implications and limitations of the study.

The study may be accessed free of charge at [BMJ.com](http://BMJ.com).

**Reference:** Firth PG, Zheng, H, Windsor JS, Sutherland AS, Imray CH, Moore GWK, Semple JL, Roach RC, Salisbury RA. Mortality on Mount Everest, 1921-2006: descriptive study. *BMJ* 2008; **337**: a2654

## Abstract

**Objective** To examine patterns of mortality among climbers on Mount Everest over an 86 year period.

**Design** Descriptive study.

**Setting** Climbing expeditions to Mount Everest, 1921-2006.

**Participants** 14 138 mountaineers; 8030 climbers and 6108 sherpas.

**Main outcome measure** Circumstances of deaths.

**Results** The mortality rate among mountaineers above base camp was 1.3%. Deaths could be classified as involving trauma (objective hazards or falls, n=113), as non-traumatic (high altitude illness, hypothermia, or sudden death, n=52), or as a disappearance (body never found, n=27). During the spring climbing seasons from 1982 to 2006, 82.3% of deaths of climbers occurred during an attempt at reaching the summit. The death rate during all descents via standard routes was higher for climbers than for sherpas (2.7% (43/1585) v 0.4% (5/1231), P<0.001; all mountaineers 1.9%). Of 94 mountaineers who

died after climbing above 8000m, 53 (56%) died during descent from the summit, 16 (17%) after turning back, 9 (10%) during the ascent, 4 (5%) before leaving the final camp, and for 12 (13%) the stage of the summit bid was unknown. The median time to reach the summit via standard routes was earlier for survivors than for non-survivors (0900-0959 v 1300-1359,  $P < 0.001$ ). Profound fatigue ( $n=34$ ), cognitive changes ( $n=21$ ), and ataxia ( $n=12$ ) were the commonest symptoms reported in non-survivors, whereas respiratory distress ( $n=5$ ), headache ( $n=0$ ), and nausea or vomiting ( $n=3$ ) were rarely described.

**Conclusions** Debilitating symptoms consistent with high altitude cerebral oedema commonly present during descent from the summit of Mount Everest. Profound fatigue and late times in reaching the summit are early features associated with subsequent death.

**Supplemental Data.**

**Table 1. Numbers and routes of summits ascents 1953-2006**

<b>Summit ascents</b>	Climbers	Sherpas	Total
<b>Routes used</b>			
South route	832 (47.1)	735 (56.9)	1567 (51.2)
North route	753 (42.6)	496 (38.5)	1249 (40.8)
Other routes	183 (10.4)	59 (4.6)	242 (7.9)
<b>Summit ascents</b>			
1953-80	84 (4.8)	20 (1.6)	104 (3.4)
1980-89	135 (7.6)	45 (3.5)	180 (5.9)
1990-99	568 (32.1)	311 (24.1)	879 (28.7)
2000-2006	985 (55.7)	884 (68.5)	1839 (60.1)
<b>Total</b>	<b>1768</b>	<b>1290</b>	<b>3058</b>

Values are the numbers of summits, with percentages in parenthesis. Repeat ascents by individuals are counted as separate ascents. The south route includes ascents and descents by the standard southeast ridge route, or minor variations. The north route refers to ascents and descents via the standard northeast ridge or minor variants. Other routes include ascents via other routes or combinations involving the standard routes.

**Table 2. Classification of deaths 1921-2006.**

<i>Location/ Class of Fatality</i>	<i>Classification of fatality</i>	<i>Climber/ Expedition Member</i>	<i>Sherpa/ Expedition employee</i>	<i>Total</i>	<i>P value (†)</i>
<b>ABOVE BC</b>					
<b><u>Trauma</u></b>		<b><u>54</u></b>	<b><u>59</u></b>	<b><u>113</u></b>	
	<b>Objective hazards</b>	<b>20</b>	<b>47</b>	<b>67</b>	
	<b>Falls</b>	<b>34</b>	<b>12</b>	<b>46</b>	0.02
<b><u>Non-trauma</u></b>		<b><u>46</u></b>	<b><u>6</u></b>	<b><u>52</u></b>	<0.001
	<b>High altitude illness</b>	<b>12</b>	<b>5</b>	<b>17</b>	0.22
	HACE	6	1	7	0.04
	HAPE	2	3	5	0.40
	HAI (Unspecified)	4	1	5	0.40
	<b>Hypothermia</b>	<b>11</b>	<b>0</b>	<b>11</b>	0.004
	<b>Sudden death</b>	<b>7</b>	<b>0</b>	<b>7</b>	0.02
	<b>Unclassified</b>	<b>16</b>	<b>1</b>	<b>17</b>	0.001
	HAI/hypothermia	6	0	6	0.006
	Unknown	9	0	9	0.01
	Unclassified	1	1	2	1.00

<b><u>Disappeared</u></b>		<b><u>25</u></b>	<b><u>2</u></b>	<b><u>27</u></b>	0.0001
Suspected cause	HAI/hypothermia/fall	13	1	14	0.006
	Fall	8	1	9	0.04
	Unknown	4	0	4	0.14
<b><u>Total</u></b>		<b><u>125</u></b>	<b><u>67</u></b>	<b><u>192</u></b>	
<b>Death rates - %</b>		1.6	1.1	1.3	<0.0001(*)
<b>BC/BELOW BC</b>		<b><u>8</u></b>	<b><u>12</u></b>	<b><u>20</u></b>	
Members	Sudden death (3); unclassified (2); avalanche (1); HAPE (1), drowning (1).	8			
Employees	Hypothermia (3), unclassified (3), drowning (1), diarrhea (1), HAPE (1), acute abdomen (1); avalanche (1); sudden death (1)		12		
<b>TOTAL</b>		<b><u>133</u></b>	<b><u>79</u></b>	<b><u>212</u></b>	
<b>Death rates (Assembly to departure) -%</b>		1.5	N/A	1.4 (**)	

Base camp (BC) was defined as the last encampment on any route before technical climbing began – Base Camp in Nepal and Advanced Base Camp in Tibet. Expedition members are defined as individuals listed on expedition permits issued by local

authorities. Professional guides from outside the Himalaya are defined as expedition members, as per permit documentation. 'Climbers' refers to expedition members who went above BC. Expedition member deaths at or below BC include non-climbers. The term 'sherpa' is used to describe all High Altitude Porters, irrespective of ethnicity. Employee deaths at or below BC include support staff and porters employed to transport equipment and provisions below BC. Accurate records of the size of this population are not available. Values are the numbers of individuals killed, except where specified.

Death that involved trauma was classified by the cause of the trauma. High-altitude cerebral oedema (HACE) was diagnosed by the presence of ataxia, altered consciousness or both, following a recent ascent in altitude, without evidence of another cause. High-altitude pulmonary oedema (HAPE) was diagnosed by the presence of respiratory distress, cough or both, following a recent ascent to altitude, without evidence of another cause, or by virtue of a reliable report. If symptoms of both HACE and HAPE were present, the death was classified as HAPE as we could not reliably distinguish pulmonary from cerebral vascular dysfunction as the primary cause of neurological symptoms. If a specific diagnosis of HACE or HAPE could not be established, or Lake Louise symptoms of Acute Mountain Sickness (AMS) were present, the cause was classified as indeterminate high-altitude illness (HAI). Death due to exposure was classified as hypothermia. Sudden death was defined as abrupt death without prior progressive symptoms. The fatality was classified as disappearance if the death or the immediate cause of death (objective hazard) was not witnessed and the body not found. Non-traumatic death where the immediate preceding circumstances were not witnessed but the

body discovered was classified as unknown. Death in which a single primary cause could not be established was listed as unclassified. Miscellaneous other fatalities were diagnosed by available evidence.

The Fleiss inter-rater kappa value for classification of fatalities was 0.63,  $p < 0.001$ .

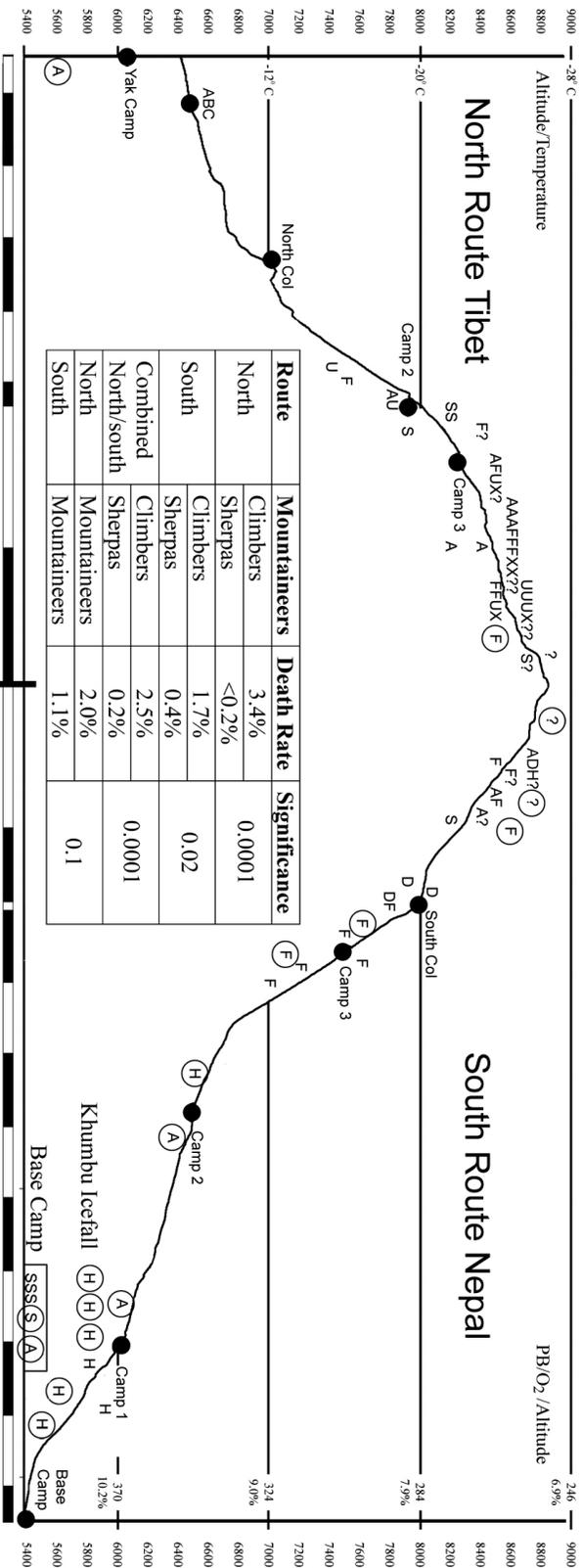
Percentage may not add up to 100 percent due to rounding.

(\*) The statistical comparison of rates assumes all deaths are independent of each other.

(\*\*) Refers to total of expedition members and high-altitude sherpas; excludes employees at or below BC, for whom accurate records of population size are not available.

(†) P-values are calculated by a Fisher's exact test when the counts are less than 30, and by a Chi-square test when counts are greater than 30. Comparison is between the death rates of climbers and sherpas. Comparisons assume independence of deaths and ascents.

**Figure 1. Fatalities on standard North and South routes during the spring climbing season, April-June 1982- 2006.**

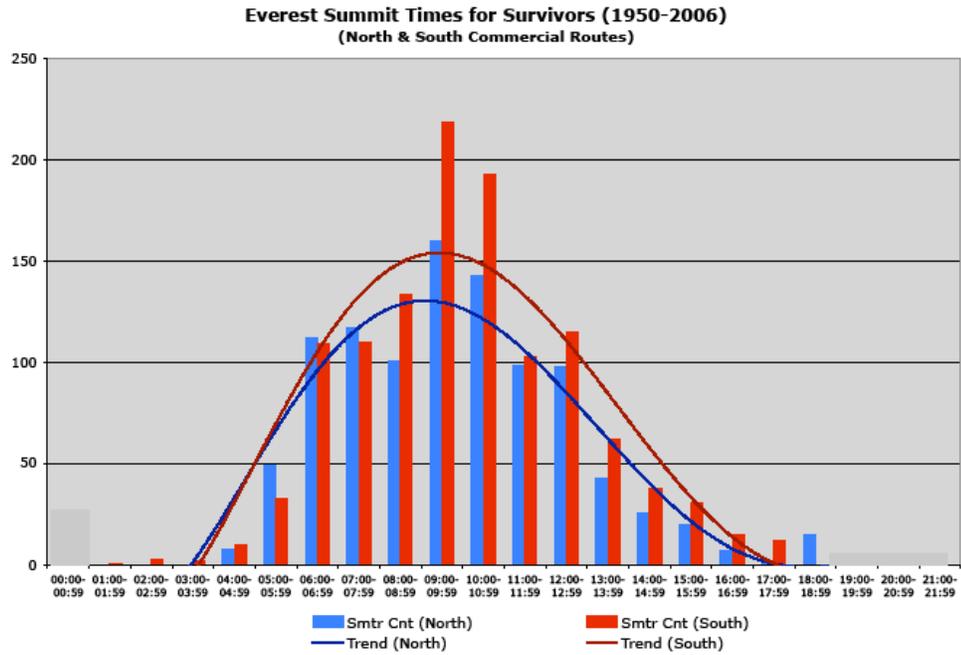


Death rates in the table are the rates of fatalities during descent from the summit. Deaths during descent from the summit are plotted above the route profile line, while deaths at base camp, or during route preparation, unsuccessful summit bids and summit bids with unknown outcome are shown below the profile. Sherpa fatalities are represented by circled letters, climber deaths by uncircled letters. Key to classification of deaths: High altitude illness – A; Unclassified hypothermia/exposure – D; Fall – F; H – Objective hazard (avalanche, ice fall collapse, crevasse fall); Unclassified hypothermia/HAI – X; Disappeared - ?; Sudden death – S; Unclassified/unknown – U. One climber’s death occurring during descent from Nepal Base Camp is shown at 5400m. One sherpa was evacuated from below Yak Camp in Tibet and died near the Rongbruk Monastery – death is plotted near the elevation of evacuation.

As in figure 1, altitude in meters above sea level is shown on both Y-axes. Estimated typical barometric pressure during May is shown on the right Y-axis.<sup>14</sup> Oxygen percentages on the right Y-axis refer to the percentage of oxygen at sea level (760mmHg) that exerts an equivalent partial pressure to the atmospheric oxygen at the relevant altitude. Estimated ambient air temperature during May is shown on the left Y-axis.<sup>15, 16</sup> X-axis is horizontal distance in kilometer intervals; the scale is expanded by a factor of two for the section corresponding to the route above 8000m.

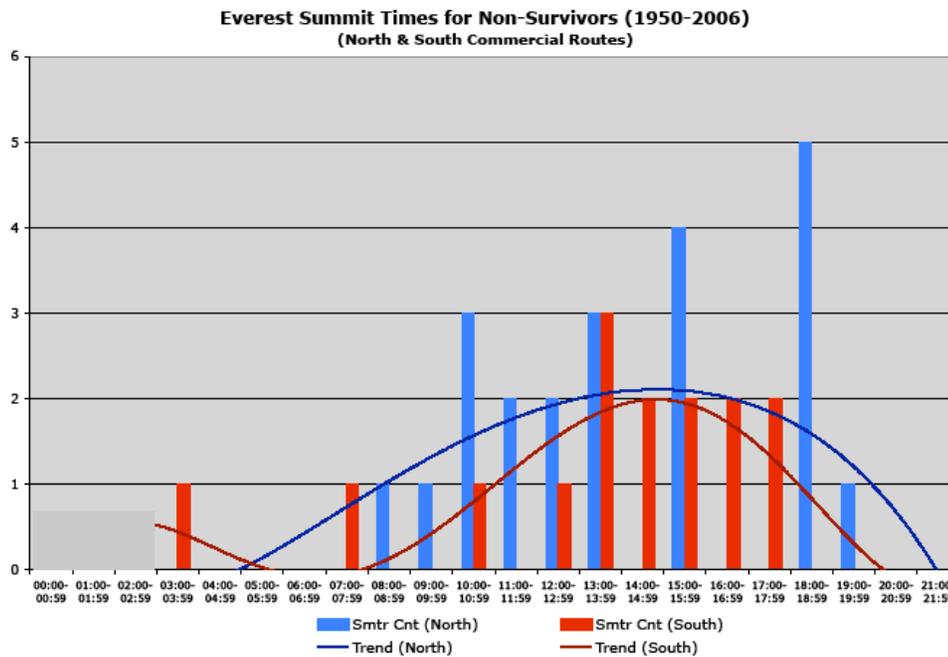
P values are calculated by a two-sided Fisher’s exact test, comparing climbers with sherpas, and north with south route.

The route profile was produced from topographical data kindly provided by Professor A. Gruen and Martin Sauerbier, Institute of Geodesy and Photogrammetry, Zurich, Switzerland.



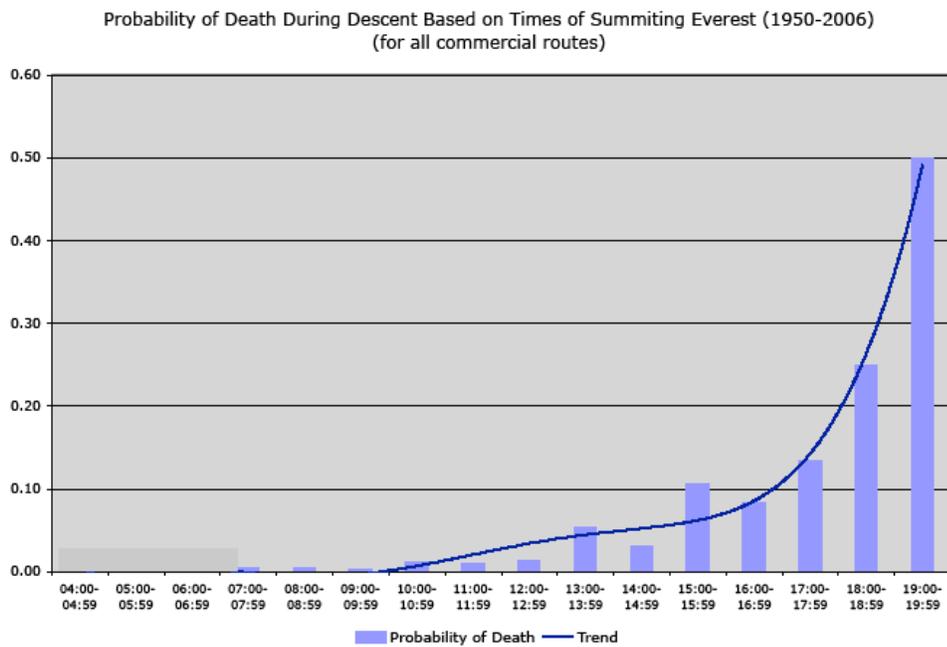
**Figure 2. Summit times for survivors (1953-2006) via the ‘standard’ north and south routes.**

Median summit time was 09h00-09h59 (interquartile range 08h00-08h59 to 11h00-11h59)



**Figure 3. Summit times for non- survivors (1953-2006) via the ‘standard’ north and south routes.**

Median summit time was 13h00-13h59 (interquartile range 11h00-11h59 to 16h00-16h59). The difference between survivors’ and non-survivors’ summit times was significant ( $P < 0.001$ ).



**Figure 4. Probability of death during descent, plotted against the time of summiting Everest.**

## **An informal discussion of the findings, implications and limitations of this study**

### **What did this study examine, and how was it done?**

The study set out to examine the circumstances of all deaths on Everest expeditions between 1921 and 2006. We identified all deaths on Everest expeditions by searching the Himalayan Database and expedition books. We then located accounts of these deaths by examining reports in the Database, expedition books, climbing journals, or on the internet, or by directly contact with witnesses. Four doctors, all of whom had climbed on Everest, classified the deaths using a descriptive classification system. Three had summited Everest, one had turned back at 8300m. All four reviewers had a particular interest in high altitude physiology, and had practical experience in managing high altitude illness. The classifications were then pooled and differences were resolved by consensus. The degree of disagreement between the independent assessments was measured to give an indication of the reliability or certainty of the final classification system.

We then looked at specific subsets of events. As most mountaineers are interested in what happens higher on the mountain we focused on events above base camps, defined as the last encampment before technical (roped) climbing began. As the majority of summits were via the 'standard' South-west and North-east routes, during the spring season, and in the last 25 years, we also examined this group in detail. This minimized variable such as season, differing difficulty of the technical routes, and the changes in expedition styles that have occurred over the decades. We looked at the specific circumstances deaths of those that died after climbing above 8000m.

To examine the effect of weather, we used measures of barometric pressure at 9000m obtained from a dataset at the National Center for Environmental Prediction. This has been used previously in studies of weather conditions on Everest.

### **What were the most significant findings?**

1. For the entire study period, the mortality rate of mountaineers above base camp was 1.3%. Among climbers it was 1.6%, among sherpas it was 1.1%.
2. Deaths could be classified as involving trauma (objective hazards or falls), as non-traumatic (high altitude illness, hypothermia or sudden death) or as a disappearance (unwitnessed death, body not found).
3. During the spring seasons on the standard routes, most climbers died above 8000m during a summit bid. The death rate on the north was 3.4%; on the south side it was 2.5%.
4. Most mountaineers who died above 8000m died during descent from the summit.
5. Climbers died at a much higher rate during the descent than sherpas.
6. Late summit times were associated with subsequent death.

7. Many developed symptoms suggestive of high altitude cerebral edema (HACE): confusion, loss of consciousness, a staggering gait.
8. Symptoms of high altitude pulmonary edema (HAPE) were rare in those that died.
9. The earliest sign was marked fatigue, as reflected in reports that the mountaineer looked exhausted, the tendency to fall behind other climbers on the party, and a late summit time.

### **Why did people die above 8000m?**

This study could not answer the question “why” since it was an observational study – we were only observing what happened rather than performing an experiment where the variables that may have caused the result were controlled. Stated differently, we were looking at what happened and then detecting factors that were associated with these events (“how” not “why”). We then suggest that some of these factors may play a role in causing these events – but this is interpretation or speculation.

We classified deaths by a descriptive technique. Above 8000m, we classified deaths as those that involved a fall (34%), disappeared (29%), high altitude illness (11%), suddenly death (5%), hypothermia (2%) or we couldn't decide exactly a single classification (15%) – usually between HACE or hypothermia. We used this technique since it allowed us to classify a very varied series of accounts, of a very varied set of circumstances, in an objective and reproducible way that minimized our own interpretation or bias. From this foundation, we could then examine the deaths in more detail and try to look for factors we speculated might have caused the fatalities.

Of these deaths, a large number involved climbers that developed neurological problems before falling or disappearing – confusion or staggering gait. This would suggest that neurological problems were an underlying ‘cause’ of many of these deaths.

### **How reliable is this classification system?**

Since an observer examining an account might read different things into what happened, we used four reviewers, all of who examined the accounts independently of one another. For all 212 deaths, there was an initial unanimous agreement for 165 (78%) of classifications. This number does not give a full measure of agreement – if, for example, only one reviewer disagreed on the remainder, this would be less disagreement than if all four reviewers came up with four completely different classifications. So we used a statistical test called the Fleiss Kappa test that measure the degree of inter-rater agreement. The kappa value was 0.63, which implies ‘substantial agreement.’ It is impossible to accurately classify all deaths due to the variability in accounts, the differing circumstances, and the possibility of unknown causes. If one has a classification system that gives 100% agreement, this would probably be too insensitive a reflection of complex events. We felt the measure of

agreement reflects the ‘sweet spot’ between agreement and uncertainty in a classification system.

### **What causes the confusion and loss of co-ordination?**

We felt that these symptoms were consistent with cerebral edema (HACE), caused by the low oxygen atmospheric content at extreme altitude. HACE is due to inadequate acclimatization to the low oxygen levels, which results in the blood vessels in the brain leaking fluid into the surrounding brain tissue (edema). Confusion and loss of co-ordination follow.

Hypothermia or extreme cold can also cause these symptoms. In some cases we felt the cold was the primary cause of the symptoms, in others it was due to HACE, and in some cases we couldn’t determine which was the primary cause. A climber who is incapacitated by HACE will easily develop hypothermia in the extreme cold above 8000m.

We also looked for signs of visual disturbances (snow blindness or retinal hemorrhages) that might masquerade as confusion or uncoordinated gait. Although we know from experience that these are significant problems at extreme altitude we didn’t find evidence to suggest that visual problems were a common primary explanation for the symptoms. We didn’t think the symptoms could be explained by visual disruption alone.

Other physical features associated with extreme altitude, such as high ionic radiation, are not known to cause these effects in the relevant time frame.

### **Couldn’t the deaths and neurological symptoms on descent simply be due to climbers running out of oxygen?**

Running out of supplemental oxygen probably doesn’t help the functioning of the brain. However, we saw similar patterns of mortality in climbers who died while climbing without supplemental oxygen. In addition, it is not uncommon for climbers to have failures of their delivery sets while climbing up (this happened to one of the authors for example). However we didn’t detect any deaths due to this. Although we suspect that loss of supplemental oxygen plays a role in subsequent deaths, we interpreted this as mountaineers typically having pre-existing problems that heighten their susceptibility to loss of supplemental oxygen.

### **So is fatal HAPE rare at extreme altitude?**

This study suggests that it is unusual for non-survivors to develop florid HAPE. This may be because mountaineers who get HAPE turn back early and survive, because those with severe HAPE are still able to get down and live, because those prone to HAPE get it lower down the mountain, or because the use of supplemental oxygen (which lowers blood pressure in the lungs) alters the response to altitude. Another

caveat is that this study is the first to examine the problems in the ‘death zone’ in detail – perhaps other future studies may have other results. However we detected very few deaths that clearly involved HAPE.

### **So how do you tell if you are getting HACE?**

Unfortunately once you get HACE you are in severe trouble – your insight is impaired, since you are confused and may not realize what is happening. As you are confused and uncoordinated, it is difficult for other mountaineers to rescue you since they have to physically haul you down the mountain. In the absence of a large team, this is hard to do (but not impossible – organized teams can get even severely impaired climbers down to lower altitude where they can make good recoveries).

The earliest sign seems to be marked fatigue – sense of extreme exhaustion, falling behind the group, and making the summit later in the day.

### **How late is late? Everyone is exhausted at 8000m – how fast do the individual need to climb?**

Unfortunately we don’t have records of what time mountaineers left summit camps at, or what camp they left from on the North Side. There is also no record of the exact oxygen flow rates that climbers used, which will impact on their climbing speed. Almost all survivors make the summit by around noon to very early afternoon. However assuming most leave around 10pm - midnight, this means a maximum of about 12 –14 hours. The risk of death associated with slower climbing speeds and later summit times goes up dramatically after this.

In recent years large numbers of climbers make summit bids on the same days. Measuring ascent speed against other mountaineers is another yardstick.

### **What about other symptoms?**

Other early signs of HACE include nausea, vomiting and headache. These symptoms seemed rare in those that died. The precise mechanisms that lead to these symptoms are unknown – one theory is that they may be related to stretching of the sensitive membranes covering the brain as it swells. We speculate that at extreme altitude, the brain does not swell as much before debilitating confusion and loss of coordination set in, so headache and nausea are not reliable early warning signs. However perhaps witnesses simply didn’t report these events, or those mountaineers with these symptoms turned back earlier and survived. Further study will be needed to confirm this.

### **Couldn’t the late summit time-death association simply be explained by more time at risk of being caught in bad weather?**

We were somewhat surprised to see how rarely bad weather played a role in deaths at very high altitudes during the spring. We were able to use weather data collected over years to look at an association of deaths with changes in atmospheric conditions. The most useful marker was barometric pressure, although other weather markers have also been collected. Most summit bids occur in good weather – and most days when deaths occur have reasonable weather too. When the weather does turn bad it can kill a lot of people if it catches teams out on the mountain. However the 1996 “Into Thin Air” storms are quiet unusual events.

### **Is it possible to acclimatize to 8000m?**

This study cannot directly address this issue. Certainly the low descent mortality rates among the sherpas suggest that mortality rates amongst climbers could be lower. Because they are employed to place ropes and transport equipment, sherpas tend to spend more time at very high altitudes. But as they have been through a competitive process to win jobs as sherpas, there is a selection bias in this group. Many are born and live at high altitude (Sherpas or ethnic Tibetans) and may have adaptive advantages to high altitude. So it is difficult to tell if they are spending more time at very high altitude because they acclimate better, or if they are better acclimated because they spend more time at very high altitude, or both. Other factors, such as greater climbing experience or the tendency to climb in groups may also play a role. So the ability of lowlanders to acclimate to very high altitudes remains ill defined and needs further work.

### **Any other notes of caution?**

This is a single study – later studies, differing interpretations and advances in the understanding of high altitude physiology at extreme altitude may alter the conclusions mountaineers should draw. A catastrophic event such as a mountaineering death is often the result of the convergence of multiple factors. While we have examined the effect of high altitude illness, other factors play a role in fatalities. The mountaineer should simply use this study as one guide in assessing the many complex decisions that need to be made to ensure safety in high places.

### **Any other messages from this study?**

A lot of people have died on Everest. Most were in the prime of their lives, with families and friends left bereft. Appropriate caution is the hallmark of the elite mountaineer; the mountain will always be there next year. Climbing Everest doesn't solve global warming, create world peace, cure cancer or save lives - mountaineering is just for fun. It's not worth dying for, nor is it worth leaving others to die. Make sure to come back to those you left behind at home, and to ensure your fellow mountaineers do too. Happy and safe climbing!

