research article

Brain death and true patient care

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In this article

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Abstract

The "brain death" standard as a criterion of death is closely associated with the need for transplantable organs from heart-beating donors. Are all of these potential donors really dead, or does the documented evidence of patients destined for organ harvesting who improve, or even recover to live normal lives, call into question the premise underlying "brain death"? The aim of this paper is to re-examine the notion of "brain death," especially its clinical test-criteria, in light of a broad framework, including medical knowledge in the field of neuro-intensive care and the traditional ethics of the medical profession. I will argue that both the empirical medical evidence and the ethics of the doctor–patient relationship point to an alternative approach toward the severely comatose patient (potential brain-dead donor).

Lay Summary: Though legally accepted and widely practiced, the "brain death" standard for the determination of death has remained a controversial issue, especially in view of the occurrence of

"chronic brain death" survivors. This paper critically re-evaluates the clinical test-criteria for "brain death," taking into account what is known about the neuro-critical care of severe brain injury. The medical evidence, together with the understanding of the moral role of the physician toward the patient present before him or her, indicate that an alternative approach should be offered to the deeply comatose patient.

Keywords: Brain death, Severe brain injury, Apnea test, Neuro-intensive care, Hippocratic ethos,

Introduction

Death is an irreversible event that occurs instantly, distinct from the process of dying. The empirical assessment of any given event, however, "requires a certain time interval" (Ramellini 2009 Ramellini, Pietro. 2009. Death in the biological literature of life. In *What is death?: A scientific, philosophical and theological exploration of life's end*, ed. Alfonso Aguilar, 21–65. Città del Vaticano: Libreria Editrice Vaticana., 60). Consequently, the very moment of death cannot be determined. In his address to the 18th International Congress of the Transplantation Society, St. John Paul II reiterated this universal truth as follows: "the death of the person … is an event *no scientific technique or empirical method can identify directly*" (emphasis original) (John Paul II 2000 John Paul II, Pope. 2000. Address to the 18th International congress of the transplantation society. August 29. http://www.vatican.va/holy_father/john_paul_ii/speeches/2000/jul-sep/documents/hf_jp-ii_spe_20000829_transplants_en.html., no. 4). More than a century earlier, Edgar Allan Poe had also stated the same truth in his short story "The Premature Burial." Expressing the widespread fear of being buried alive, Poe wrote, "The boundaries which divide Life from Death, are at best shadowy and vague. Who shall say where the one ends, and where the other begins?" (Poe 1966 Poe, Edgar Allan. 1966. *Complete stories and poems of Edgar Allan Poe*. Garden City, NY: Doubleday., 261). The fear of being buried alive is nothing but a manifestation of the well-known "universal fear of a premature, mistaken declaration of death" (Childress 2014 Childress, James F. 2014. Difficulties of determining death: What should we do about the "dead donor rule"? *Zif-Mitteilungen*, no. 1: 28–39. http://www.uni-bielefeld.de/(cen,en)/ZIF/Publikationen/Mitteilungen/Ausgaben/2014-1.pdf., 28). Today, advanced technology and scientific progress have all but eliminated the possibility of being buried alive. As will be shown below, the same cannot be said, however, about the risk of a premature declaration of death, especially when viewed in the context of organ procurement for transplantation.

Currently, the great majority of organs for transplantation still come from heart-beating donors (that is, donation after the declaration of "brain death"), even after the introduction of donation after "circulatory death" in the early 1990s (Steinbrook 2007 Steinbrook, Robert. 2007. Organ donation after cardiac death. *New England Journal of Medicine* 357, no. 3: 209–13. doi: 10.1056/NEJMp078066, 211). The inherent link between organ transplantation and "brain death" is self-evident,[1] despite repeated efforts to deny it with the claim that the concept of "brain death" only came about as "a consequence of the development of intensive care" and not because of the need for organs (Kerridge et al. 2003 Kerridge, I.H., P. Saul, M. Lowe, J. McPhee, and D. Williams. 2003. Commentary on: A definition of human death should not be related to organ transplants. *Journal of Medical Ethics* 29, no. 3: 202.; Machado 2003 Machado, C. 2003. A definition of human death should not be related to organ transplants. *Journal of Medical Ethics* 29, no. 3: 201–2. doi: 10.1136/jme.29.3.201, 201).[2] The link already existed even before the publication of the seminal report of the Harvard Medical School Ad Hoc Committee (1968) Harvard Medical School, Ad Hoc Committee to Examine the Definition of Brain Death. 1968. A definition of irreversible coma. *Journal of the American Medical Association* 205, no. 6: 337–40. doi: 10.1001/jama.1968.03140320031009. At the 1966 international symposium on "Ethics in Medical Progress: With Special Reference to Transplantation" sponsored by the Ciba Foundation in London, several discussions were held on the issue of equating *le coma dépassé* (that is, irreversible coma) with death for the purpose of procuring more viable organs, thereby circumventing the poor quality of organs retrieved from true cadavers (Mollaret and Goulon 1959 Mollaret, P., and M. Goulon. 1959. Le coma dépassé. *Revue Neurologique* 101: 3–15.).[3] More importantly, the earlier drafts

of the Harvard report and memos between the committee members reveal an explicit connection between "brain death" and the need for organs.[4] For instance, the advanced manuscript draft of June 3, 1968, contains the following passage:

With increased experience and knowledge and development in the field of transplantation, there is great need for the tissues and organs of the hopelessly comatose in order to restore to health those who are still salvageable. (quoted in Giacomini 1997 Giacomini, Mita. 1997. A change of heart and a change of mind? Technology and the redefinition of death in 1968. *Social Science & Medicine* 44, no. 10: 1465–82. doi: 10.1016/S0277-9536(96)00266-3, 1475)

Evidently, the wording in the above passage reflects a pragmatic-utilitarian philosophy in which the principle of utility, aiming at maximizing the good for the greatest number of individuals, prevails. Decision-making processes and actions can then be justified by "cost-benefit" analyses or "the ends justify the means." The pragmatic-utilitarian motives of the Harvard Committee, however toned down they might be, remained apparent in the opening paragraph of its 1968 report. They were brought out more explicitly in the subsequent discourses of Beecher, the committee's chairman, however.[5]

In the discussion on "brain death," most of the attention has been devoted to the legal or philosophical aspects. Where the medical aspect is touched upon, the focus has been on the clinical criteria for brain death,[6] or the management of organ donors, that is, the care of organs prior to their surgical harvesting.[7] In other words, once a patient is labeled "brain dead," then his or her status becomes nothing more than that of an "organ container," albeit a special one which is connected to a ventilator, and in which the heart continues to beat, thereby maintaining the circulation of oxygenated blood to the various organs soon-to-be harvested. Such a reality does not convey the image of a corpse, one ready to be cremated or buried, however.

Therefore, the purpose of this essay is to seriously re-examine the status of the potential heart-beating organ donor: is he or she really a corpse? Or is he or she a very debilitated patient with severe brain injury, whose condition can potentially improve or even return to a full normal life, *if* he or she is given: (i) timely and aggressive neuro-intensive treatment, and (ii) ample time for the slow recovery of brain functions, instead of being quickly declared brain-dead (during the first few days of acute brain injury) and destined for organ harvesting? The essay opens with a survey of patients who have survived "brain death," a phenomenon which seriously contradicts the assertion that "brain death" equals death. The phenomenon of brain-dead survivors leads, therefore, to the necessity of a critical re-evaluation of the clinical criteria for "brain death." From this, it will become clear that the severely brain-injured patient, so-called "brain dead," deserves a different medical approach, one that would both respect his or her dignity and cohere better with the *telos* of the medical profession, and consequently, with the vocation of a Christian physician.

Survivors of "Brain Death"

For decades, the concept of "brain death," since its inception in 1968 as the neurological standard for determining death, has been the basis for current policies of organ harvesting from heart-beating "cadavers." Despite the widespread medical and legal acceptance of "brain death," there have been persistent misgivings that many heart-beating patient-donors have been declared "brain-dead" when, in fact, they are not truly dead. Indeed, the contemporary history of medicine is not lacking in "modern day versions of Poe's horror story" (Childress 2014 Childress, James F. 2014. Difficulties of determining death: What should we do about the "dead donor rule"? *Zif-Mitteilungen*, no. 1: 28–39. http://www.uni-bielefeld.de/(cen,en)/ZIF/Publikationen/Mitteilungen/Ausgaben/2014-1.pdf., 29), namely the accounts of "brain-dead" patients who narrowly escaped the fatal ordeal of organ harvesting. A common pattern runs through most of these accounts: the patient is invariably a non-elderly person, constitutionally healthy, who became deeply comatose and ventilator-dependent because of a sudden and severe brain injury.[8] Within 24 to 48 hours after the accident, he or she was

declared dead according to the neurological standard and destined for the soon-to-be performed organ removal. Among the many narrow "escape" stories that came to the attention of the media, one can think of: (i) the recovery of a 21-year-old man, who in 2008 was declared "brain-dead" thirty-six hours after his accident (Morales 2008 Morales, Natalie. 2008. "Dead" man recovering after ATV accident. NBC News. March 23. http://www.nbcnews.com/id/23768436/ns/dateline_nbc-newsmakers/t/dead-man-recovering-after-atv-accident.); (ii) the recovery of a 41-year-old woman, so-called "brain-dead," who unexpectedly woke up in the operating room just as her organs were about to be removed in 2009 (O'Brien and Mulder 2013 O'Brien, John, and James T. Mulder. 2013. St. Joe's "dead" patient awoke as docs prepared to remove organs. Syracuse.com. July 7. http://www.syracuse.com/news/index.ssf/2013/07/st_joes_fined_over_dead_patien.html.); and (iii) the recovery of a 19-year-old woman, who in 2011 also suddenly woke up while the doctors gathered around her bedside were discussing her presumed "brain death" and possible organ donation (Malm 2012 Malm, Sara. 2012. The girl who wouldn't die: Incredible story of the 19-year-old who woke up as doctors were preparing to harvest her organs. Daily Mail (UK). October 17. http://www.dailymail.co.uk/news/article-2219085/Carina-Melchior-The-girl-wouldnt-die-Miracle-Danish-girl-woke-doctors-prepared-organs-donated.html.). Recently, two cases of reversible "brain death" have been reported from academic tertiary hospitals (Joffe et al. 2009 Joffe, Ari R., Hanna Kolski, Jonathan Duff, and Allan R. deCaen. 2009. A 10-month-old infant with reversible findings of brain death. *Pediatric Neurology* 41, no. 5: 378–82. doi: 10.1016/j.pediatrneurol.2009.05.007; Webb and Samuels 2011 Webb, Adam C., and Owen B. Samuels. 2011. Reversible brain death after cardiopulmonary arrest and induced hypothermia. *Critical Care Medicine* 39, no. 6: 1538–42. doi: 10.1097/CCM.0b013e3182186687).

In addition to the above-mentioned dramatic, spontaneous full recovery from "brain death," there are also many well-documented cases of "brain-death" survivors. Though pronounced dead according to the neurological standard, these patients continued to live, albeit in the severe disabling state of chronic "brain death." The following is not an unlikely scenario: a severely brain-injured patient was declared "brain-dead"; the family, however, declined organ donation; the patient did not die, that is, he or she did not have cardiac arrest, contrary to the insistent claim that imminent asystole necessarily follows "brain death."[9] After a few weeks, once the initial hemodynamic instability subsides, gastrointestinal motility returns along with spinal hyperreflexia, and the patient continues to live on for weeks and months without aggressive medical intervention, requiring only a mechanical ventilator, tube feeding, and basic nursing care (Shewmon 1998a Shewmon, D. Alan. 1998a. "Brainstem death," "brain death" and death: A critical re-evaluation of the purported equivalence. *Issues in Law & Medicine* 14, no. 2: 125–45., 136).

Shewmon (1998b) Shewmon, D. Alan. 1998b. Chronic "brain death": Meta-analysis and conceptual consequences. *Neurology* 51, no. 6: 1538–45. doi: 10.1212/WNL.51.6.1538 reported a collection of 175 "brain-dead patients," whose survival extended well beyond the few days (48–72 hours) claimed to be the maximum limit after the declaration of "brain death." There have been additional cases since then, in particular the well-publicized McMath case, whose clinical and laboratory data no longer indicate "brain death."[10] Most notable in Shewmon's (2001) Shewmon, D. Alan. 2001. The brain and somatic integration: Insights into the standard biological rationale for equating "brain death" with death. *Journal of Medicine and Philosophy* 26, no. 5: 457–78. doi: 10.1076/jmep.26.5.457.3000 collection is the fact that children with chronic "brain death" underwent proportional physical growth as well as sexual maturation; features indicative of neuroendocrine function in the brain. One of the children, the longest survivor, lived on for twenty years after having been declared "brain-dead" at age four from acute meningitis (Shewmon 2007 Shewmon, D. Alan. 2007. Mental disconnect: "physiological decapitation" as a heuristic for understanding "brain death". In *The signs of death*, ed. M.S. Sorondo, 292–333. Vatican City: Pontifical Academy of Sciences., 308). If "brain death" equals death, which implies that the "brain-dead" patient is a corpse, then the corpses of "brain-dead" children-survivors certainly demonstrate unusual properties. These are none other than holistic/integrative properties (each emerging from the mutual interaction of the parts of a whole),

which account for the integrative unity and survival of patients with chronic "brain death." Even though these patients have lost some of the emergent holistic functions, namely consciousness and spontaneous breathing, they still retain a whole host of other important holistic/integrative properties including diverse homeostatic activities, elimination of bodily wastes, wound healing, inflammatory and immunological responses against infections, physical growth and maturation, and the capacity for successful gestation, among others.[11] The empirical medical evidence certainly does not support the prevailing theory that the brain is the master organ responsible for the integration of the body. This rationale was advanced in Bernat, Culver, and Gert (1981 Bernat, James L., Charles M. Culver, and Bernard Gert. 1981. On the definition and criterion of death. *Annals of Internal Medicine* 94, no. 3: 389–94. doi: 10.7326/0003-4819-94-3-389), and Bernat (2006 Bernat, James L. 2006. The whole-brain concept of death remains optimum public policy. *Journal of Law, Medicine & Ethics* 34, no. 1: 35–43. doi: 10.1111/j.1748-720X.2006.00006.x). This theory has its origins in Loeb's mechanistic conception of life (Loeb 1912 Loeb, Jacques. 1912. *The mechanistic conception of life*. Chicago: University of Chicago Press.), which has been recognized to be scientifically flawed, and has been supplanted by theories which better reflect biological reality, namely the systems perspective of life and the notion of autopoiesis (Varela 1979 Varela, Francisco J. 1979. *Principles of biological autonomy*. New York: North Holland., 3–60; Maturana, Varela, and Beer 1980 Maturana, Humberto R., Francisco J. Varela, and Stafford Beer. 1980. *Autopoiesis and cognition: The realization of the living*. Dordrecht: Reidel.).

In addition to the reported survivors in the "brain death" literature, the data from the neuro-intensive care literature regarding the survival of brain-injured patients admitted with Glasgow Coma Scale 3 (GCS 3) is also revealing.[12] Most of the victims of severe head trauma are children and young to middle-aged adults who, prior to their accident, were constitutionally healthy (Dunn and Smith 2008 Dunn, Juliet, and Martin Smith. 2008. Critical care management of head injury. *Anaesthesia & Intensive Care Medicine* 9, no. 5: 197–201. doi: 10.1016/j.mpaic.2008.03.004, 197; Maas, Stocchetti, and Bullock 2008 Maas, Andrew I.R., Nino Stocchetti, and Ross Bullock. 2008. Moderate and severe traumatic brain injury in adults. *Lancet Neurology* 7, no. 8: 728–41. doi: 10.1016/S1474-4422(08)70164-9, 728; Werner and Engelhard 2007 Werner, C., and K. Engelhard. 2007. Pathophysiology of traumatic brain injury. *British Journal of Anaesthesia* 99, no. 1: 4–9. doi: 10.1093/bja/aem131, 4). This age group coincides with the heart-beating donor age group that provides the greater bulk of organ donation. A recent study of 3,306 trauma patients admitted with GCS three showed an overall survival rate of 58.2 percent, more than twice the survival rate noted in the 1980s thanks to the advances in neuro-intensive care (Ley et al. 2011 Ley, Eric J., Morgan A. Clond, Omar N. Hussain, Marissa Srour, James Mirocha, Marko Bukur, Dan R. Margulies, and Ali Salim. 2011. Mortality by decade in trauma patients with Glasgow coma scale 3. *American Surgeon* 77, no. 10: 1342–5.). In this study, patients in the fourth and fifth decades achieved the highest survival rate (64%). Reports of smaller groups of patients from Germany and Japan showed similar encouraging results. The study from Nihon University Hospital in Japan used intracranial pressure (ICP)-targeted therapy with hypothermia on twenty cases of traumatic brain injury (TBI) and twelve cases of post-cardiac arrest, all of whom had an initial GCS of 3 or 4, plus bilateral fixed and dilated pupils. Fourteen of the TBI group, and six of the post-cardiac arrest group "returned to normal daily life, with their verbal communication restored, except in one patient" (Watanabe 1997 Watanabe, Yoshio. 1997. Once again on cardiac transplantation: Flaws in the logic of the proponents. *Japanese Heart Journal* 38, no. 5: 617–24. doi: 10.1536/ihj.38.617, 619). The study from Regensburg University Hospital in Germany on ten patients with severe head injury (all unresponsive to pain, seven with GCS 3, two with GCS 4, one with GCS 6) revealed similar results: following moderate hypothermia therapy, two patients with GCS 3 died of multi-organ failure, seven "returned to their previous occupation (good recovery) [and] one patient survived severely disabled" (Metz et al. 1996 Metz, Christoph, Matthias Holzschuh, Thomas Bein, Christian Woertgen, Anton Frey, Irmgard Frey, Kai Taeger, and Alexander Brawanski. 1996. Moderate hypothermia in patients with severe head injury: Cerebral and extracerebral effects. *Journal of Neurosurgery* 85, no. 4: 533–41. doi: 10.3171/jns.1996.85.4.0533, 536). It is thus possible, with the current state of the art neuro-intensive care to achieve a survival rate of 50 to 70 percent, at least in

selected cases. In view of such an encouraging survival rate, and the continuously improving medical technology, it has been concluded that "aggressive care is indicated for patients who present to the emergency department with GCS 3" (Ley et al. 2011 Ley, Eric J., Morgan A. Clond, Omar N. Hussain, Marissa Srour, James Mirocha, Marko Bukur, Dan R. Margulies, and Ali Salim. 2011. Mortality by decade in trauma patients with Glasgow coma scale 3. *American Surgeon* 77, no. 10: 1342–5., 1344). In their presentations, defenders of "brain death" have not taken into consideration the advances in neuro-intensive care, however.

The "brain-death" literature and the neuro-intensive care literature use different terminologies to designate similar groups of patients: for the first, the terminology "heart-beating" or "brain-dead" donor; and for the second, the terminology "severe brain injury" (SBI). Like any other vital organ in the body, the function of the brain and its organic survival depend on an adequate supply of blood flow to provide the proper levels of oxygen and other necessary elements. Severe brain injury encompasses a wide range of etiological injuries which result in a sharp decrease in blood flow or oxygen level to the brain.[13] Excluding malignancies and inflammatory disorders affecting the brain, such injuries can be conceptually grouped as follows: (i) failure of the "pump," namely, a cardiac arrest;[14] (ii) failure in the "delivery system," which can be precipitated by catastrophic events such as extensive hemorrhage,[15] thromboembolism to a major cerebral artery, or a ruptured cerebral aneurysm; and (iii) direct injurious impact to the head from an external source, such as in road or sports accidents, referred to as traumatic brain injury (TBI). Overall, cardiac arrest and TBI comprise the leading causes of SBI. The TBI category "is a common cause of death and neurological disabilities in young people" (Polderman 2008 Polderman, K.H. 2008. Hypothermia and neurological outcome after cardiac arrest: State of the art. *European Journal of Anaesthesiology* 25, Suppl 42: 23–30. doi: 10.1017/S026502150700333X, 1955), whereas the former portends a high rate of mortality (70%) and morbidity "despite advances in cardiopulmonary resuscitation and post-cardiac arrest care" (Stevens and Sutter 2013 Stevens, Robert D., and Raoul Sutter. 2013. Prognosis in severe brain injury. *Critical Care Medicine* 41, no. 4: 1104–23. doi: 10.1097/CCM.0b013e318287ee79, 1104). In any of the above categories of SBI, the clinical outcome depends on: (i) the type of precipitating etiology (primary injury); (ii) the promptness and efficacy of the medical intervention; (iii) the presence of concomitant injuries; and (iv) the general constitution of the patient prior to the injury (namely his or her age and comorbidities). Concomitant multi-organ injury (e.g., in road accidents) or multi-organ failure (e.g., in post-cardiac arrest syndrome) produce multiple physiological instabilities such as hypotension, pyrexia, and coagulopathy, among others (Maas, Roozenbeek, and Manley 2010 Maas, Andrew I.R., Bob Roozenbeek, and Geoffrey T. Manley. 2010. Clinical trials in traumatic brain injury: Past experience and current developments. *Neurotherapeutics* 7, no. 1: 115–26. doi: 10.1016/j.nurt.2009.10.022, 115; Neumar et al. 2008 Neumar, Robert W., Jerry P. Nolan, Christophe Adrie, Mayuki Aibiki, Robert A. Berg, Bernd W. Böttiger, Clifton Callaway, Robert S.B. Clark, Romergryko G. Geocadin, Edward C. Jauch, Karl B. Kern, Ivan Laurent, W.T. Longstreth, Raina M. Merchant, Peter Morley, Laurie J. Morrison, Vinay Nadkarni, Mary Ann Peberdy, Emanuel P. Rivers, Antonio Rodriguez-Nunez, Frank W. Sellke, Christian Spaulding, Kjetil Sunde, and Terry Vanden Hoek. 2008. Post-cardiac arrest syndrome: Epidemiology, pathophysiology, treatment, and prognostication. *Circulation* 118, no. 23: 2452–83. doi: 10.1161/CIRCULATIONAHA.108.190652, 2456). These, in turn, increase the risk of further brain damage and worsen the patient's outcome. Similarly, Shewmon's meta-analysis of brain-dead patients shows that those with multisystem insults "deteriorate[d] quickly to asystole despite aggressive therapy," in contrast to those who survived longer than six weeks (Shewmon 1998b Shewmon, D. Alan. 1998b. Chronic "brain death": Meta-analysis and conceptual consequences. *Neurology* 51, no. 6: 1538–45. doi: 10.1212/WNL.51.6.1538, 1543–4). Of note is that,

in a chapter on head injury, multisystem derangements are interpreted as therapeutic challenges to keep a critically injured patient alive, whereas in a typical chapter on BD ["brain death"] the same derangements are cited as evidence that the patient has already died. (Shewmon 1998b Shewmon, D. Alan. 1998b. Chronic "brain death": Meta-analysis and conceptual consequences. *Neurology* 51, no. 6: 1538–45. doi: 10.1212/WNL.51.6.1538, 1544)

In other words, from the viewpoint of neuro-intensive care, patients with a potential good outcome are those with minimal hemodynamic instability or multisystem disturbances; but from the standpoint of organ transplantation, such patients are the best organ donors because of their healthy organs, especially since "cardiac stability [is] a relative *requirement* for heart donation" (emphasis original) (Shewmon 1998b Shewmon, D. Alan. 1998b. Chronic "brain death": Meta-analysis and conceptual consequences. *Neurology* 51, no. 6: 1538–45. doi: 10.1212/WNL.51.6.1538, 1544).[16]

A crucial factor affecting the clinical outcome of the severely brain-injured patient is the timeliness and intensity (aggressiveness) of the neuro-intensive intervention. It would be naïve to think that this is not influenced by the general orientation/conviction of the medical staff in charge or that of the medical center itself. The few unfortunate stories that made it to publication, in particular the case reported by Coimbra (2009b) Coimbra, Cicero Galli. 2009b. Are "brain dead" (or "brain-stem dead'') patients neurologically recoverable? In *Finis vitae: "brain death" is not true death*, ed. Roberto De Mattei, and Paul A. Byrne, 313–78. Oregon, Ohio: Life Guardian Foundation., confirm the sad truth that physicians who favor brain death/organ transplantation tend to handle the severely brain-injured patient differently from those who do not.[17] Put bluntly, the difference comes down to whether the severely brain-injured patient (constitutionally healthy prior to the injury) is anticipated as a potential organ donor or whether he or she is viewed as a patient who deserves the maximum therapeutic intervention with a view to full recovery. The resulting consequences are of great import to the patient, however, since it means the difference between life and death, or between full recovery and the severely disabled state of chronic "brain death."

The most important concern in neuro-intensive care is the control of brain edema. To this end, several protocols of intracranial pressure-targeted therapy have been developed.[18] In addition to the specific measures for controlling ICP and maintaining adequate cerebral perfusion, the aim is also to avoid anything that may produce systemic disturbances such as hypotension, hypoxemia, and hyperthermia, all of which may cause further insults to the already injured brain (Smith 2014 Smith, Martin. 2014. Critical care management of severe head injury. *Anaesthesia & Intensive Care Medicine* 15, no. 4: 164–7. doi: 10.1016/j.mpaic.2014.01.016, 164). It is in light of these neuro-intensive care requirements that the clinical tests (especially the apnea test) for "brain death" are evaluated in the next section.

Critical Evaluation of the Clinical Tests for "Brain Death"

The clinical test-criteria for the determination of "brain-death" are well engrained in the "brain-death" literature since they have not changed much between the 1968 Harvard Report and the 2010 American Academy of Neurology Guidelines for Brain Death Determination (Wijdicks et al. 2010 Wijdicks, Eelco F.M., Panayiotis N. Varelas, Gary S. Gronseth, and David M. Greer. 2010. Evidence-based guideline update: Determining brain death in adults: Report of the quality standards subcommittee of the American academy of neurology. *Neurology* 74, no. 23: 1911–18. doi: 10.1212/WNL.0b013e3181e242a8). While the number of possible confirmatory tests, which are ancillary and optional, has increased, the core clinical tests performed at the bedside have remained unchanged. They include: (i) coma, with complete unresponsiveness even to the most painful stimuli; (ii) absence of brain stem reflexes; and (iii) apnea, that is, absence of a breathing drive as tested with a CO_2 challenge. Ancillary tests are of two types, for the purpose of detecting either brain electrical activity or evidence of brain blood flow. The 2010 guidelines specify, however, that "in adults, ancillary tests are not needed for the clinical diagnosis of brain death and cannot replace a neurologic examination" (Wijdicks et al. 2010 Wijdicks, Eelco F.M., Panayiotis N. Varelas, Gary S. Gronseth, and David M. Greer. 2010. Evidence-based guideline update: Determining brain death in adults: Report of the quality standards subcommittee of the American academy of neurology. *Neurology* 74, no. 23: 1911–18. doi: 10.1212/WNL.0b013e3181e242a8, 1916). Without the use of ancillary tests, the determination of the US-based "whole brain death" is identical to that of the UK-based "brainstem death."

Problems with the clinical tests for "brain death" in general

With the worldwide legal adoption of "brain death" as death (undoubtedly driven by the society's need for organs), it appears that the medical community has accepted the clinical test-criteria for "brain death" as if they were scientific "truths," even though they are fraught with known flaws and problems. Only a few of these are mentioned below.

The first fundamental flaw is the lack of scientific data required for the validation of the clinical tests for the determination of "brain death." Medicine is an empirical science; every test procedure and medical product must be validated through various phases of rigorous testing before they can be put to use in clinical practice. Yet, the Harvard report cited no medical studies or any patient data that would validate the clinical tests put forth for establishing "brain death" and equating it with death. The one single prospective study was the 1977 Collaborative Study conducted by the National Institutes of Health evaluating the data collected on 503 "brain-dead" patients (National Institutes of Health 1977 National Institutes of Health. 1977. An appraisal of the criteria of cerebral death: A summary statement. *Journal of the American Medical Association* 237, no. 10: 982–86. doi: 10.1001/jama.1977.03270370054022). It then called for a larger clinical trial, which still remains to be carried out. Without a valid scientific foundation, the whole of the clinical test battery (and thus the "brain-death" standard itself) is merely opinion-based, that is, reflecting the opinion of the thirteen members of the Harvard Committee and their subsequent followers.

The second major problem is the lack of consensus regarding the clinical testing itself.[19] The law leaves it to the individual physician to determine "brain death" according to accepted medical standards, which can be national, regional, or local. Thus, a 2008 survey revealed "wide variability in the practice and determination of brain death among the … top 50 institutions for neurology and neurosurgery" (Greer et al. 2008 Greer, David M., Panayiotis N. Varelas, Shamael Haque, Eelco F.M. Wijdicks, David M. Greer, Panayiotis N. Varelas, Shamael Haque, and Eelco F.M. Wijdicks. 2008. Variability of brain death determination guidelines in leading US neurologic institutions. *Neurology* 70, no. 4: 284–289. doi: 10.1212/01.wnl.0000296278.59487.c2, 287). Variability was found in all areas, including apnea testing, "an area with the greatest possibility for inaccuracies," (Greer et al. 2008 Greer, David M., Panayiotis N. Varelas, Shamael Haque, Eelco F.M. Wijdicks, David M. Greer, Panayiotis N. Varelas, Shamael Haque, and Eelco F.M. Wijdicks. 2008. Variability of brain death determination guidelines in leading US neurologic institutions. *Neurology* 70, no. 4: 284–289. doi: 10.1212/01.wnl.0000296278.59487.c2, 288) in which there were marked variations in technique between institutions. Such variability is indeed worrisome. It calls into question the accuracy of "brain-death" determination across institutions and even among individual physicians within the same institution. This is not a theoretical question, especially since it has been shown that "physicians involved in declaring brain death were unable to correctly identify or apply the whole brain criteria for determination of brain death" (Van Norman 1999 Van Norman, Gail. 1999. A matter of life and death: What every anesthesiologist should know about the medical, legal, and ethical aspects of declaring brain death. *Anesthesiology* 91, no. 1: 275–87. doi: 10.1097/00000542-199907000-00036, 281). The three cases described by Van Norman prove that it is not impossible that a patient may be inappropriately labeled as dead according to the neurological standard.

The third major flaw concerns logical and scientific incoherencies. The line of reasoning used for each of the clinical bedside tests for "brain death," which include noxious stimulus, brainstem reflexes, and apnea testing, is the following: if a response is observed, then the brain is alive; if no response is observed, then the brain is dead (not alive). There are two problems with this line of reasoning, however. First, from the logic standpoint, it has the form of "if P then Q; not P, therefore not Q." This is none other than the *inverse fallacy*, well-known in formal logic, which renders the whole reasoning invalid. Shewmon thus rightly points out, "the medical community has fallen into the logical fallacy of accepting that *absence of evidence* of conscious activity constitutes evidence of *absence*" (emphasis original),[20] when it adopted uncritically the Harvard Committee's "brain-death" standard. Second, from the scientific standpoint, these tests only give us information regarding the motor (efferent) arm of the

neural reflex pathway. There exists no means to obtain direct information on the activities of the sensory (afferent) arm and interneurons. Hence, both logically and scientifically, the bedside clinical test-criteria do not constitute sufficient grounds to conclude with certainty that the brain is dead. Moreover, it is beyond empirical science to determine sensations such as pain, a subjective, first-person, conscious experience. Thus, one cannot assume offhand from the absence of external reactions that the persons labeled brain-dead do not feel pain or that they cannot hear and are not aware of what is being said about them.[21] With regard to pain, it is known that in several countries, anesthesia is administered to brain-dead patients as they are being laparotomized for organ harvesting.

Fourth, a proper understanding of the pathophysiology of brain injury helps to explain why the absence of response to the bedside clinical tests, as well as the lack of detectable electrical activity or cerebral flow, do not necessarily indicate "brain death." Injury to the brain, just like injury to any other organ system, triggers two immediate interrelated phenomena: (i) a shutdown of brain function as a self-protective mechanism to reduce its metabolic requirement, and (ii) a cascade of inflammatory response with the release of numerous immune mediators, which leads to increased ICP and decreased cerebral blood flow (CBF).[22] The net result is a condition known as global ischemic penumbra, as "the blood supply to the brain falls down to levels … between 50 to 80% lower than the normal values… [It can remain] within that range for up to 48 hours" (Coimbra 2009a Coimbra, Cicero Galli. 2009a. The apnea test—a bedside lethal "disaster" to avoid a legal "disaster" in the operating room. In *Finis vitae: "brain death" is not true death*, ed. Roberto De Mattei, and Paul A. Byrne, 129–61. Oregon, Ohio: Life Guardian Foundation., 132). Such a drop in CBF adds to the suppression of neurological functions, but without loss of organic vitality because physiologically, the level of energy required for sustaining the vitality of an organ is much lower than that needed for maintaining its function. Neurological functions, therefore, remain recoverable, provided that CBF is maintained such that "the oxygen extraction fraction continues to be [sufficiently] elevated" (Coimbra 1999 Coimbra, C.G. 1999. Implications of ischemic penumbra for the diagnosis of brain death. *Brazilian Journal of Medical and Biological Research* 32: 1479–87. doi: 10.1590/S0100-879X1999001200005, 1480). This is why the penumbra is such a critical time-window when prompt application of aggressive therapeutic intervention can significantly improve the outcomes of patients with SBI.

The severely depressed brain activity during the penumbra explains why external stimuli (clinical bedside tests) elicit no response and no electrical signals are recorded on the electroencephalogram (EEG). Besides the known limitations of EEG testing, it is also known that "when CBF reaches about 20 ml/100 mg/min, EEG isoelectricity occurs;"[23] that level of CBF is still above the CBF threshold (10 ml/100 mg/min) at which neuronal injury becomes irreversible. In other words, the lack of detectable brainstem reflexes and a flat EEG in an apneic, comatose patient do not necessarily indicate the loss of neuronal vitality or "brain death." Thus, it cannot be simply decreed that the lack of detectable functions is equivalent to the *irreversible* loss of function (which implies the organic death of the organ). In this regard, post-mortem studies showed that in at least 60 percent of cases, the brains of heart-beating donors had no or minimal structural change of the brainstem (Wijdicks and Pfeifer 2008 Wijdicks, Eelco F.M., and Eric A. Pfeifer. 2008. Neuropathology of brain death in the modern transplant era. *Neurology* 70, no. 15: 1234–7. doi: 10.1212/01.wnl.0000289762.50376.b6, 1236); one cannot, therefore, exclude the possibility that brainstem functions could have returned if the patients had not been rushed to organ donation.[24]

Similarly, the reduction in CBF during the penumbra can fall below the detection threshold of radionuclide angiography, while still remaining above the critical level at which infarction of neuronal tissue occurs. Therefore, the absence of intracranial blood vessels on angiographic studies cannot be taken as "indisputable evidence for intracranial circulatory arrest" (Coimbra 1999 Coimbra, C.G. 1999. Implications of ischemic penumbra for the diagnosis of brain death. *Brazilian Journal of Medical and Biological Research* 32: 1479–87. doi: 10.1590/S0100-879X1999001200005, 1485). Recently, Bernat strongly asserted that "the absence of intracranial blood flow proves that the loss of clinical brain functions is total and permanent," and therefore a laboratory test for intracranial blood flow should be

made mandatory (Bernat 2004 Bernat, James L. 2004. On irreversibility as a prerequisite for brain death determination. In *Brain death and disorders of consciousness*, ed. Calixto Machado and D. Alan Shewmon, 161–7. New York: Springer., 164). Bernat recommended various tests, including radionuclide angiography, transcranial Doppler ultrasound, and other newly developed imaging techniques.[25] As pointed out by Shewmon (2012a Shewmon, D. Alan. 2012a. Brain death or brain dying? *Journal of Child Neurology* 27, no. 1: 4–6. doi: 10.1177/0883073811421986, 5), however, "even tests of cerebral blood flow could be misleading, given that none of the standard 'confirmatory' tests for brain death has been validated to possess sufficient sensitivity to reliably distinguish penumbra-level flow from no flow."

In brief, taking into account the knowledge about the penumbra, the test-criteria for "brain death" only demonstrate that neurological activity and blood flow are not detectable at the time of the testing (when the brain is most likely in penumbra). Certainly, the results of the clinical tests fulfill the criteria for "brain death," but they cannot be taken as the *sine qua non* of the irreversible loss of neurological functions, let alone "brain death" or the death of the patient. In fact, what could very well push the injured brain to a state of irreversible damage would be the apnea test itself.

Problems with the apnea test

According to the published guidelines, the apnea test is the cornerstone procedure for declaring a patient brain-dead. This procedure, just like all the other clinical test-criteria put forth by the Harvard Committee, has never been validated. A recent review of current-practice apnea testing reveals that "it still lacks consensus standardization regarding the actual procedure, monitored parameters, and evidence-based safety measures that may be used to prevent complications" (Scott et al. 2013 Scott, J Brady, Michael A Gentile, Stacey N Bennett, MaryAnn Couture, and Neil R MacIntyre. 2013. Apnea testing during brain death assessment: A review of clinical practice and published literature. *Respiratory Care* 58, no. 3: 532–38. doi: 10.4187/respcare.01962, 532). A close look at the apnea test in light of the neuro-intensive care requirements for SBI demonstrates that this procedure does not take into account the pathophysiology of brain injury and coma, namely those factors which can worsen cerebral ischemia, brain edema, and cerebral hypertension (increased ICP).

In SBI, it is known that "the presence of cerebral ischemia is associated with a poor ultimate neurological outcome, that is, dead or vegetative state" (Werner and Engelhard 2007 Werner, C., and K. Engelhard. 2007. Pathophysiology of traumatic brain injury. *British Journal of Anaesthesia* 99, no. 1: 4–9. doi: 10.1093/bja/aem131, 5). For this very reason, one of the aims in neuro-intensive care is to prevent anything that may cause an increase in the partial pressure of arterial carbon dioxide ($PaCO_2$) because hypercarbia leads to cerebrovascular vasodilatation and increased ICP (Mongardon et al. 2011 Mongardon, Nicolas, Florence Dumas, Sylvie Ricome, David Grimaldi, Tarik Hissem, Frédéric Pène, and Alain Cariou. 2011. Postcardiac arrest syndrome: From immediate resuscitation to long-term outcome. *Annals of Intensive Care* 1, no. 1: 1–11. doi: 10.1186/2110-5820-1-45, 6), which, in turn, worsens cerebral ischemia and potentially contributes to brain herniation (Joffe, Anton, and Duff 2010 Joffe, A.R., N.R. Anton, and J.P. Duff. 2010. The apnea test: Rationale, confounders, and criticism. *Journal of Child Neurology* 25, no. 11: 1435–43. doi: 10.1177/0883073810369380, 1437). The injured brain has decreased tolerance to CO_2, such that even a minor increase in $PaCO_2$ can aggravate the existing brain edema. Cerebral ischemia, brain edema, and increased ICP mutually affect one another, resulting in a vicious cycle. Brain edema leads to increased ICP, which in turn causes compression of the cerebral vasculature resulting in further reduction of CBF. With hypoperfusion, there is decreased availability of oxygen to brain tissue and concomitant accumulation of CO_2, resulting in intracellular metabolic stress, increased membrane permeability, and worsening edema.

In the apnea test, the patient is disconnected from the ventilator to let the $PaCO_2$ rise above a certain threshold (e.g., 60 mmHg in the United States, 50 mmHg in the UK) or at least 20 mmHg above the

baseline, while oxygenation is preserved via a catheter down the endotracheal tube delivering 100 percent O_2 (Joffe, Anton, and Duff 2010 Joffe, A.R., N.R. Anton, and J.P. Duff. 2010. The apnea test: Rationale, confounders, and criticism. *Journal of Child Neurology* 25, no. 11: 1435–43. doi: 10.1177/0883073810369380, 1435; Wijdicks et al. 2010 Wijdicks, Eelco F.M., Panayiotis N. Varelas, Gary S. Gronseth, and David M. Greer. 2010. Evidence-based guideline update: Determining brain death in adults: Report of the quality standards subcommittee of the American academy of neurology. *Neurology* 74, no. 23: 1911–18. doi: 10.1212/WNL.0b013e3181e242a8, 1916). Given the above information regarding the effect of CO_2, the harmful effect of the apnea test, which induces hypercarbia in comatose patients with SBI is self-evident. The apnea-induced hypercarbia can easily push CBF to below the critical level of 10–15 ml/100 g/min, below which cellular membrane depolarization and disruption of ionic homeostasis (that is, irreversible damage of brain tissue) occur (Coimbra 1999 Coimbra, C.G. 1999. Implications of ischemic penumbra for the diagnosis of brain death. *Brazilian Journal of Medical and Biological Research* 32: 1479–87. doi: 10.1590/S0100-879X1999001200005, 1850; Werner and Engelhard 2007 Werner, C., and K. Engelhard. 2007. Pathophysiology of traumatic brain injury. *British Journal of Anaesthesia* 99, no. 1: 4–9. doi: 10.1093/bja/aem131, 5).

The vasodilatation effect of increased $PaCO_2$ also affects systemic vasculature, thus causing hypotension. In the neuro-intensive care literature, hypotension is a significant predictor of death and is associated with poor neurological outcome among survivors (Trzeciak et al. 2009 Trzeciak, Stephen, Alan E. Jones, J. Hope Kilgannon, Barry Milcarek, Krystal Hunter, Nathan I. Shapiro, Steven M. Hollenberg, Phillip Dellinger, and Joseph E. Parrillo. 2009. Significance of arterial hypotension after Resuscitation from cardiac arrest. *Critical Care Medicine* 37, no. 11: 2895–903. doi: 10.1097/CCM.0b013e3181b01d8c). Similarly, in the "brain-death" literature, several studies have shown that the most frequent complication of apnea testing, as high as 39 percent, is hypotension (Goudreau et al. 2000 Goudreau, John L., Eelco F.M. Wijdicks, and Steve F. Emery. 2000. Complications during apnea testing in the determination of brain death: Predisposing factors. *Neurology* 55, no. 7: 1045–8. doi: 10.1212/WNL.55.7.1045; Jeret and Benjamin 1994 Jeret, J.S., and J.L. Benjamin. 1994. Risk of hypotension during apnea testing. *Archives of Neurology* 51, no. 6: 595–9. doi: 10.1001/archneur.1994.00540180073016; Saposnik et al. 2004 Saposnik, Gustavo, Graciela Rizzo, Adriana Vega, Roberto Sabbatiello, and Jorge L. Deluca. 2004. Problems associated with the apnea test in the diagnosis of brain death. *Neurology India* 52, no. 3: 342–5.), as a result of peripheral vasodilatation, acidosis, and the cardio-depressant effects of CO_2. However brief the episode of hypotension may be, it can cause a sharp drop in cerebral perfusion pressure (CPP). Thus, the combined effect of increased ICP and hypotension caused by the apnea test-induced hypercarbia is "collapse of the cerebral vasculature" which cannot even be reversed by measures to lower ICP (Joffe, Anton, and Duff 2010 Joffe, A.R., N.R. Anton, and J.P. Duff. 2010. The apnea test: Rationale, confounders, and criticism. *Journal of Child Neurology* 25, no. 11: 1435–43. doi: 10.1177/0883073810369380, 1437).

The net result is that the apnea test adds further insults to an already injured brain, for it is not unlikely that "the apnea test can convert functioning brain to non-functioning penumbral brain … and can convert penumbral brain to irreversibly non-recoverable brain" (Joffe, Anton, and Duff 2010 Joffe, A.R., N.R. Anton, and J.P. Duff. 2010. The apnea test: Rationale, confounders, and criticism. *Journal of Child Neurology* 25, no. 11: 1435–43. doi: 10.1177/0883073810369380, 1437). In other words, "the apnea test itself can result in failing the apnea test, creating a self-fulfilling prophecy" (Joffe, Anton, and Duff 2010 Joffe, A.R., N.R. Anton, and J.P. Duff. 2010. The apnea test: Rationale, confounders, and criticism. *Journal of Child Neurology* 25, no. 11: 1435–43. doi: 10.1177/0883073810369380, 1437).

These harmful side-effects of the apnea test are known in the medical community, even by scholars who support "brain death." From the perspective of medical ethics, it is most disturbing that the apnea test directly violates the fundamental principles for the management of SBI, in which the prevention of

hypercarbia and hypotension is one of the most important measures. As pointed out by Mongardon, in reference to the prevention of any further brain damage as part of the management of post-cardiac syndrome, not only should ventilator support aim to maintain $PaCO_2$ levels within normal limits, but "hypercapnia [hypercarbia], leading to cerebrovascular vasodilatation and increased intracranial pressure, should be banned" (Mongardon et al. 2011 Mongardon, Nicolas, Florence Dumas, Sylvie Ricome, David Grimaldi, Tarik Hissem, Frédéric Pène, and Alain Cariou. 2011. Postcardiac arrest syndrome: From immediate resuscitation to long-term outcome. *Annals of Intensive Care* 1, no. 1: 1–11. doi: 10.1186/2110-5820-1-45, 6). Equally disturbing are reports of patients who, after having failed the apnea test, had a return of their respiratory capacity (Joffe et al. 2009 Joffe, Ari R., Hanna Kolski, Jonathan Duff, and Allan R. deCaen. 2009. A 10-month-old infant with reversible findings of brain death. *Pediatric Neurology* 41, no. 5: 378–82. doi: 10.1016/j.pediatrneurol.2009.05.007; Joffe, Anton, and Duff 2010 Joffe, A.R., N.R. Anton, and J.P. Duff. 2010. The apnea test: Rationale, confounders, and criticism. *Journal of Child Neurology* 25, no. 11: 1435–43. doi: 10.1177/0883073810369380, 1437).[26] Taking all these factors into consideration, it is rather evident that any hypercarbia-producing test can be of no therapeutic benefit to a ventilator-dependent and brain-injured individual; it may even cause him or her great harm (however slight the risk might be), which is contrary to the *telos* of medicine. It is in view of this *telos* that the role of the physician toward his or her severely brain-injured patient is examined next.

Ethical Foundations of Patient Care—Primum Non Nocere

In recent decades, scientific progress, sociocultural changes, and moral pluralism have altered the practice of the medical profession. Besides a weakening of the moral fabric in the medical community,[27] there has been also a "transformation of the physician into a variety of roles" (Pellegrino and Thomasma 1993 Pellegrino, Edmund D., and David C. Thomasma. 1993. *The virtues in medical practice*. New York: Oxford University Press., 35),[28] much to the detriment of the doctor–patient relationship. Yet, it is this very relationship that constitutes the essence of the medical profession. It is a relationship in which the very sick person finds him- or herself in a most vulnerable and exploitable condition; his or her welfare thus depends not just on the knowledge and skills of his or her doctor, but also on the latter's ethical outlook. This is why medicine is "an inherently moral practice" (Sulmasy 2014 Sulmasy, Daniel P. 2014. Edmund Pellegrino's philosophy and ethics of medicine: An overview. *Kennedy Institute of Ethics Journal* 24, no. 2: 105–12. doi: 10.1353/ken.2014.0015, 107),[29] in which the role of the physician as a moral agent is of central importance. Thus, despite the dominance of principle-based ethics, introduced by Beauchamp and Childress (1979) Beauchamp, Tom L., and James F. Childress. 1979. *Principles of biomedical ethics*. New York: Oxford University Press., the truth remains that in medicine, we are dealing with a sick human being, rather than with abstract principles. The *telos* of the medical profession thus rests on the *telos* of the doctor–patient relationship. This, in turn, means that the physician's moral attitude, and consequently, his or her clinical acts "must be directed to what is necessary to heal and to help *this* patient" (emphasis original) (Pellegrino 1995 Pellegrino, Edmund D. 1995. Toward a virtue-based normative ethics for the health professions. *Kennedy Institute of Ethics Journal* 5, no. 3: 253–77. doi: 10.1353/ken.0.0044, 267).

The above notion coheres with the Hippocratic Oath, which contains the following important clause, "I will use treatment to help the sick according to my ability and judgment but never with a view to injury and wrong doing."[30] Thus, the most fundamental obligation of the physician is to do no harm, and any medical decision regarding the health or the life and death of a patient must be for his or her own good. Medically speaking, this good requires that he or she is "not only to be protected from harm, but also … to have health restored … or … his [or her] disability lessened" (Pellegrino and Thomasma 1988 Pellegrino, Edmund D., and David C. Thomasma. 1988. *For the patient's good: The restoration of beneficence in health care*. New York: Oxford University Press., vii–viii). This twofold notion of beneficence and non-maleficence has been, since time immemorial, "the central moral principle in the

ethics of medicine" (Pellegrino and Thomasma 1988 Pellegrino, Edmund D., and David C. Thomasma. 1988. *For the patient's good: The restoration of beneficence in health care*. New York: Oxford University Press., vii). At the same time, however, the Hippocratic ethos also requires that physicians recognize when treatment is no longer effective.[31] Such an ethos coheres with the teaching of the *Catechism of the Catholic Church*, in which it is written: "Discontinuing medical procedures that are burdensome, dangerous, extraordinary, or disproportionate to the expected outcome can be legitimate" (Catechism 2003 Catechism of the Catholic Church. 2003. Vatican City: Libreria Editrice Vaticana., no. 2278).

The end of medical care is thus to relieve pain or suffering and restore health, and as such, preserve life. It is not to sustain life by futile and disproportionate means when death is imminent, however. The common scenario that fits this description is a patient with multi-organ failure as the result of a steadily downhill course caused by a pre-existing illness (e.g., cancer). It is legitimate, therefore, in such cases, to withdraw life support to allow the patient to progress to a natural death.

There is a distinction between allowing the patient to die and killing him or her, however. As stated in the *Catechism*, "whatever its motives and means, direct euthanasia consists in putting an end to the lives of handicapped, sick, or dying persons. It is morally unacceptable" (Catechism 2003 Catechism of the Catholic Church. 2003. Vatican City: Libreria Editrice Vaticana., no. 2277). The *Catechism*, while praising organ donation as a noble act of charity, emphasizes that it must cohere with the moral law, precisely because "it is *not morally admissible to bring about the* disabling mutilation or *death of a human being, even in order to delay the death of other persons*" (emphasis added) (Catechism 2003 Catechism of the Catholic Church. 2003. Vatican City: Libreria Editrice Vaticana., no. 2296).

As demonstrated in the previous sections, brain death cannot be equated with the biological death of the human person. This, in turn, implies that harvesting vital organs from brain-dead donors is the event that brings about their true and premature death. In other words,

heart-beating organ procurement from patients with impaired consciousness is de facto a concealed practice of active euthanasia and physician-assisted death, both of which, either concealed or overt, the Catholic Church opposes. (Verheijde and Potts 2010 Verheijde, J.L., and M. Potts. 2010. Commentary on the concept of brain death within the Catholic bioethical framework. *Christian Bioethics* 16, no. 3: 246–56. doi: 10.1093/cb/cbq019, 246)

The objective medical evidence regarding "brain death" has been such that even some scholar-advocates of organ donation (namely, Truog) have openly admitted that, "beginning with the development of the concept of brain death[,] … both the United States and Europe [have been] experimenting with active hastening of the dying process through euthanasia and physician assisted suicide" (Truog 2008 Truog, R.D. 2008. End-of-life decision-making in the United States. *European Journal of Anaesthesiology* 25, Suppl 42: 43–50. doi: 10.1017/S0265021507003419, 43). In a way, these words echo the prophetic warning of St. John Paul II regarding furtive euthanasia for the interests of organ transplantation (John Paul II 1995 John Paul II, Pope. 1995. Evangelium vitae. http://www.vatican.va/holy_father/john_paul_ii/encyclicals/documents/hf_jp-ii_enc_25031995_evangelium-vitae_en.html., no. 15). On the other hand, his Address to the Transplantation Society (John Paul II 2000 John Paul II, Pope. 2000. Address to the 18th International congress of the transplantation society. August 29. http://www.vatican.va/holy_father/john_paul_ii/speeches/2000/jul-sep/documents/hf_jp-ii_spe_20000829_transplants_en.html.) has been often cited by pro-"brain death" Catholic scholars as a strong evidence that the Church has accepted the "brain death" standard (see Furton 2002 Furton, Edward J. 2002. Brain death, the soul, and organic life. *National Catholic Bioethics Quarterly* 2, no. 3: 455–70. doi: 10.5840/ncbq20022332; Haas 2011 Haas, John M. 2011. Catholic teaching regarding the legitimacy of neurological criteria for the determination of death. *National Catholic Bioethics Quarterly* 11, no. 2: 279–99. doi: 10.5840/ncbq201111254), even though the pope in fact formulated his cautious acceptance in explicit conditional terms. According to the pope's words, for the neurological standard to be truly accepted, it is required not only that it be "rigorously applied," but

also (i) that it has been established by "clearly determined parameters *commonly held* by the international scientific community" (emphasis added) (that is, a medical consensus, which the pope presupposed), and (ii) that it coheres with sound Christian anthropology.[32] Space limitation does not permit a detailed analysis of the pope's address in this paper.[33] Suffice it to note, however, that the pope's requirement of a medical consensus is yet to be fulfilled.

It is also worthwhile to note that Pope Benedict XVI, who in his discourses often made reference to his predecessors (in particular John Paul II), did not make any reference to the 2000 address during his address to the participants of the 2008 international congress organized by the Pontifical Academy for Life. Instead, Benedict XVI gave the following short and clear reminder:

Individual organs cannot be extracted except *ex cadevere*… In an area such as this [that is, in the area of the determination of death], in fact, there cannot be the slightest suspicion of arbitration [arbitrariness] and where certainty has not been attained the principle of precaution must prevail… The principal criteria of respect for the life of the donator [donor] must always prevail so that the extraction of organs be performed only in the case of his/her true death.[34] (Benedict XVI 2008 Benedict XVI, Pope. 2008. Address to participants at an International Congress organized by the Pontifical academy for life. November 7. http://www.vatican.va/holy_father/benedict_xvi/speeches/2008/november/documents/hf_ben-xvi_spe_20081107_acdlife_en.html.)

In a way, the cautious admonition of Benedict XVI is a reminder of the fundamental principle in medical care, *primum non nocere*. The responsibility of the physician is therefore a grave one, when by virtue of his or her medical subspecialty, he or she is involved in the care of a patient suffering so-called irreversible coma, who up until then has been constitutionally healthy, with no prior comorbidity such as heart disease or cancer, and no significant associated systemic injury which could affect the quality of the organs. Because such a patient is comatose, the decision making concerning his or her medical condition falls upon the family members. The family, being in a state of great distress, cannot adequately partake in the process, not only because of their limited medical knowledge, but above all, because the full truth about "brain death" has never been officially made known to the public. The content of the available information is mostly promotional, to promote organ donation under the putative good reasons of noble charity or "finding meaning in death"; critical information regarding the means and procedures used in organ harvesting is noticeably absent, however.[35] In the same vein, organ-procurement organizations have carefully kept the public uninformed with regard to the many controversies surrounding "brain death" (Nair-Collins 2010 Nair-Collins, Mike. 2010. Death, brain death, and the limits of science: Why the whole-brain concept of death is a flawed public policy. *Journal of Law, Medicine & Ethics* 38, no. 3: 667–83. doi: 10.1111/j.1748-720X.2010.00520.x, 678).

It is at the moment of great distress and vulnerability that the request of organ donation is posed to the family. It is not infrequent, among those who work with the families of "brain-dead" donors, to encounter families who subsequently express negative feelings about their experience of donation, and who "doubt whether their [loved one] was in fact dead at the time [of the declaration of death]" (Tonti-Filippini 1998 Tonti-Filippini, Nicholas. 1998. Revising brain death: Cultural imperialism? *Linacre Quarterly* 65, no. 2: 51–72., 57). Such doubts only surface after the relatives have had enough time to reflect (and investigate), and in the process, to question the information which health professionals have given them concerning the death of their loved one. They feel assaulted and exploited when they find out, subsequently, that "organs are taken while the heart still beats" and that the harvesting procedure necessitates the use of general anesthesia "to suppress the capacity to feel pain" (Nair-Collins 2010 Nair-Collins, Mike. 2010. Death, brain death, and the limits of science: Why the whole-brain concept of death is a flawed public policy. *Journal of Law, Medicine & Ethics* 38, no. 3: 667–83. doi: 10.1111/j.1748-720X.2010.00520.x, 677–8).[36]

This is why the physician, by virtue of his or her expertise, carries the greater share of the moral responsibility toward the severely brain-injured patient. The moral decision is then between (a)

declaring the patient brain-dead and sending him or her for organ harvesting, or (b) administering state-of-the-art modalities for the acute management of SBI, thereby offering the patient a chance to recover. In the field of neuro-intensive care, it is known that it is not possible to predict the patient's outcome in the immediate acute phase of SBI. To determine the patient's prognosis requires not only several sequential evaluations, but also that some period of time (in terms of days and weeks) has elapsed to give a more complete picture of the patient's clinical course.[37] What the patient needs is prompt neuro-intensive care during the therapeutic window of ischemic penumbra, and a generous amount of time to permit brain function to recover, even if just partially.[38] Instead, too often, he or she is given just "supportive measures to maintain vital signs, [which] consume the critical time window" (Coimbra 2009b Coimbra, Cicero Galli. 2009b. Are "brain dead" (or "brain-stem dead'') patients neurologically recoverable? In *Finis vitae: "brain death" is not true death*, ed. Roberto De Mattei, and Paul A. Byrne, 313–78. Oregon, Ohio: Life Guardian Foundation., 332), followed by a declaration of "brain death" and a request for organ donation) within 24 to 36 hours of admission. In light of the Hippocratic ethos (*primum non nocere*) and the teaching of the Catholic Church, to which the principle *in dubio, pro vita* should be added, I, as a Catholic physician and moral theologian, would follow the second moral option, even if this seems counter-cultural. A brief overview of neuro-intensive care is thus presented as the conclusion of this paper.

Conclusion—Alternative to Brain Death: Neuro-Intensive Care of Severe Brain Injury

The state of the art of neuro-intensive care for severely brain-injured patients is based on our current understanding of the pathophysiology of SBI. The primary brain damage caused by the initial insult triggers a cascade of pathophysiological processes, including "neurotransmitter release, free-radical generation, calcium-mediated damage, gene activation, mitochondrial dysfunction, and inflammatory responses" (Maas, Stocchetti, and Bullock 2008 Maas, Andrew I.R., Nino Stocchetti, and Ross Bullock. 2008. Moderate and severe traumatic brain injury in adults. *Lancet Neurology* 7, no. 8: 728–41. doi: 10.1016/S1474-4422(08)70164-9, 730; Mongardon et al. 2011 Maas, Andrew I.R., Nino Stocchetti, and Ross Bullock. 2008. Moderate and severe traumatic brain injury in adults. *Lancet Neurology* 7, no. 8: 728–41. doi: 10.1016/S1474-4422(08)70164-9, 2–3). The synergistic interplay of these processes produces secondary brain injury, leading to the vicious cycle of brain edema, raised ICP, decreased CPP, and CBF.[39] Another factor that has adverse effects on CBF is $PaCO_2$. A rise in $PaCO_2$ produces vasodilatation leading to vasogenic edema, increased ICP (Baxter and Wilson 2012 Baxter, David, and Mark Wilson. 2012. The fundamentals of head injury. *Surgery* (Oxford) 30, no. 3: 116–21. doi: 10.1016/j.mpsur.2011.12.002, 118), and hypotension, a known "independent predictor of poor outcome" (Marik et al. 1999 Marik, Paul, Karen Chen, Joseph Varon, Robert FrommJr, and George L. Sternbach. 1999. Management of increased intracranial pressure: A review for clinicians. *Journal of Emergency Medicine* 17, no. 4: 711–19. doi: 10.1016/S0736-4679(99)00055-4, 712; Trzeciak et al. 2009 Trzeciak, Stephen, Alan E. Jones, J. Hope Kilgannon, Barry Milcarek, Krystal Hunter, Nathan I. Shapiro, Steven M. Hollenberg, Phillip Dellinger, and Joseph E. Parrillo. 2009. Significance of arterial hypotension after Resuscitation from cardiac arrest. *Critical Care Medicine* 37, no. 11: 2895–903. doi: 10.1097/CCM.0b013e3181b01d8c). The concomitant hypotension and high ICP is thus a fatal combination to the already injured brain. In addition, patients with SBI often have disturbances of the hypothalamus-pituitary axis, resulting in diabetes insipidus, acute hypothyroidism, and acute adrenal failure (Coimbra 2009b Coimbra, Cicero Galli. 2009b. Are "brain dead" (or "brain-stem dead'') patients neurologically recoverable? In *Finis vitae: "brain death" is not true death*, ed. Roberto De Mattei, and Paul A. Byrne, 313–78. Oregon, Ohio: Life Guardian Foundation., 320–2; Powner et al. 2006 Powner, David J., Cristina Boccalandro, M. Serdar Alp, Dennis G. Vollmer, David J. Powner, Cristina Boccalandro, M. Serdar Alp, and Dennis G. Vollmer. 2006. Endocrine failure after traumatic brain injury in adults. *Neurocritical Care* 4, no. 1: 61–70. doi: 10.1385/NCC:5:1:61;

Tsagarakis, Tzanela, and Dimopoulou 2005 Tsagarakis, S., M. Tzanela, and I. Dimopoulou. 2005. Diabetes insipidus, secondary hypoadrenalism and hypothyroidism after traumatic brain injury: Clinical implications. *Pituitary* 8, no. 3–4: 251–54. doi: 10.1007/s11102-006-6049-x), all of which contribute to the worsening of the patient's condition.

The aim of neuro-intensive care in SBI is therefore to minimize secondary brain insults, thereby preventing the vicious cycle and promoting neurologic recovery. This means, first and foremost, the prevention of cerebral ischemia, which, in turn, requires hemodynamic stability, adequate CPP, control of ICP, and the use of neuroprotective agents (Marik et al. 1999 Marik, Paul, Karen Chen, Joseph Varon, Robert FrommJr, and George L. Sternbach. 1999. Management of increased intracranial pressure: A review for clinicians. *Journal of Emergency Medicine* 17, no. 4: 711–19. doi: 10.1016/S0736-4679(99)00055-4, 711; Smith 2014 Smith, Martin. 2014. Critical care management of severe head injury. *Anaesthesia & Intensive Care Medicine* 15, no. 4: 164–7. doi: 10.1016/j.mpaic.2014.01.016, 165). For this reason, it has been recommended that patients with severe TBI be treated in neurosurgical units where intensive protocol-driven therapies are available. Studies have shown that "specialist neurocritical care with protocol-driven therapy [that is, ICP/CPP-directed] is associated with a significant improvement in outcome for all patients with severe head injury" (Patel et al. 2002 Patel, Hiren C., David K. Menon, Susan Tebbs, Rebecca Hawker, Peter J. Hutchinson, and Peter J. Kirkpatrick. 2002. Specialist neurocritical care and outcome from head injury. *Intensive Care Medicine* 28, no. 5: 547–53. doi: 10.1007/s00134-002-1235-4, 547), whereas "care in a non-neurosurgical center is associated with a twofold increase in the odds of death" (Patel et al. 2005 Patel, H.C., O. Bouamra, M. Woodford, A.T. King, D.W. Yates, and F.E. Lecky. 2005. Trends in head injury outcome from 1989 to 2003 and the effect of neurosurgical care: An observational study. *Lancet* 366, no. 9496: 1538–44. doi: 10.1016/S0140-6736(05)67626-X, 1542).

One of the new modalities used in ICP/CPP-targeted therapy is hypothermia. There is overwhelming evidence in the medical literature that properly administered mild to moderate hypothermia (32–35°C) in the early phase of SBI significantly lowered ICP with a resulting decreased mortality and morbidity.[40] Hypothermia produces multiple protective effects on the injured brain, by inhibiting the deleterious pathophysiological cascade in injured brain cells, reducing cerebral metabolic rate for oxygen (Metz et al. 1996 Metz, Christoph, Matthias Holzschuh, Thomas Bein, Christian Woertgen, Anton Frey, Irmgard Frey, Kai Taeger, and Alexander Brawanski. 1996. Moderate hypothermia in patients with severe head injury: Cerebral and extracerebral effects. *Journal of Neurosurgery* 85, no. 4: 533–41. doi: 10.3171/jns.1996.85.4.0533, 535), and decreasing vasogenic edema, among others (Azzopardi and Edwards 2007 Azzopardi, D., and A.D. Edwards. 2007. Hypothermia. *Seminars in Fetal and Neonatal Medicine* 12, no. 4: 303–10. doi: 10.1016/j.siny.2007.01.022, 304; Polderman et al. 2002 Polderman, Kees H., Rudi Tjong Tjin Joe, Saskia M. Peerdeman, William P. Vandertop, and Armand R.J. Girbes. 2002. Effects of therapeutic hypothermia on intracranial pressure and outcome in patients with severe head injury. *Intensive Care Medicine* 28, no. 11: 1563–73. doi: 10.1007/s00134-002-1511-3, 1571; Polderman et al. 2004 Polderman, Kees H., Arthur R.H. Van Zanten, Martine D. Nipshagen, and Armand R.J. Girbes. 2004. Induced hypothermia in traumatic brain injury: Effective if properly employed. *Critical Care Medicine* 32, no. 1: 313–4. doi: 10.1097/01.CCM.0000104936.28288.7A). Since the cascade of secondary ischemic brain injury begins soon after the primary injury, "patients need to be cooled soon enough, cold enough, and long enough to minimize hypoxic-ischemic damage" (Shann 2003 Shann, Frank. 2003. Hypothermia for traumatic brain injury: How soon, how cold, and how long? *Lancet* 362, no. 9400: 1950–1. doi: 10.1016/S0140-6736(03)15083-0, 1950). Not infrequently, however, victims of severe TBI did not receive ICP/CPP-directed therapy, despite the fact that ICP monitoring is required by guidelines for the care of severe TBI (Coimbra 2009b Coimbra, Cicero Galli. 2009b. Are "brain dead" (or "brain-stem dead'') patients neurologically recoverable? In *Finis vitae: "brain death" is not true death*, ed. Roberto De Mattei, and Paul A. Byrne, 313–78. Oregon, Ohio: Life Guardian Foundation., 313–4; Watanabe 2009 Watanabe, Yoshio. 2009. Controversies on brain death in Japan and our seven-year experience after the enforcement of the organ transplantation law. In *Finis vitae: "brain death" is not true death*, ed.

Roberto De Mattei, and Paul A. Byrne, 283–92. Oregon, OH: Life Guardian Foundation., 288).[41]

In addition, the management of SBI also includes other measures of general intensive care, with particular attention to the correction of hemodynamic instability, as well as electrolyte disturbances and hormonal deficiencies caused by hypothalamic-pituitary dysfunction. In this regard, it is ironic that the guidelines for the care of the organs of brain-dead donors are closely similar to those for patients with SBI (albeit, minus the brain-targeted therapy). In the care of organs, the goal is "to maintain body temperature, ensure adequate oxygenation, circulating blood volume, cardiovascular stability, and adequate urine output … [in order to avoid] the most common derangements, [namely] hypothermia, hypotension, and diabetes insipidus" (McKeown, Bonser, and Kellum 2012 McKeown, D.W., R.S. Bonser, and J.A. Kellum. 2012. Management of the heartbeating brain-dead organ donor. *British Journal of Anaesthesiology* 108 Suppl 1: i96–107. doi: 10.1093/bja/aer351, i98–9). Some guidelines even include thyroid hormones to maximize the number of organs to be harvested (Salim et al. 2007 Salim, Ali, Matthew Martin, Carlos Brown, Kenji Inaba, Bradley Roth, Pantelis Hadjizacharia, Angela Mascarenhas, Peter Rhee, and Demetrios Demetriades. 2007. Using thyroid hormone in brain-dead donors to maximize the number of organs available for transplantation. *Clinical Transplantation* 21, no. 3: 405–9. doi: 10.1111/j.1399-0012.2007.00659.x). Thus, it is not a far-fetched exaggeration to say that "once the patient goes brain dead and his relatives sign his organ donation consent form, he will get the best medical care of his life" (Teresi 2012 Teresi, Dick. 2012. *The undead: Organ harvesting, the ice-water test, beating-heart cadavers—How medicine is blurring the line between life and death*. New York: Vintage Books., 146–7).

In conclusion, medicine considered as a whole, has for its lofty *raison d'être* healing and saving or preserving life; it can therefore be presumed that transplantation medicine *per se* is interested in saving the lives of human beings. Nevertheless, as Spaemann pointed out, "*it has to be ensured, however, that saving lives does not happen at the expense of the lives of other people*" (emphasis added) (Spaemann 2007 Spaemann, Robert. 2007. Is brain death the death of the human being? On the current state of the debate. In *The signs of death: The proceedings of the working group 11–12 September 2006*, edited by Marcelo Sánchez Sorondo, 130–41. Vatican City: Pontifical Academy of Sciences., 133). To do otherwise is both a gross abuse and misunderstanding of the notion of organ donation as a noble act of solidarity with the neighbor, and a violation of the most basic human right of the donor, the right to life. This is what has taken place since the introduction of the Harvard criteria. Spaemann thus wrote:

The new definition of death as "brain death" makes it possible to declare people dead while they are still breathing and to bypass the dying process in order to quarry spare parts for the living from the dying. Death no longer comes at the end of the dying process, but—by the *fiat* of a Harvard commission—at its beginning. (Spaemann 2006 Spaemann, Robert. 2006. When death becomes inhuman. *Communio* 33, no. 2: 298–300., 299)

In other words, even a noble act such as organ donation can be manipulated to serve utilitarian, materialistic, and dehumanizing motives, whereby the most vulnerable members of society are conveniently excluded from the human moral community (Veatch 2004 Veatch, Robert M. 2004. Abandon the dead donor rule or change the definition of death? *Kennedy Institute of Ethics Journal* 14, no. 3: 261–76. doi: 10.1353/ken.2004.0035, 267–8).[42] As mentioned in the introduction, pragmatic-utilitarian motives were evident in the workings of the Harvard Committee to bring about the birth of "brain death." Such motives have remained well hidden behind both the veil "of the powerful metaphor of the 'gift of life' associated with the transplant world" (Lock 2002 Lock, Margaret. 2002. Inventing a new death and making it believable. *Anthropology & Medicine* 9, no. 2: 97–115. doi: 10.1080/1364847022000029705, 114), and the loud appeal to altruism and noble charity. With respect to brain-dead donors, however, we must ask ourselves, "Is such a vigorous appeal to altruism and charity grounded in truth?"[43]

Notes

Notes

1 The term “brain death” in this paper refers to the notion of “whole brain death.” The term “brain death” is also put in quotation marks because of its semantic ambiguity; see Shewmon (1989) Shewmon, D. Alan. 1989. “Brain death”: A valid theme with invalid variations, blurred by semantic ambiguity. In *The determination of brain death and its relationship to human death*, ed. RJ White, H Angstwurm and Carrasco de Paula, 23–51. Vatican City: Pontifical Academy of Sciences.. It is so ingrained in the literature, however, that it is used in this paper as a stand-in for the longer, but more precise phrase “brain-based criteria for the determination of death.”

2 As pointed out by Kerridge et al. Kerridge, I.H., P. Saul, M. Lowe, J. McPhee, and D. Williams. 2003. Commentary on: A definition of human death should not be related to organ transplants. *Journal of Medical Ethics* 29, no. 3: 202. (2003 Kerridge, I.H., P. Saul, M. Lowe, J. McPhee, and D. Williams. 2003. Commentary on: A definition of human death should not be related to organ transplants. *Journal of Medical Ethics* 29, no. 3: 202., 202), “it is only where vital organs are sought that a diagnosis of brain death is required.” Life support can be withdrawn from an imminently dying elderly patient or a terminally ill patient with multisystem failure, comatose or not, without a declaration of “brain death.” The problem of futility of care thus does not require a medical redefinition of death since the teaching of the Church, namely that of Pope Pius XII (Pius XII 1957 Pius XII, Pope. 1957. Address to an International Congress of Anesthesiologists. November 24. http://www.lifeissues.net/writers/doc/doc_31resuscitation.html.) is that there is “no absolute [moral] obligation to prolong the life of a gravely suffering or irreversibly unconscious patient by extraordinary means” (Seifert 2000 Seifert, Josef. 2000. Brain death and Euthansia. In *Beyond brain death: The case against brain based criteria for human death*, edited by Michael Potts, Paul A. Byrne and Richard G. Nilges, 201–27. Dordrecht, Netherlands: Kluwer Academic Publishers., 206). Even Wijdicks (a leading “brain-death” proponent) admitted at the 2006 “The Signs of Death” conference that “the diagnosis of brain death is driven by … [a] transplantation programme” (Sorondo 2007 Sorondo, Marcelo Sánchez, ed. 2007. *The signs of death: The proceedings of the working group 11–12 September 2006*. Vatican City: Pontifical Academy of Sciences., 50).

3 Mollaret and Goulon did not equate *le coma dépassé* with death. The same syndrome was later called irreversibe coma. At the Ciba symposium, the terminology “brain-dead donor” was not yet invented; labels such as “heart-lung preparation” or the oxymoron term “living cadavers” were used instead to refer to patients with *coma dépassé*. Among the strong supporters for adopting a new criterion for death was Joseph E. Murray, a transplant surgeon and future member of the Harvard Committee. See Murray (1966 Murray, Joseph E. 1966. Organ transplantation: The practical possibilities. In *Ethics in medical progress: With special reference to transplantation*, edited by G.E.W. Wolstenholme and M. O'Connor, 54–77. Boston: Little Brown and Company., 69). It is also very telling that some physicians, although supporting the redefinition of death, specifically stated that they would not permit this to be done to their loved ones (Platt 1966 Platt, Robert. 1966. “Ethical problems in medical procedures.” In *Ethics in medical progress: With special reference to transplantation*, edited by G.E.W. Wolstenholme and M. O'Connor, 149–70. Boston: Little Brown and Company., 163).

4 For passages quoted from the committee's drafts and memos, see Giacomini (1997) Giacomini, Mita. 1997. A change of heart and a change of mind? Technology and the redefinition of death in 1968. *Social Science & Medicine* 44, no. 10: 1465–82. doi: 10.1016/S0277-9536(96)00266-3. All documents related to the committee's work in progress are part of the Beecher manuscripts, preserved at the Francis Countway Library of Medicine at Harvard University. The records are closed to the public, however, except to some specific persons.

5 After the publication of the Harvard report, Beecher subsequently reiterated his views, openly stating that, “*at whatever level we choose to call death, it is an arbitrary decision*… It *is* best to choose a level where, although the brain is dead, *usefulness of other organs is still present*” (emphasis added). In other words, the new definition of death “will lead to greater availability than formerly of essential organs in

viable condition, for transplantation, and thus countless lives now inevitably lost will be saved" (Beecher and Dorr 1971 Beecher, H.K., and H.I. Dorr. 1971. The new definition of death. Some opposing views. *Internationale Zeitschrift für klinische Pharmakologie, Therapie, und Toxikologie* 5, no. 2: 120–4., 120). With regard to his own ethical views, Beecher endorsed the situation-ethics approach of his friend, the Protestant theologian John Fletcher who, in Beecher's own words, "has thoroughly convinced [him] … that *only* the end justifies the means" (emphasis original) (Beecher 1970 Beecher, Henry Knowles. 1970. *Research and the individual: Human studies*. Boston: Little Brown and Company., 211; Belkin 2014 Belkin, Gary S. 2014. *Death before dying*. Oxford: Oxford University Press., 83–4).

6 See, for instance, Wijdicks (2001) Wijdicks, Eelco F.M. 2001. The diagnosis of brain death. *New England Journal of Medicine* 344, no. 16: 1215–21. doi: 10.1056/NEJM200104193441606; Wijdicks et al. (2010) Wijdicks, Eelco F.M., Panayiotis N. Varelas, Gary S. Gronseth, and David M. Greer. 2010. Evidence-based guideline update: Determining brain death in adults: Report of the quality standards subcommittee of the American academy of neurology. *Neurology* 74, no. 23: 1911–18. doi: 10.1212/WNL.0b013e3181e242a8.

7 See, for example, Soifer and Gelb (1989) Soifer, Betsy E., and Adrian W. Gelb. 1989. The multiple organ donor: Identification and management. *Annals of Internal Medicine* 110, no. 10: 814–23. doi: 10.7326/0003-4819-110-10-814; Linos et al. (2007) Linos, Konstantinos, John Fraser, William D. Freeman, and Carole Foot. 2007. Care of the brain-dead organ donor. *Current Anaesthesia & Critical Care* 18, no. 5–6: 284–94. doi: 10.1016/j.cacc.2007.09.003.

8 For self-evident reasons related to the health of the organs, 85% of all donors fall within the age group from the second to the fifth decade of life (data retrieved from the Organ Procurement and Transplantation Network [2015] Organ Procurement and Transplantation Network. 2015. http://optn.transplant.hrsa.gov.).

9 Soifer and Gelb (1989 Soifer, Betsy E., and Adrian W. Gelb. 1989. The multiple organ donor: Identification and management. *Annals of Internal Medicine* 110, no. 10: 814–23. doi: 10.7326/0003-4819-110-10-814, 815–6). The authors wrote: "Somatic death closely follows the declaration of brain death. Despite all efforts to maintain the donor's circulation, irreversible cardiac arrest usually occurs within 48 to 72 hours of brain death in adults, although it may take as long as 10 days in children. Indeed, general acceptance of the concept of brain death depended on this close temporal association between brain death and cardiac arrest."

10 Jahi McMath was pronounced "brain-dead" (with absence of brain electrical activity and of blood flow to the brain) in December 2013 by three different neurologists, including Dr. Paul Fisher from Stanford University. By October 2014, however, it was shown that she could move her hands and feet, in response to her mother's verbal requests. She has also entered menarche and started to menstruate. In addition, ancillary studies showed the following: (i) the presence of brain electrical activity; (ii) evidence of blood flow to the brain; (iii) damage to the corpus callosum and pons, but vast areas of the brain are structurally preserved; and (iv) changes in Jahi's heart rate in response to her mother's voice. Jahi's clinical and laboratory data were reviewed by four neurologists: Drs. Alan Shewmon, Calixto Machado, Charles Prestigiacomo, and Ivan Mikolaenko, all of whom gave sworn declarations that Jahi is not "brain-dead." Dr. Paul Fisher dismissed all the evidence, however, and still considered Jahi to be "brain-dead." See Luce (2015) Luce, John M. 2015. The uncommon case of Jahi McMath. *Chest* 147, no. 4: 1144–51. doi: 10.1378/chest.14-2227, as well as Matier and Ross (2014) Matier, Phillip, and Andrew Ross. 2014. Videos show mom coaxing, Jahi McMath moving. SFGate. October 3. http://www.sfgate.com/bayarea/article/Videos-show-Mom-coaxing-Jahi-McMath-moving-5797622.php., and McGovern (2015) McGovern, Celeste. 2015. Top neurologist: Jahi McMath is "no longer" dead. National Catholic register. November 30. http://www.ncregister.com/daily-news/top-neurologist-jahi-mcmath-is-no-longer-dead.. The sworn declarations of Shewmon, Machado, Prestigiacomo and Mikolaenko, as well as Fisher's rebuttal, are available on-line (Pope 2015 Pope, Thaddeus Mason. 2015. Jahi McMath—a dispute over brain death. Thaddeuspope.com.

http://thaddeuspope.com/jahimcmath.html.).

11 For a more detailed (albeit non-exhaustive) list of integrative functions observed in brain-dead survivors, see Shewmon (2001 Shewmon, D. Alan. 2001. The brain and somatic integration: Insights into the standard biological rationale for equating "brain death" with death. *Journal of Medicine and Philosophy* 26, no. 5: 457–78. doi: 10.1076/jmep.26.5.457.3000, 462–71).

12 The lower the GCS score, the worse the head injury. A brain-injured patient is classified as GCS 3 (that is, the lowest possible score) when at the initial assessment, he or she demonstrates no eye opening, no verbal response, and no motor response (flaccid).

13 In a way, the phenomenon of SBI reflects the holistic interconnection that inherently exists between the heart, lungs, circulatory system, and brain. Irrespective of the etiology, the pathophysiological process of SBI is basically the same, consisting of a complex inflammatory response to ischemia. In clinical practice, a therapeutic intervention which reflects this close interconnection is mild to moderate hypothermia, which has been recommended for both cardiac arrest and TBI. See Polderman et al. (2002) Polderman, Kees H., Rudi Tjong Tjin Joe, Saskia M. Peerdeman, William P. Vandertop, and Armand R.J. Girbes. 2002. Effects of therapeutic hypothermia on intracranial pressure and outcome in patients with severe head injury. *Intensive Care Medicine* 28, no. 11: 1563–73. doi: 10.1007/s00134-002-1511-3; Polderman (2008) Polderman, K.H. 2008. Hypothermia and neurological outcome after cardiac arrest: State of the art. *European Journal of Anaesthesiology* 25, Suppl 42: 23–30. doi: 10.1017/S026502150700333X; Sunde (2013) Sunde, Kjetil. 2013. Therapeutic hypothermia in cardiac arrest. *Revista Espanola de Cardiologia* 66, no. 5: 346–9. doi: 10.1016/j.recesp.2012.10.005.

14 See Neumar et al. (2008) Neumar, Robert W., Jerry P. Nolan, Christophe Adrie, Mayuki Aibiki, Robert A. Berg, Bernd W. Böttiger, Clifton Callaway, Robert S.B. Clark, Romergryko G. Geocadin, Edward C. Jauch, Karl B. Kern, Ivan Laurent, W.T. Longstreth, Raina M. Merchant, Peter Morley, Laurie J. Morrison, Vinay Nadkarni, Mary Ann Peberdy, Emanuel P. Rivers, Antonio Rodriguez-Nunez, Frank W. Sellke, Christian Spaulding, Kjetil Sunde, and Terry Vanden Hoek. 2008. Post-cardiac arrest syndrome: Epidemiology, pathophysiology, treatment, and prognostication. *Circulation* 118, no. 23: 2452–83. doi: 10.1161/CIRCULATIONAHA.108.190652; Mongardon et al. (2011) Mongardon, Nicolas, Florence Dumas, Sylvie Ricome, David Grimaldi, Tarik Hissem, Frédéric Pène, and Alain Cariou. 2011. Postcardiac arrest syndrome: From immediate resuscitation to long-term outcome. *Annals of Intensive Care* 1, no. 1: 1–11. doi: 10.1186/2110-5820-1-45. SBI due to cardiac arrest is part of the post-cardiac arrest syndrome which may occur in the post-resuscitation period. The severity of the syndrome parallels the time elapsed between the collapse of circulation and its re-establishment. The underlying complex pathophysiology is basically that of global ischemia/reperfusion with associated oxygen debt, which results in a generalized activation of the systemic inflammatory and coagulation pathways. The syndrome thus presents a set of stereotypic post-cardiac arrest complications (superimposed on the pre-existing pathology leading to the cardiac arrest) including: (i) myocardial dysfunction, (ii) brain injury, (iii) and multiple organ failure. The overall clinical picture is reminiscent of septic shock. As a severe systemic illness, the syndrome portends a poor prognosis, with 60–70% mortality despite aggressive therapeutic measures. Of those who survive, "69% were considered to have a good neurological recovery at discharge" (Mongardon et al. 2011 Mongardon, Nicolas, Florence Dumas, Sylvie Ricome, David Grimaldi, Tarik Hissem, Frédéric Pène, and Alain Cariou. 2011. Postcardiac arrest syndrome: From immediate resuscitation to long-term outcome. *Annals of Intensive Care* 1, no. 1: 1–11. doi: 10.1186/2110-5820-1-45, 8).

15 Jahi McMath is a case in point in which unchecked hemorrhage led to cardiac arrest and "brain death." The 13-year-old patient was left to bleed profusely for five hours, after the surgical removal of her tonsils and adenoids, without any intervention from the responsible physicians. See Dreger and Haskell (2015) Dreger, Alice, and Helen Haskell. 2015. Was Jahi McMath's case preventable. Pacific Standard. March 13. http://www.psmag.com/health-and-behavior/was-jahi-mcmaths-case-preventable..

16 A conflict of interest is evident. Younger age group and absence of comorbidity are good prognostic

factors from the viewpoint of brain-injury management. But SBI individuals with these very characteristics are ideal donors from the viewpoint of "brain-death" advocates. See also notes 2 and 8 above.

17 Coimbra (2009b) Coimbra, Cicero Galli. 2009b. Are "brain dead" (or "brain-stem dead'') patients neurologically recoverable? In *Finis vitae: "brain death" is not true death*, ed. Roberto De Mattei, and Paul A. Byrne, 313–78. Oregon, Ohio: Life Guardian Foundation. recounts the story of a 15-year-old brain-injured female patient referred to as BBA. Not only was the patient not given aggressive neuro-intensive therapy for the reduction of brain edema, she was subjected to the apnea test twice, and declared "brain dead" less than 24 hours after admission. The family "declined repeated requests for organ donation," however, and succeeded in getting the patient transferred on the 5th day to a different hospital to be under the care of Dr. Coimbra. By then, the precious time window (namely, the first 48 hours after the onset of injury) for efficacious aggressive therapy, which might have brought the patient back to full recovery, was already lost. Under Coimbra's appropriate care, which included hormonal replacement for secondary thyroid and adrenal insufficiencies, and other measures, the patient's condition gradually stabilized. She lived on with chronic "brain death" for at least another 7 to 8 months.

18 See, for instance, Patel et al. (2002) Patel, Hiren C., David K. Menon, Susan Tebbs, Rebecca Hawker, Peter J. Hutchinson, and Peter J. Kirkpatrick. 2002. Specialist neurocritical care and outcome from head injury. *Intensive Care Medicine* 28, no. 5: 547–53. doi: 10.1007/s00134-002-1235-4.

19 Even the pro-"brain death" scholars attending the 2006 "The Signs of Death" conference organized by the Pontifical Academy of Sciences were concerned about the problem of the lack of consensus (Sorondo 2007 Sorondo, Marcelo Sánchez, ed. 2007. *The signs of death: The proceedings of the working group 11–12 September 2006*. Vatican City: Pontifical Academy of Sciences., lxiii, 43–48, 99, 176, 219). In presenting his paper at the conference, Bernat indicated that two of the three goals for the future are: (i) "an international consensus on the clinical tests for brain death," and (ii) "a consensus on the role of confirmatory testing in brain death" (Bernat 2007 Bernat, James L. 2007. How do physicians prove irreversibility in the determination of death? In *The signs of death: The proceedings of the working group 11–12 September 2006*, ed. Marcelo Sánchez Sorondo, 159–76. Vatican City: Pontifical Academy of Sciences., 176).

20 Statement made by Shewmon, cited in Diamond (2007 Diamond, Eugene F. 2007. John Paul II and brain death. *National Catholic Bioethics Quarterly* 7, no. 3: 491–7. doi: 10.5840/ncbq20077330, 495).

21 A case in point is Zack Dunlap who recovered after being declared brain dead in accordance with the established published guidelines (Morales 2008 Morales, Natalie. 2008. "Dead" man recovering after ATV accident. NBC News. March 23. http://www.nbcnews.com/id/23768436/ns/dateline_nbc-newsmakers/t/dead-man-recovering-after-atv-accident.). The patient heard what was being said about him (the claim that he was dead), but was physically unable to react.

22 For detailed discussion on the pathophysiology of traumatic brain injury, see Werner and Engelhard (2007) Werner, C., and K. Engelhard. 2007. Pathophysiology of traumatic brain injury. *British Journal of Anaesthesia* 99, no. 1: 4–9. doi: 10.1093/bja/aem131, as well Baxter and Wilson (2012) Baxter, David, and Mark Wilson. 2012. The fundamentals of head injury. *Surgery* (Oxford) 30, no. 3: 116–21. doi: 10.1016/j.mpsur.2011.12.002.

23 Patel (2007 Patel, Piyush. 2007. Brain protection—the clinical reality. *Revista Mexicana de Anestesiologia* 30, Suppl. 1: S101–7., S101). In humans, the normal CBF averages about 50 ml/100 mg/min.

24 Note the correspondence between the 60% of "brain dead" donors with nil to minimal damage of the brainstem on post-mortem examination, and the rougly 60% survival rate among patients with severe TBI as reported in the neuro-intesive literature; see Metz et al. (1996) Metz, Christoph, Matthias Holzschuh, Thomas Bein, Christian Woertgen, Anton Frey, Irmgard Frey, Kai Taeger, and Alexander

Brawanski. 1996. Moderate hypothermia in patients with severe head injury: Cerebral and extracerebral effects. *Journal of Neurosurgery* 85, no. 4: 533–41. doi: 10.3171/jns.1996.85.4.0533, Watanabe (1997) Watanabe, Yoshio. 1997. Once again on cardiac transplantation: Flaws in the logic of the proponents. *Japanese Heart Journal* 38, no. 5: 617–24. doi: 10.1536/ihj.38.617; Ley et al. (2011) Ley, Eric J., Morgan A. Clond, Omar N. Hussain, Marissa Srour, James Mirocha, Marko Bukur, Dan R. Margulies, and Ali Salim. 2011. Mortality by decade in trauma patients with Glasgow coma scale 3. *American Surgeon* 77, no. 10: 1342–5.; Patel et al. (2002) Patel, Hiren C., David K. Menon, Susan Tebbs, Rebecca Hawker, Peter J. Hutchinson, and Peter J. Kirkpatrick. 2002. Specialist neurocritical care and outcome from head injury. *Intensive Care Medicine* 28, no. 5: 547–53. doi: 10.1007/s00134-002-1235-4.

25 Intracranial blood flow studies are not necessarily as clear-cut as Bernat's categorical statement made it appear. For instance, transcranial Doppler has been praised for its high sensitivity and specificity with regard to "brain death" (Ducrocq et al. 1998 Ducrocq, Xavier, Marc Braun, Marc Debouverie, Christel Junges, Mireille Hummer, and Hervé Vespignani. 1998. Brain death and transcranial doppler: Experience in 130 cases of brain dead patients. *Journal of the Neurological Sciences* 160, no. 1: 41–6. doi: 10.1016/S0022-510X(98)00188-9). However, recent studies have reported persistence of blood flow in "brain death" (Cabrer et al. 2003 Cabrer, C., J.M. Domínguez-Roldan, M. Manyalich, E. Trias, D. Paredes, A. Navarro, J. Nicolás, R. Valero, C. García, A. Ruiz, and A. Vilarrodona. 2003. Persistence of intracranial diastolic flow in transcranial doppler sonography exploration of patients in brain death. *Transplantation Proceedings* 35, no. 5: 1642–3. doi: 10.1016/S0041-1345(03)00692-4; de Freitas et al. 2003 de Freitas, G.R., C. André, M. Bezerra, R.G. Nunes, and M. Vincent. 2003. Persistence of isolated flow in the internal carotid artery in brain death. *Journal of the Neurological Sciences* 210, no. 1–2: 31–4. doi: 10.1016/S0022-510X(03)00065-0). In these studies, even though the presence of intracranial blood flow (not accounted for by any other factor) contradicted the notion of "whole brain death," the diagnosis of "brain death" remained unchanged. Even Wijdicks recognized that "our experience with cerebral blood flow is still limited with insufficient validation" (Sorondo 2007 Sorondo, Marcelo Sánchez, ed. 2007. *The signs of death: The proceedings of the working group 11–12 September 2006*. Vatican City: Pontifical Academy of Sciences., 178). The PET (positron emission tomography), if it were to be performed, would probably be the best test for "brain death," since it measures brain metabolism (its glucose uptake).

26 The case of Zack Dunlap is another example of a patient who recovered to normal life even though he failed the apnea test (Morales 2008 Morales, Natalie. 2008. "Dead" man recovering after ATV accident. NBC News. March 23. http://www.nbcnews.com/id/23768436/ns/dateline_nbc-newsmakers/t/dead-man-recovering-after-atv-accident.).

27 A case in point are physicians who condone abortion and euthanasia/physician-assisted suicide, thereby rejecting the tenets of the Hippocratic Oath.

28 Roles such as businessperson, scientist, proletarian, bureaucrat, or corporate executive.

29 For a full development on why medicine is a moral enterprise, see Pellegrino and Thomasma (1993) Pellegrino, Edmund D., and David C. Thomasma. 1993. *The virtues in medical practice*. New York: Oxford University Press., especially Chapter 3, "Medicine as a Moral Community."

30 Hippocrates, *The Oath*, quoted in Edmund D. Pellegrino (2001 Pellegrino, Edmund D. 2001. Physician-assisted suicide and euthanasia: Rebuttals of rebuttals—the moral prohibition remains. *Journal of Medicine & Philosophy* 26, no. 1: 93–100. doi: 10.1076/jmep.26.1.93.3034, 94).

31 In Hippocrates, *On the Art* III, we read: "First I will define what I conceive medicine to be. In general terms it is to do away with the sufferings of the sick, to lessen the violence of their diseases and to refuse to treat those who are overmastered by their diseases realizing that in such cases medicine is powerless." Quoted in Pellegrino (2001 Pellegrino, Edmund D. 2001. Physician-assisted suicide and euthanasia: Rebuttals of rebuttals—the moral prohibition remains. *Journal of Medicine & Philosophy* 26, no. 1: 93–100. doi: 10.1076/jmep.26.1.93.3034, 94).

32 In article 5 of the 2000 address, John Paul II specifically stated, “the [neurological] criterion adopted in more recent times for ascertaining the fact of death, namely the *complete and irreversible* [original emphasis] cessation of all brain activity, *if* [emphasis added] rigorously applied, *does not seem to conflict* [emphasis added] with the essential elements of a sound anthropology.” In the paragraph preceding this statement, the pope presupposed that there has been an international consensus on the parameters (that is, the clinical tests) of the “brain-death” standard. “Sound anthropology” refers to the understanding (stated in article 4) that death is “a single event” – the separation of the soul from the body – resulting in “the total disintegration of [the] … integrated whole that [was] the [human] person.”

33 An in-depth analysis (with philosophical discussion) of John Paul II's Address to the Transplatation Society is being treated in a manuscript in preparation.

34 Since Pope Benedict XVI is a non-medical person, it is reasonable to think that he used the term *cadavere* in the sense of a true corpse/cadaver, and not in the oxymoron sense of “heart-beating cadaver” promoted by “brain death” advocates. The pope has never explicitly expressed his personal thought on the matter of “brain death.” However, it is publicly known that the German philosopher Robert Spaemann is a close friend of Benedict XVI—it was at the pope's request that the Pontifical Academy of Sciences had to invite (at the last minute) Spaemann to the “The Signs of Death” conference (see Shewmon 2012b Shewmon, D. Alan. 2012b. You only die once: Why brain death is not the death of a human being; a reply to Nicholas Tonti-Filippini. *Communio* 39: 422–94., 484). Hence, it would not be unreasonable to think that Benedict XVI most likely shares the views which Spaemann holds with regard to “brain death” (see Spaemann 2007 Spaemann, Robert. 2007. Is brain death the death of the human being? On the current state of the debate. In *The signs of death: The proceedings of the working group 11–12 September 2006*, edited by Marcelo Sánchez Sorondo, 130–41. Vatican City: Pontifical Academy of Sciences.).

35 For most people, the understanding about organ donation is limited to the consent forms filled in at their state Department of Motor Vehicles, or online at regional organ procurement organization websites. The information provided on such websites is unidimensional, geared toward promoting donation and reinforcing consent. In particular there is no mention of any other options for end-of-life care (e.g., hospice). In that sense, it is difficult to say that the average lay person is fully informed when he or she signs the consent for organ donation after death. See Nair-Collins (2010 Nair-Collins, Mike. 2010. Death, brain death, and the limits of science: Why the whole-brain concept of death is a flawed public policy. *Journal of Law, Medicine & Ethics* 38, no. 3: 667–83. doi: 10.1111/j.1748-720X.2010.00520.x, 677–8).

36 Unbiased information on the donation experience of families of brain-dead donors is extremely scarce. After the donation process, some have experienced the guilt of having left their loved one to be treated like “a used car to be parted out with [their] permission.” See Bartucci (1987 Bartucci, Marilyn Rossman. 1987. Organ donation: A study of the donor family perspective. *Journal of Neuroscience Nursing* 19, no. 6: 305–9. doi: 10.1097/01376517-198712000-00005, 307). More recently, the large study by Ralph et al. (2014 Ralph, A., J.R. Chapman, J. Gillis, J.C. Craig, P. Butow, K. Howard, M. Irving, B. Sutanto, and A. Tong. 2014. Family perspectives on deceased organ donation: Thematic synthesis of qualitative studies. *American Journal of Transplantation* 14, no. 4: 923–35. doi: 10.1111/ajt.12660, 935) also reveals that despite the positive effect for having consented to the lifesaving act of donation, grieving families experience “an overwhelming sense of uncertainty about death and the donation process.” For a personal statement from a bereaved mother, see White (2009) White, Hilary. 2009. “Brain death” as criteria for organ donation is a “deception”: Bereaved mother. Catholic Exchange. February 24. http://catholicexchange.com/brain-death-as-criteria-for-organ-donation-is-a-deception-bereaved-mother..

37 To establish that the loss of neurological functions is indeed irreversible would require at least two determinations, and these should not be too close to one another in time. In practice, however, in most institutions, only one determination is carried out. It is deemed that to lengthen the time of observation

and to perform a "second brain death examination [would] negatively affect organ donation." See Lustbader et al. (2011) Lustbader, D., D. O'Hara, E.F.M. Wijdicks, L. Maclean, W. Tajik, A. Ying, E. Berg, and M. Goldstein. 2011. Second brain death examination may negatively affect organ donation. *Neurology* 76, no. 2: 119–24. doi: 10.1212/WNL.0b013e3182061b0c.

38 Every organ in the body, once injured, requires a good amount of time to recover. The more severe the injury and the more vulnerable or vital the organ is, the more time it will take, not in terms of hours or days, but rather weeks.

39 Cerebral blood flow is regulated by CPP (the difference between the mean arterial pressure driving the blood in and the ICP forcing the blood out, divided by the cerebral vascular resistance). See Marik et al. (1999 Marik, Paul, Karen Chen, Joseph Varon, Robert FrommJr, and George L. Sternbach. 1999. Management of increased intracranial pressure: A review for clinicians. *Journal of Emergency Medicine* 17, no. 4: 711–19. doi: 10.1016/S0736-4679(99)00055-4, 712).

40 See Metz et al. (1996) Metz, Christoph, Matthias Holzschuh, Thomas Bein, Christian Woertgen, Anton Frey, Irmgard Frey, Kai Taeger, and Alexander Brawanski. 1996. Moderate hypothermia in patients with severe head injury: Cerebral and extracerebral effects. *Journal of Neurosurgery* 85, no. 4: 533–41. doi: 10.3171/jns.1996.85.4.0533; Watanabe (1997) Watanabe, Yoshio. 1997. Once again on cardiac transplantation: Flaws in the logic of the proponents. *Japanese Heart Journal* 38, no. 5: 617–24. doi: 10.1536/ihj.38.617; Marion et al. (1997) Marion, Donald W., Louis E. Penrod, Sheryl F. Kelsey, Walter D. Obrist, Patrick M. Kochanek, Alan M. Palmer, Stephen R. Wisniewski, Steven T. Dekosky, Donald W. Marion, Louis E. Penrod, Sheryl F. Kelsey, Walter D. Obrist, Patrick M. Kochanek, Alan M. Palmer, Stephen R. Wisniewski, and Steven T. DeKosky. 1997. Treatment of traumatic brain injury with moderate hypothermia. *New England Journal of Medicine* 336, no. 8: 540–46. doi: 10.1056/NEJM199702203360803; Bernard et al. (2002) Bernard, Stephen A., Timothy W. Gray, Michael D. Buist, Bruce M. Jones, William Silvester, Geoff Gutteridge, and Karen Smith. 2002. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. *New England Journal of Medicine* 346, no. 8: 557–63. doi: 10.1056/NEJMoa003289; Polderman et al. (2002) Polderman, Kees H., Rudi Tjong Tjin Joe, Saskia M. Peerdeman, William P. Vandertop, and Armand R.J. Girbes. 2002. Effects of therapeutic hypothermia on intracranial pressure and outcome in patients with severe head injury. *Intensive Care Medicine* 28, no. 11: 1563–73. doi: 10.1007/s00134-002-1511-3; Gal et al. (2002) Gal, Roman, Ivan Cundrle, Iveta Zimova, and Martin Smrcka. 2002. Mild hypothermia therapy for patients with severe brain injury. *Clinical Neurology and Neurosurgery* 104, no. 4: 318–21. doi: 10.1016/S0303-8467(02)00023-9; Zhi, Zhang, and Lin (2003) Zhi, Dashi, Sai Zhang, and Xin Lin. 2003. Study on therapeutic mechanism and clinical effect of mild hypothermia in patients with severe head injury. *Surgical Neurology* 59, no. 5: 381–5. doi: 10.1016/S0090-3019(03)00148-4; Polderman (2008) Polderman, K.H. 2008. Hypothermia and neurological outcome after cardiac arrest: State of the art. *European Journal of Anaesthesiology* 25, Suppl 42: 23–30. doi: 10.1017/S026502150700333X; Neumar et al. Neumar, Robert W., Jerry P. Nolan, Christophe Adrie, Mayuki Aibiki, Robert A. Berg, Bernd W. Böttiger, Clifton Callaway, Robert S.B. Clark, Romergryko G. Geocadin, Edward C. Jauch, Karl B. Kern, Ivan Laurent, W.T. Longstreth, Raina M. Merchant, Peter Morley, Laurie J. Morrison, Vinay Nadkarni, Mary Ann Peberdy, Emanuel P. Rivers, Antonio Rodriguez-Nunez, Frank W. Sellke, Christian Spaulding, Kjetil Sunde, and Terry Vanden Hoek. 2008. Post-cardiac arrest syndrome: Epidemiology, pathophysiology, treatment, and prognostication. *Circulation* 118, no. 23: 2452–83. doi: 10.1161/CIRCULATIONAHA.108.190652 (2008 Neumar, Robert W., Jerry P. Nolan, Christophe Adrie, Mayuki Aibiki, Robert A. Berg, Bernd W. Böttiger, Clifton Callaway, Robert S.B. Clark, Romergryko G. Geocadin, Edward C. Jauch, Karl B. Kern, Ivan Laurent, W.T. Longstreth, Raina M. Merchant, Peter Morley, Laurie J. Morrison, Vinay Nadkarni, Mary Ann Peberdy, Emanuel P. Rivers, Antonio Rodriguez-Nunez, Frank W. Sellke, Christian Spaulding, Kjetil Sunde, and Terry Vanden Hoek. 2008. Post-cardiac arrest syndrome: Epidemiology, pathophysiology, treatment, and prognostication. *Circulation* 118, no. 23: 2452–83. doi: 10.1161/CIRCULATIONAHA.108.190652, 2463–2464); Sahuquillo et al. (2009) Sahuquillo, Juan, Jon Pérez-Bárcena, Alberto Biestro, Elizabeth Zavala, Mari-Angels Merino, Anna Vilalta, Maria Antonia

Poca, Angel Garnacho, Ramon Adalia, Javier Homar, and Juan Antonio Llompart-Pou. 2009. Intravascular cooling for rapid induction of moderate hypothermia in severely head-injured patients: Results of a multicenter study (Intracool). *Intensive Care Medicine* 35, no. 5: 890–8. doi: 10.1007/s00134-008-1357-4; Fox et al. (2010) Fox, James L., Erik N. Vu, Mary Doyle-Waters, Jeffrey R. Brubacher, Riyad Abu-Laban, and Zengxuan Hu. 2010. Prophylactic hypothermia for traumatic brain injury: A quantitative systematic review. *CJEM: Journal of the Canadian Association of Emergency Physicians* 12, no. 4: 355–64. doi: 10.1017/S1481803500012471; Lee et al. (2010) Lee, Han-Chung, Hao-Che Chuang, Der-Yang Cho, Kuang-Fu Cheng, Pao-Hsuan Lin, and Chun-Chung Chen. 2010. Applying cerebral hypothermia and brain oxygen monitoring in treating severe traumatic brain injury. *World Neurosurgery* 74, no. 6: 654–60. doi: 10.1016/j.wneu.2010.06.019, Hayashi (2009) Hayashi, Nariyuki. 2009. Management of pitfalls for the successful clinical use of Hypothermia treatment. *Journal of Neurotrauma* 26, no. 3: 445–53. doi: 10.1089/neu.2008.0648; Peberdy et al. (2010 Peberdy, Mary Ann, Clifton W. Callaway, Robert W. Neumar, Romergryko G. Geocadin, Janice L. Zimmerman, Michael Donnino, Andrea Gabrielli, Scott M. Silvers, Arno L. Zaritsky, Raina Merchant, Terry L. Vanden Hoek, and Steven L. Kronick. 2010. Part 9: Post-cardiac arrest care: 2010 American heart association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation* 122, no. 18 Suppl 3: S768–86. doi: 10.1161/CIRCULATIONAHA.110.971002, S771–2). The smaller studies like those reported by Metz or Watanabe provided "raw" patient data (e.g., the GCS level of individual patients), which is usually not available from the larger studies.

41 The two patients reported by Watanabe and Coimbra are typical examples of patients with severe TBI who were not given the required ICP/CPP-directed therapy because they were seen as potential organ donors. The patient reported by Coimbra was even subjected to two apnea tests.

42 According to Robert Veatch (2004 Veatch, Robert M. 2004. Abandon the dead donor rule or change the definition of death? *Kennedy Institute of Ethics Journal* 14, no. 3: 261–76. doi: 10.1353/ken.2004.0035, 267–8), who worked closely with the Harvard Committee members as a graduate student, "*none of the members was so naive as to believe that people with dead brains were dead in the traditional biological sense of the irreversible loss of bodily integration*. … Rather, committee members implicitly held that, *even though these people are not dead in the traditional biological sense, they have lost the moral status of members of the human moral community*. They believed that people with dead brains *no longer should be protected by norms prohibiting homicide.* … In effect, the committee and its fellow travelers proposed an entirely new definition of death, one that assigned the label 'death' for social and policy purposes to people who no longer are seen as having the full moral standing assigned to other humans. … Among the implications would be that organs that normally preserve life could be removed without the elaborate moral defense normally necessary to justify a homicide" (emphasis added).

43 According to the teaching of St. Thomas Aquinas, truth has to do with the conformity of our intellect to the reality (the real properties) of "a thing known." The proper notion of truth is encapsulated in the well-known expression "*veritas est adaequatio rei et intellectus*." With respect to natural "things," our intellect gets its scientific knowledge from the "things" themselves. Therefore, our intellect is measured by natural "things," and not vice versa; the human intellect measures only man-made things. Natural "things" are measured only by the divine intellect that has created them. See Thomas Aquinas (1952 Aquinas, Thomas. 1952. *Truth: A translation of* Quaestiones Disputatae De Veritate, *Volume I, Questions I–IX*. translated by Robert W. Mulligan. Chicago: Henry Regnery Company. http://dhspriory.org/thomas/QDdeVer1.htm#2., q. 1, a. 2).