The financial crisis comes at a time when commitment to global health has never been higher. It comes in the midst of the most ambitious drive in history to tackle the root causes of poverty, reduce the gaps in health outcomes, and ensure that the benefits of social and economic progress are more evenly distributed. A fair, efficient, and affordable system of health care is our best insurance policy, our best route to health security.

Investment in health systems and services is investment in human capital. Healthy human capital is the foundation for productivity and prosperity. Equitable distribution of health care and equity in the health status of populations is the foundation for social cohesion, which is our best protection against social unrest.

At the end of 2007, nearly 3 million people in low-income and middle-income countries were receiving antiretroviral therapy for AIDS,[11] and we should thank the G8 for its contribution to this achievement. But, if funding dries up in this or other areas, the health sector can produce fairly precise estimates of what will happen, measured by the number of lives lost. Human suffering and misery are not as easily calculated, but our common humanity should make us care on this count as well. I believe that when the G8 takes on a health issue, they give a human face to the political leadership that our world so greatly needs.

The net result of all our international policies should be to improve the quality of life for as many of the world's people as possible. Greater equity in the health status of populations, within and among countries, should be regarded as a key measure of how we, as a civilised society, are making progress. Strengthened health systems, ideally based on primary health care, are indeed the route to greater efficiency and fairness in health care and greater security in the health sector and beyond.

Margaret Chan
WHO, CH-1211 Geneva 27, Switzerland
chanm@who.int

I am the Director-General of WHO.

1 WHO. The world health report 2008—primary health care: now more than ever. 2008. http://www.who.int/whr/2008/en/index.html (accessed Dec 17, 2008).
2 African Development Bank. Gender, poverty and environmental indicators on African Countries, vol 9. 2008. http://www.afdb.org/pls/portal/docs/PAGE/ADB_ADMIN_PG/DOCUMENTS/STATISTICS/GENDER%202008_WEB.PDF (accessed Dec 17, 2008).
3 UNICEF. The state of the world's children 2008. 2008. http://www.unicef.org/sowc08/docs/sowc08.pdf (accessed Dec 17, 2008).
4 WHO. Monitoring the achievement of the health-related Millennium Development Goals. 2008. http://www.who.int/gb/ebwha/pdf_files/EB124/B124_10-en.pdf (accessed Dec 17, 2008).
5 Maxwell D, Levin C, Armar-Klemesu M, et al. Urban livelihoods and food and nutrition security in Greater Accra, Ghana. 2000. https://www.who.int/nutrition/publications/WHO_multicountry_%20study_Ghana.pdf (accessed Dec 17, 2008).
6 Drewnowski A, Specter SE. Poverty and obesity: the role of energy density and energy costs. *Am J Clin Nutr* 2004; **79:** 6–16.
7 Stern N. The economics of climate change: the Stern review. 2007. http://www.hm-treasury.gov.uk/stern_review_report.htm (accessed Dec 17, 2008).
8 Cruz RV, Harasawa H, Lal M, et al, eds. Intergovernmental Panel on Climate Change, 4th assessment report: impacts, adaptation and vulnerability. 2007: 469–506. http://www.ipcc.ch/ipccreports/ar4-wg2.htm (accessed Dec 17, 2008).
9 Jack A. From symptom to system. *Financial Times* Sept 28, 2007. http://www.ft.com/cms/s/0/2318ea9c-6d60-11dc-ab19-0000779fd2ac.html?nclick_check=1 (accessed Dec 17, 2008).
10 Freedman LP, Waldman RJ, de Pinho H, et al. Who's got the power? Transforming health systems for women and children. London: Earthscan, 2005.
11 WHO. Towards universal access: scaling up priority HIV/AIDS interventions in the health sector. 2008. http://www.who.int/hiv/mediacentre/2008progressreport/en/index.html (accessed Dec 17, 2008).

Certification of brain death: take care

Use of neurological criteria to define death has become accepted worldwide[1] and is implemented widely in support of transplantation programmes and family directives. As a fundamental principle, moral abhorrence of making a false diagnosis of death with neurological criteria should be absolute. This standard should persuade all doctors that strict adherence to testing that does not risk further injury to the patient and provides an infallible conclusion is mandatory.

Eelco Wijdicks and colleagues[2] recently provided useful information about the process and results of apnoea testing at Mayo Clinic hospitals during clinical assessment of brain death. This team analysed implementation and outcomes of a procedure recommended by the American Academy of Neurology[3] to assess medullary respiratory response after induced hypercapnia. Of 228 patients investigated retrospectively, concurrent haemodynamic instability or hypoxaemia prevented testing in 7%, and these disorders arose during the assessment in another 3%.

These data are similar to those of other studies in which the same or a different technique was used to measure

	Method	Events and comments
Wu et al[4]	Prospective, 93 patients; preoxygenation; O_2 catheter (6 L/min) via endotracheal tube; basal normocapnia; $PaCO_2$ goal >8·0 kPa	21% of patients had diminished systolic blood pressure (<90 mm Hg), PaO_2<8·0 kPa, or both; more hypoxaemia when pretest PaO_2<26·7 kPa; no change in heart rate; 1·6% of tests were stopped
Lévesque et al[5]	Prospective, 20 patients; O_2 (6 L/min) via catheter in endotracheal tube *vs* T-piece (100% O_2) *vs* CPAP; preoxygenation; pretest $PaCO_2$ 4·7–6·0 kPa and PaO_2>26·7 kPa; $PaCO_2$ endpoint >8·0 kPa	Less reduction in PaO_2 with CPAP; 15% of tests stopped due to hypotension or hypoxaemia
al Jumah et al[6]	Prospective, 24 patients; preoxygenation and normalisation of $PaCO_2$; patient attached to ventilator but ventilation stopped; bulk O_2 flow past endotracheal tube at 40 or 60 L/min; 10-min test; PaO_2 goal >8·0 kPa	Test stopped (4·1%) for hypotension; mean increase in $PaCO_2$ was 0·5 kPa
Sharpe et al[7]	Retrospective, 60 patients; preoxygenation; CO_2 administered via ventilator; end-tidal CO_2 monitored, ventilation rate decreased to reach exhaled CO_2 and blood pH goals	Hypoxaemia or hypotension did not occur

CPAP=continuous positive airway pressure. $PaCO_2$=partial pressure of carbon dioxide in arterial blood. PaO_2=partial pressure of oxygen in arterial blood. 1 kPa=7·5 mm Hg=7·5 torr.

Table: **Representative methods and results of apnoea testing**

apnoea. Representative findings (table) corroborate potential harm to the patient during testing and reiterate the importance of preoxygenation, induction of normocapnia before assessment, and assurance that the oxygen catheter is smaller than the diameter of the endotracheal tube (if that method is used).[4-7] The intention of all approaches is to safely allow predetermined levels of hypercapnia and acidosis to develop without induction of hypoxaemia or barotrauma. Absent spontaneous-breathing efforts at this maximum physiological stimulus (usually, partial pressure of carbon dioxide in arterial blood >8·0 kPa) confirms loss of medullary respiratory function.

Indirect validation that diagnosis of brain death is equivalent to patient's death comes from several studies and case reports of about 70 patients who were supported somatically after certification of brain death.[8,9] None of these patients awoke or showed neurological improvement during periods of observation, which included up to 107 days for pregnant brain-dead women[9] and several years in another description.[8]

Policies and guidelines for assessment and procedures for testing established by governmental statute, legal precedent, or individual hospitals vary[1,10,11] and deviate from recommendations made by authoritative groups.[3] In an international survey of 80 countries, a national standard was found in 69% but apnoea testing was required in only 59%. Further ancillary tests to confirm the clinical diagnosis of brain death were needed in only 40%, and other important differences in processes for certification were identified.[1] A survey of 106 hospital policies in the USA showed much variability, including failure to identify exclusionary conditions for testing (eg, drugs, hypothermia) in 12% and no required apnoea testing in 4%; furthermore, 18% did not list any testing (eg, intracranial blood flow, electroencephalogram) to confirm a clinical diagnosis of brain death.[10]

Clinicians do not always follow an established policy or provide appropriate documentation.[12,13] Surveys or chart reviews showed that doctors sometimes failed to document specifics of clinical examinations, omitted criteria demanded by local policy, or did not exclude pre-existing confounding circumstances. Of particular relevance to this discussion of apnoea testing, Earnest and colleagues[13] surveyed 129 neurologists and noted that 12% did not do apnoea testing during brain-death examinations at all and 65% observed the patient off the ventilator for 3 min or less.

As diagnosis of brain death and the processes and procedures for its confirmation have become more frequent in the intensive-care unit, clinical practice must not be permitted to become careless, abbreviated, or casual. The many reported cases of brain death diagnosed inappropriately or incorrectly and the history of rescued patients cautions that commonplace is not a reason for carelessness.

David J Powner
Departments of Neurosurgery and Internal Medicine, University of Texas Medical School-Houston, Houston, TX 77030, USA
david.j.powner@uth.tmc.edu

I declare that I have no conflicts of interest.

1 Wijdicks EFM. Brain death worldwide: accepted fact but no global consensus in diagnostic criteria. *Neurology* 2002; **58:** 20–25.
2 Wijdicks EFM, Rabinstein AA, Manno EM, Atkinson JD. Pronouncing brain death: contemporary practice and safety of the apnea test. *Neurology* 2008; **71:** 1240–44.

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.