3 Wijdicks EFM. The diagnosis of brain death. *N Engl J Med* 2001; **344:** 1215–21.
4 Wu X, Fang Q, Li L, Qiu Y, Luo B. Complications associated with the apnea test in the determination of the brain death. *Chin Med J* 2008; **121:** 1169–72.
5 Lévesque S, Lessard MR, Nicole PC, et al. Efficacy of a T-piece system and a continuous positive airway pressure system for apnea testing in the diagnosis of brain death. *Crit Care Med* 2006; **34:** 2213–16.
6 al Jumah M, McLean DR, al Rajeh S, Crow N. Bulk diffusion apnea test in the diagnosis of brain death. *Crit Care Med* 1992; **20:** 1564–67.
7 Sharpe MD, Young GB, Harris C. The apnea test for brain death determination: an alternative approach. *Neurocrit Care* 2004; **1:** 363–66.
8 Shewmon DA. Chronic "brain death": meta-analysis and conceptual consequences. *Neurology* 1998; **51:** 1538–45.
9 Powner DJ, Bernstein IM. Extended somatic support for pregnant women after brain death. *Crit Care Med* 2003; **31:** 1241–49.
10 Powner DJ, Hernandez M, Rives TE. Variability among hospital policies for determining brain death in adults. *Crit Care Med* 2004; **32:** 1284–88.
11 Greer DM, Varelas PN, Haque S, Wijdicks EFM. Variability of brain death determination guidelines in leading US neurologic institutions. *Neurology* 2008; **70:** 284–89.
12 Wang MY, Wallace P, Gruen JP. Brain death documentation: analysis and issues. *Neurosurgery* 2002; **51:** 731–36.
13 Earnest MP, Beresford HR, McIntyre HB. Testing for apnea in suspected brain death: methods used by 129 clinicians. *Neurology* 1986; **36:** 542–44.

Arterial blood measurements in climbers on Mount Everest

Michael Grocott and colleagues[1] recently reported findings from arterial blood samples taken near the summit of Mount Everest, including some extraordinarily low values for partial pressure of arterial oxygen (PaO_2). The highest point on earth is near the limit of human survival because of oxygen deprivation. Evidence for critical conditions near the summit of Mount Everest comes from early expeditions beginning in 1921. In 1924, Edward Norton climbed to within 300 m of the summit without supplementary oxygen, but the first complete ascent without oxygen was not made until 1978.[2] The maximum oxygen consumption on the summit is only about 1 l/min, which is equivalent to walking slowly on level ground. In other words, almost no oxygen is available for climbing.

Current information about human physiology on Everest's summit comes mainly from the 1981 American Medical Research Expedition to Everest.[3] During this study, barometric pressure was measured, and data for alveolar gas samples showed the crucial importance of extreme hyperventilation. Hyperventilation allowed partial pressure of alveolar oxygen (PAO_2) to be defended at about 35 mm Hg (1 mm Hg=0·133 kPa) in the face of an inspired partial pressure of oxygen of only 43 mm Hg; the calculated PaO_2 was about 30 mm Hg.[3] Similar PaO_2 values were reported in two low-pressure-chamber studies,[4,5] although these data suggested that participants were not as well acclimatised to the low oxygen as were mountain climbers. Therefore great interest arose in obtaining arterial blood on or near the summit of Mount Everest, and possible strategies have been proposed.[6]

The Caudwell Xtreme Everest Expedition of 2007, reported on by Grocott and colleagues, was an ambitious and successful undertaking. Although weather conditions were too severe for arterial blood to be taken on the summit, samples were obtained from four climbers at 8400 m (barometric pressure 272 mm Hg; figure). A small tent was erected and right femoral arterial blood samples were taken and transported rapidly in an ice-water slurry to 6400 m, where analytical equipment was located. This method of transportation preserves blood gases almost unchanged for several hours.[6]

Two climbers had PaO_2 values of 29·5 and 28·7 mm Hg, which is in line with calculations from alveolar gas values and measurements in low-pressure chambers.[4,5] However, the other two climbers had much lower values of 21·0 and 19·1 mm Hg. The partial pressure of arterial carbon dioxide ($PaCO_2$) was 10·3–15·7 mm Hg, with the climbers who had the lowest PaO_2 having the highest $PaCO_2$ values. Blood pH was between 7·45 and 7·60, and

Caudwell Xtreme Everest

Figure: **Site on Mount Everest at which arterial blood samples were taken**
Site was at altitude of 8400 m. Photograph shows tent and thermos bottle in which samples were transported.

the calculated alveolar–arterial oxygen difference varied between 2·9 and 7·8 mm Hg. Arterial oxygen saturation was 69·7% and 68·1% in two climbers but was lower in the other two (43·7% and 34·4%). The two highest saturation values accord with measurements from other studies,[4,7,8] but the other results are unexpectedly low. The great variability in the data is difficult to explain but hardly detracts from the extraordinary achievement of obtaining arterial blood gases under these arduous conditions.

The results from two of the climbers are broadly in line with previous expectations, but the other two had values—particularly for PaO_2 and oxygen saturation—that hardly seem compatible with the ability to climb the mountain. Although the arterial blood was taken while the climbers were breathing ambient air, they all used supplementary oxygen during the ascent.

These new data throw light on human physiology under challenging hypoxic conditions. It would be intriguing to know more about the other characteristics of these climbers, including their ventilatory response to hypoxia. Does this study mean that we should relax our attitude to trying to maintain adequate values for oxygenation in the intensive-care unit? I think not, but the findings remind us of the resilience of the human body.

John B West
Department of Medicine, University of California San Diego, La Jolla, CA 92093, USA
jwest@ucsd.edu

I declare that I have no conflicts of interest.

1 Grocott MPW, Martin DS, Levett DZH, McMorrow R, Windsor J, Montgomery HE, for the Caudwell Xtreme Everest Research Group. Arterial blood gases and oxygen content in climbers on Mount Everest. *N Engl J Med* 2009; **360:** 140–49.
2 West JB. High life: a history of high-altitude physiology and medicine. New York: Oxford University Press, 1998.
3 West JB, Hackett PH, Maret KH, et al. Pulmonary gas exchange on the summit of Mount Everest. *J Appl Physiol* 1983; **55:** 678–87.
4 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.
5 Richalet J-P, Robach P, Jarrot S, et al. Operation Everest III (COMEX '97): effects of prolonged and progressive hypoxia on humans during a simulated ascent to 8,848 m in a hypobaric chamber. *Adv Exp Med Biol* 1999; **474:** 297–317.
6 Catron TF, Powell FL, West JB. A strategy for determining arterial blood gases on the summit of Mt. Everest. *BMC Physiol* 2006; **6:** 3.
7 Winslow RM, Samaja M, West JB. Red cell function at extreme altitude on Mount Everest. *J Appl Physiol* 1984; **56:** 109–16.
8 Peacock AJ, Jones PL. Gas exchange at extreme altitude: results from the British 40th Anniversary Everest Expedition. *Eur Respir J* 1997; **10:** 1439–44.

Influenza begs many questions

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"Will we all die?" is just one question I was asked by the mass media after the outbreak of influenza A (H1N1) in Mexico. The answer (given wisely by another commentator) is "yes we will, but probably not from flu". Outside Mexico, the strain of H1N1 seems to cause mild disease, no worse than seasonal influenza, with low mortality. Nevertheless, cases have been confirmed in 20 countries on four continents, and there is evidence of chains of transmission in two countries in the WHO American (Mexico and USA) and European (Spain and UK) regions. WHO is reported to be considering raising the pandemic alert level to six, which will signify that we are in the midst of the first influenza pandemic since 1968.

Fortunately, advances in medicine over the past 40 years, including availability of antiviral drugs, mean we are now in a better position to manage a pandemic than ever before. However, if a pandemic is declared, an urgent issue will be whether to continue the practice of giving oseltamivir prophylactically to contacts of confirmed cases. At four schools in the UK where pupils have confirmed infection, prophylactic oseltamivir has been issued to hundreds of fellow students. Although this policy follows Health Protection Agency advice, I wonder about sustainability. At a *Lancet* conference on pandemic influenza in 2006, Frederick Hayden (University of Virginia, USA) stated that for a group of 1000 people 16 times as much antiviral was needed to prevent influenza infection as to treat it. By this measure, the UK has stockpiles of oseltamivir sufficient to treat 30 million people (about half the population) but to prevent infection in only 1·9 million.

On May 17, *The Lancet* will be supporting a late-breaker session on influenza A (H1N1) at the European Conference on Clinical Microbiology and Infectious Diseases in Helsinki, Finland. We plan to make freely available from thelancet.com website a video recording of this session, plus additional content relevant to the current outbreak taken from the *Lancet* journals and others published by Elsevier.

John McConnell
The Lancet Infectious Diseases, London NW1 7BY, UK