

Brain Death: What Physicians Need to Know

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Brain death (BD) is both a concept and a determination of death by neurological criteria. Introduced by the Ad Hoc Committee of the Harvard Medical School (“Harvard Committee” or “Committee”), the criteria entered clinical practice in 1968.¹ It was not until 1981, however, that the President’s Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research (“President’s Commission”) advanced both a post-hoc conceptual rationale for BD and the Uniform Determination of Death Act (UDDA). The UDDA gives BD a firm legal standing in the U.S., as it makes BD equivalent to the traditional circulatory-respiratory standard for the determination of death.²

BD has raised mounting scholarly criticism ever since its inception. In recent years, the contention has extended from academic circles into the courtrooms, as attested by increasing numbers of legal actions, some of which have received attention in the national media—such as the cases of Aden Hailu and Jahi McMath in 2015 and 2019, respectively. Because brain-dead donors constitute the primary source of transplantable organs, such legal challenges are seen by BD proponents and organ procurement organizations as a cause for concern, undermining public confidence in the BD paradigm.³

Consequently, since late 2019, pro-BD scholars have been insistently advocating for a radical revision of the UDDA whereby the current medicolegal definition of BD—“the irreversible cessation of all functions of the entire brain, including the brain stem”²—would be changed to a formulation that is subordinate to the BD diagnostic guidelines promoted by the American Academy of Neurology (AAN) and other medical stakeholders in BD determination.⁴⁻⁶ The proposal for such a revision was presented to the Uniform Law Commission (ULC) in July 2020.⁷ As of this writing, the ULC has created a drafting committee to work on a revision of the UDDA.⁶

This paper has a two-fold aim: (1) to provide a critical overview of the most salient aspects of BD and its controversies, and (2) to alert the medical community to the serious ethical significance of revising the UDDA.

The Harvard Report—the Birth of Brain Death

The reference point for understanding BD is the concept of death itself. Death is a biological phenomenon. It is a permanent and irreversible physical condition in which the dead body (the corpse) undergoes progressive somatic disintegration as relentless entropy sets in at the moment of death. As pointed out by pro-BD scholars Culver and Gert, biological death applies “equally to related species. When we talk of the death of a human being, we mean the same thing as we do when we talk of the death of a dog or a cat.”⁸ They share the same

constellation of signs indicative of somatic disintegration that result from the *complete irreversible cessation of all vital bodily functions*. This holistic understanding of death, which reflects the fact that death pertains to the entire organism and not to any one part, remains valid today. It was clearly expressed in the definitions of death found in medical dictionaries prior to 1968.

As noted by William Arnet, “these traditional medical definitions do not isolate the function of any one organ; rather, they emphasize the total stoppage of all vital bodily functions,...as evidenced by absence of heartbeat and respiration,...beyond the possibility of resuscitation.”⁹ Heartbeat and breathing were singled out because of the well-established dependence of all bodily functions on circulation and respiration. This is known as the traditional cardiopulmonary standard or the circulatory-respiratory criterion for death.

The Harvard Committee’s introduction of BD marked a major paradigm shift in the medical definition of death. The opening sentence of its report—“our primary purpose is to define irreversible coma as a new criterion for death”¹—clearly indicates what BD truly is, namely, that *BD is none other than a state of coma*, deemed to be irreversible based on a set of clinical tests (diagnostic criteria) set forth by the Committee. Most of the medical community quickly accepted this new definition of death despite two major problematic issues in the Harvard report:

(1) The Committee gave no conceptual rationale to explain why a patient in irreversible coma should be declared dead. A post-hoc rationale was not to come until 1981 (see below).

(2) There were no patient data or clinical studies showing that the diagnostic tests for establishing BD and equating it with death had been properly validated prior to their clinical usage.

Put differently, the introduction of BD was based solely on the opinion of the 13 members of the Harvard Committee and not on any valid scientific basis.¹⁰ The Harvard Committee essentially stretched the definition of the term “death” to include the phenomenon of irreversible coma (or changed the meaning of the term “irreversible coma” to make it a new criterion of death).¹¹ In doing so, however, the Committee could not in any way change the reality of these two diverse phenomena. This is why BD has stirred unrelenting controversies since its inception.

One of the two reasons given by the Committee for redefining irreversible coma as death was the burden posed by the comatose patients on themselves, their families, and the hospitals.¹ However, according to medical historians, analyses of the correspondence among the Committee members and the manuscript-drafts of the Harvard report have revealed that the real reason was none other than organ

transplantation.^{12,13} For instance, a passage in the penultimate draft read: “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.”¹²

The diagnostic test-criteria for BD listed in the Harvard report are as follows: (1) complete unresponsiveness even to the most painful stimuli; (2) no spontaneous breathing as documented by the apnea test, (3) “no spontaneous muscular movements”; (4) no reflexes, i.e., brainstem reflexes are absent, plus “as a rule the stretch tendon reflexes cannot be elicited”; and (5) a flat encephalogram (EEG).¹ Thus, based on these criteria, the diagnosis of BD requires that the whole central nervous system (CNS)—both the brain and spinal cord—be silent. For reasons that will become clear below, these criteria have since undergone substantial modifications.

The President’s Commission and the Uniform Determination of Death Act

Between 1968 and 1981, the legal adoption of the BD paradigm by state legislatures produced a patchwork of conflicting new and old methods for determining death. The need for both a uniform criterion and a conceptual rationale to justify BD prompted the President’s Commission to issue a report that endorsed the Harvard standard and led to the promulgation of the UDDA. The UDDA or some variant thereof quickly became the legal definition of death in all 50 states.

The President’s Commission provided a conceptual rationale for BD by adopting the thesis of “whole BD” advanced by Julius Korein and James Bernat, according to which the brain is the master integrator of the body and BD is biological death. This thesis made a two-fold claim that: (1) the brain is “responsible for the function of the organism as a whole: the integration of organ and tissue subsystems by neural and neuroendocrine control of temperature, fluids and electrolytes, nutrition, breathing, circulation, appropriate responses to danger, among others,”¹⁴ and (2) as such, it is the “single critical vital system” of which the complete and irreversible loss indicates death.¹⁵

The President’s Commission thus argued that BD is the same biological state as death determined by the traditional circulatory-respiratory standard, and that the signs of death in BD are temporarily “masked” by the ventilator and life-support measures.² Stated differently, according to this view, the body of a brain-dead patient is a corpse that appears alive (i.e., somatically integrated) only because of the work of the ventilator and intensive care measures.

Note, however, that both arguments—that the brain is the “central integrator of the body” and that the intensive care masks the disintegration of the alleged brain-dead corpse—are demonstrably false. The brain is *a part* of the body. According to a fundamental two-fold principle in biology, a living organic whole (an organism as a whole) is greater than the sum of its parts and is ontologically prior to its parts.¹¹ In the embryo, the neural groove from which the brain will develop does not form until the fourth week of gestation. It is thus self-evident that no part (in this case, the brain) can integrate itself, let alone integrate an organic whole (a human

being). Furthermore, the ventilator only blows air in and out of the lungs; it has no role in the exchange of oxygen and carbon dioxide or in any other vegetative function such as circulation, assimilation of nutrients, and excretion of waste.¹⁶ Neither the ventilator nor the intensive care interventions provided to a brain-dead patient have any power in and of themselves to “integrate” a body or stop the dead body from disintegrating.

Despite these fundamental flaws, the President’s Commission’s report culminated in the promulgation of the UDDA which states: “An individual who has sustained either (1) irreversible cessation of circulatory and respiratory functions or (2) irreversible cessation of all functions of the entire brain, including the brainstem, is dead. A determination of death must be made in accordance with accepted medical standards.”² Because the UDDA makes the whole BD standard equivalent to the traditional circulatory-respiratory standard, *patients declared brain dead are considered biologically and legally dead when, in fact, they are not.*

In formulating the UDDA, the President’s Commission was judicious to recognize “that the definition contained in the statute ought to address general physiological standards rather than medical criteria and tests, which will change with advances in biomedical knowledge and refinements in technique.”² However, the clause “in accordance with accepted medical standards” has made it possible for the AAN and medical stakeholders in BD determination to alter the diagnostic test-criteria of BD, even to the point of contradicting the UDDA definition of BD itself, as we shall see now.

Modifying the Criteria of Brain Death: from Whole Brain Death to Brainstem Death

During the 1980s and 1990s, certain inconvenient truths about BD have come to light in terms of both concept and determination. With respect to the concept of BD, Shewmon’s series of “chronic BD” cases—patients who survived for weeks, months, or years after being declared brain-dead, even in cases where total brain destruction could be demonstrated¹⁷—constitute irrefutable evidence that falsifies the conceptual rationale that the brain is the central integrator of the body and that BD is biological death.

With respect to the determination of BD, many reports have documented that patients who are declared brain-dead may still retain the following functions pertaining to the CNS:

(1) **Persistent brain wave activity** in patients declared brain-dead by clinical criteria (i.e., based solely on a bedside neurological examination):¹⁸ for example, Grigg and colleagues reported a series of 56 consecutive brain-dead patients of whom 11 demonstrated persistent EEG activity documented up to 168 hours after the declaration of BD.¹⁹ Faced with such inconvenient findings that contradicted the UDDA requirement of “irreversible cessation of all functions of the entire brain,” BD proponents declared, without any scientific evidence, that the activity was due to random nests of insignificant neurons and could be ignored.^{19,20} Here, one may ask: On which criteria can one judge that some nests of neurons are significant or insignificant? How does one establish the numbers and locations of such “insignificant nests” that can be allowed in BD? Based on the aforementioned

reasoning, the pro-BD medical community decided that EEG testing (required by the Harvard standard) is optional and no longer required for BD determination.

(2) **Persistent secretion of antidiuretic hormone (ADH)** by the hypothalamus-pituitary complex:¹⁸ ADH plays a critical integrative function in water-electrolyte homeostasis and hemodynamic stability. Its presence provides the best indicator of preserved brain function since loss of ADH secretion produces diabetes insipidus. According to Nair-Collins's recent literature review, half of the patients declared brain dead did not have diabetes insipidus.²¹ Nevertheless, the pro-BD medical community decided to ignore the role of ADH,²² even though this neuro-hormone is physiologically more vital for life than any brainstem reflex. This decision directly contradicts the UDDA because a patient with preserved ADH secretion cannot in any way meet the UDDA definition of BD, the "irreversible cessation of all functions of the entire brain."²

(3) **Persistent reflexes and movements:** Though the Harvard standard required complete silence of the whole CNS, many reports have documented the occurrence among brain-dead patients of various types of stretch tendon reflexes, spontaneous movements²³⁻²⁵ (found in 50% of the patients),²⁶ and autonomic reflexes such as "dramatic increases in blood pressure and heart rate" in response to surgical incision and sternotomy for organ removal.²⁷⁻²⁸ Based merely on their assertion that the spinal cord plays no critical integrative role, BD proponents argued that reflexes and spontaneous movements in brain-dead patients are insignificant and do not invalidate the diagnosis of BD because they originate from the spinal cord.²⁹ Yet, one can legitimately ask: On what basis can the integrative functions of the spinal cord be deemed insignificant, especially when high spinal cord injury produces irreversible apnea, cardiovascular instability, and poikilothermia—symptoms identical to those observed in BD?³⁰ Moreover, since the spinal cord is in full continuity with the brain with "neural tracts running in both directions, then why do the reflexes above the foramen magnum (brainstem reflexes) qualify as critical and clinical functions, while those below it (spinal reflexes) are dismissed as irrelevant?"¹¹

In sum, what the pro-BD community has done is basically a selective discarding of various physiological functions that, by their persistence in brain-dead patients, represent a challenge to the validity of whole BD. This process of selective discarding culminated in the AAN promulgating its guidelines which, ever since 1995, have become the diagnostic guidelines for establishing BD.^{31,32} The key features of the AAN guidelines are as follows: (1) EEG and cerebral blood flow studies are not required, except when the apnea test is medically not feasible. A bedside neurologic examination alone is sufficient for BD determination. (2) Persistent secretion of ADH by the hypothalamic-pituitary complex, as evidenced by normal blood pressure and absence of diabetes insipidus, is compatible with BD. (3) Spontaneous movements, stretch reflexes of the limbs, as well as lacrimation, sweating, blushing, tachycardia, and sudden increases in blood pressure, are all compatible with BD.

The AAN guidelines diverge from both the Harvard standard and the UDDA. With its emphatic recommendation

that a bedside neurologic examination alone is sufficient for BD determination, the guidelines contradict the UDDA stipulation that *all* functions of the *entire* brain must be lost. Neurologic bedside examinations can only evaluate some of the brainstem functions—namely, the level of arousal, brainstem reflexes, and apnea. Therefore, the diagnostic criteria of the AAN guidelines are not those of whole BD, but of brainstem death (BSD) instead. BSD, also referred to as "apneic unconsciousness," is a standard used in the UK.³³ Unlike whole BD, however, BSD has never been claimed to be biological death. Rather, it is a variation of a personhood-based or consciousness-based determination of death.³⁴

It is rather specious that, on the one hand, BD proponents insist that BD is whole BD and claim it to be biological death, and on the other hand, in practice, they limit the criteria for the determination of BD to those of BSD. This is one of the main reasons why BD has stirred unrelenting contention that, in recent years, has moved from academic circles into the courtrooms. At the heart of the contention lies this crucial question: if BD is biological death, how is it possible that the alleged brain-dead "corpse" manifests brain wave activity, ADH secretion, autonomic reflexes, and spontaneous movements/reflexes of the limbs?

An Incoherent Proposal for a Revision of the Uniform Determination of Death Act

Though vigorously promoted by the medical and legal communities for decades, the practice of BD is facing a rise in public distrust as evidenced by increased numbers of lawsuits filed by families. The reason is rather obvious: families of patients declared brain-dead can see with their own eyes that their loved ones continued to manifest many overt signs of the living (warm and pink flesh and excretion of urine, among others). They also see the stark difference in the appearance of their loved ones before and after organ removal.³⁵

For BD proponents, however, the aforementioned increase in public distrust has to do with lack of uniformity in BD laws among the states and this, in turn, is caused by the UDDA.^{4,5,36} In other words, the UDDA itself is to be blamed for rising litigations against BD. For this reason, in 2016 the AAN convened a summit to advocate for the revision of the UDDA.³⁷ This led to the proposal of a revised UDDA (RUDDA) by Ariane Lewis and colleagues, which includes three specific elements:^{4,5} (1) a change in the definition of BD to the "irreversible cessation of functions of the entire brain, including the brainstem, leading to unresponsive coma with loss of capacity for consciousness, brainstem areflexia and the inability to breathe spontaneously"; (2) a mandate that the AAN guidelines for adults as well as the joint society pediatric guidelines, and all future revisions thereof, be the medical standard for BD determination; and (3) an explicit authorization that BD determinations be made without the consent of families.

The proposed RUDDA is seriously flawed for the following reasons:

(1) Intrinsic inconsistency: the RUDDA defines BD as the "irreversible cessation of functions of the *entire* brain," and, concomitantly, restricts the definition to three specific

functions (consciousness, brainstem reflexes, and breathing). As previously discussed, this triad of unresponsiveness, brainstem areflexia, and apnea is none other than the diagnostic criteria of the UK-BSD standard. Note that the phrase “loss of the capacity for consciousness” in the RUDDA refers to unresponsiveness and not unconsciousness *per se*. Consciousness involves two dimensions: (a) the level of consciousness, i.e., arousal (responsiveness to stimuli), which can be assessed by an observer and (b) the content of consciousness, i.e., awareness, which is a first-person experience inaccessible to third-party observers.^{11,38} Arousal and awareness do not necessarily go hand in hand; a person can be fully aware yet unarousable. Currently, “there is no reliable way to distinguish unresponsive patients who are inwardly conscious from those who are not.”³⁹

(2) It designates the AAN guidelines and the joint society pediatric guidelines, and all future revisions thereof, as the statutorily mandated medical standard for the determination of death. As mentioned earlier, these guidelines omit ancillary testing and specifically exclude the function of the hypothalamic-pituitary complex as a brain function. Consequently, they carry a significant risk of false positive diagnosis whereby patients who still retain brain functions are declared brain-dead and, therefore, dead.³⁹ The percentage of false positives is not insignificant, as attested by reports of brain-dead patients with persistent EEG activity or evoked potentials, persistent brain perfusion and, most importantly, persistent ADH secretion.^{18,19,21,39} Moreover, from a societal standpoint, it seems plainly unwise to impose on the general public the guidelines of a private medical organization (the AAN) as a nationwide standard for the legal determination of death. If anything, the medical practice of determining BD according to the AAN guidelines should be brought into line with the law (the UDDA), since the AAN guidelines do not conform to the UDDA statutory definition of BD.

(3) The RUDDA stipulates that BD determination can be performed without the consent of families. Its proponents argue that BD is equivalent to traditional death determined by the circulatory-respiratory standard; since consent is not required for the latter, it should not be required for BD determination either.^{4,36} In addition, this argument appeals to the results of two surveys, according to which 78% of 201 adult neurologists and 72% of 197 pediatric neurologists opined that it is not necessary to obtain consent before performing an evaluation for BD.^{40,41} It also appeals to the fact that U.S. law (including the UDDA and state regulations) do not require consent for BD testing.⁴² However, both appeals to the majority and to authority are very weak arguments since they are well-known logical fallacies. More importantly, the issue at stake is that apnea testing is a crucial component for the diagnosis of BD since, according to the AAN guidelines, “the three cardinal findings in BD are coma or unresponsiveness, absence of brainstem reflexes and apnea.”³¹ As will be shown in the excursus below, “apnea testing is a procedure with well-known and potentially life-threatening risks” [emphasis in original].⁴³ This alone should be an indication that an explicit informed consent must be obtained from the patient’s family or proxy prior to apnea testing.

One may wonder what the impact would be on families

of brain-dead patients if the UDDA were to be revised along the lines of Lewis and colleagues’ RUDDA. If the AAN and joint society pediatric guidelines (and all their future revisions) were to become the statutory medical standard, and if physicians were allowed to perform BD determination without consent, families would be precluded from objecting to the declaration of BD, both at the bedside and in the courtroom.

Excursus: Apnea Testing and its Complications

According to the current guidelines for BD determination,³² the apnea test is performed to evaluate the functionality of the respiratory centers in the lower brainstem (the medulla) in response to a rising arterial carbon dioxide partial pressure (PaCO₂). The guidelines require that the deeply comatose patient have stable normal blood pressure and be pre-oxygenated with 100% O₂ for at least 10 minutes to prevent hypoxemia. Based on the assumption that PaCO₂ rises at the rate of 3 mmHg/min, the patient is disconnected from the ventilator for 8 to 10 minutes to let PaCO₂ rise above the designated threshold of 60 mmHg or at least 20 mmHg above the baseline, while oxygenation is preserved via a cannula down the endotracheal tube delivering 100% O₂.

There are three points worth noting: (1) the apnea test has never been validated, (2) contrary to the aforementioned assumption, in practice the rate of PaCO₂ increase is unpredictable and highly variable, ranging from 0.5 to 10.5 mmHg/min, and (3) the PaCO₂ threshold required to maximally stimulate the chemoreceptors of the respiratory center remains unknown.^{31,44} Put differently, the current PaCO₂ threshold of 60 mmHg is based on expert consensus and not any scientific evidence. This lack of an evidentiary basis is of concern, as it could lead to a false positive declaration of death because, as Haun and colleagues pointed out, “it is not inconceivable that a patient could meet brain death criteria (utilizing a PaCO₂ of 8 kPa (60 mmHg) as the endpoint of apnoea testing) and still have medullary activity.”⁴⁴ This is not just a theoretical concern. The literature contains (1) six reported instances of apnea testing in which spontaneous breathing was initiated at PaCO₂ values of 112, 91, 77, 71, > 61, and > 60 mmHg, and (2) six patients who resumed breathing after positive apnea testing.⁴⁵ It remains unknown how many more such cases have gone unreported. Likewise, “the incidence of potential for return of breathing after positive apnea testing is unknowable, because virtually all patients with positive test results have support withdrawn or organs removed.”⁴⁵

BD advocates have consistently insisted that the apnea test is a “safe” procedure if performed according to the prescribed AAN guidelines.⁴⁶⁻⁴⁸ Nevertheless, as shown below, a close look at the data reported in the literature (including publications from authors who followed the updated AAN 2010 guidelines) reveal that the risk of complications is far from nil.

The complications reported in the literature can be categorized into two broad groups:

(1) **Barotrauma complications** such as pneumothorax,^{49,50} pneumomediastinum,⁴⁹ and pneumoperitoneum.^{49,50} These are rare complications, produced mechanically by the cannula for O₂ insufflation. It has been suggested that using a smaller-sized cannula, placing it toward the tip of the endotracheal

tube, and keeping a low O₂ flow rate should help to avoid the occurrence of barotrauma.⁴⁶

(2) **Hemodynamic complications** inherent to the nature of the apnea test itself: if the patient is apneic, both increased PaCO₂ (hypercarbia/hypercapnia) and respiratory acidosis will develop, which in turn lead to peripheral vasodilatation and cardiac depression. Thus, the most commonly reported complications include hypotension,^{47,48,51,52} bradycardia,⁵¹ cardiac arrhythmia,^{51,52} and cardiac arrest.^{51,52} Hypoxemia has also been reported;^{51,52} its occurrence can exacerbate hemodynamic and cardiac instabilities. The incidence of these complications varies from one study to the next. According to the 2013 review article by Scott et al. summarizing the data of nine published studies (six were from 2000 and later) on apnea testing performed on 608 patients, the incidences of the complications are as follows: (a) cardiac arrest: <1%–3%, (b) bradycardia: 3%; (c) arrhythmia: 1–10%; (d) hypotension: 1–43%, and (e) hypoxemia: 5–25%. Even when apnea testing was performed by experienced neurointensivists in a study of 63 patients, the complications of hypotension and hypoxemia still remained significant, at 17.4% and 6.3%, respectively.⁴⁷

In addition to the aforementioned complications, which can cause immediate harm to the patient, there exists a more insidious complication that may not be immediately recognized at the time of apnea testing, namely, exacerbation of brain damage caused by hypercarbia in the setting of severe brain injury. BD proponents have consistently dismissed this complication as theoretical.⁵⁴ Here, it worth noting that all the guidelines for the management of severe brain injury have consistently recommended that, as part of neuroprotective measures, O₂ and CO₂ levels be maintained within the normal range and that conditions leading to hypercarbia and acidosis should be avoided.⁵⁵⁻⁵⁸ In cases of hypoxic-ischemic brain injury, the guidelines also warn that hyperoxia can exacerbate neuronal injury.^{54,57} Yet, the very three things that must be avoided—hypercarbia, acidosis, and hyperoxia—are part of the apnea test. The management guidelines also recommend to avoid hypotension, itself a frequent complication of the apnea test. In what way, then, is apnea testing in conformity with the management guidelines of severe brain injury?

The danger of apnea testing to the already severely injured brain lies in the fact that hypercarbia can result in decreased cerebral perfusion pressure (CPP). CPP is mean arterial pressure (MAP) minus intracranial pressure (ICP). Either a decrease in MAP or a rise in ICP can result in decreased CPP. In normal subjects, cerebral autoregulation maintains CPP at a steady and adequate level, thus protecting the brain from fluctuations in arterial pressure.⁵⁹ However, this process is often impaired in severe brain injury.⁶⁰ Increased PaCO₂ causes vasodilation in the peripheral vasculature and can lead to hypotension. In the setting of markedly increased ICP, a slight decrease in MAP, while not sufficient to cause hypotension, could nevertheless lower CPP enough to exacerbate cerebral ischemia. Increased PaCO₂ also leads to vasodilation of cerebral blood vessels. This results in increased cerebral blood flow (cerebral blood volume), which in turns elevates ICP. Thus, in the context of severe brain injury with an already high ICP, it is conceivable that this additional increase in ICP could result in decreased CPP and, therefore, decreased cerebral blood flow which, in

turn, causes further ischemic brain injury.

In a study of 16 apnea tests on 13 patients, there were 13 instances of increased ICP during apnea testing, with ICP rising from 86.6 +/- 22.9 to 95 +/- 27.7 mmHg, and returning to 83.8 +/- 21 after the test. Even though MAP also rose during the test, from 95.4 +/- 21.6 to 108.5 +/- 20.5 mmHg, it dropped to lower levels after apnea testing (86.1 +/- 16.1 mmHg). The net result was a significant decrease in CPP (already low before the test) from 8.4 +/- 16.8 to 1 +/- 16.4 mmHg after the apnea test.⁶¹ In another study on 19 patients, “there was a progressive statistically significant decrease in MAP during apnea (from 77 +/- 10 to 63 +/- 11 mmHg).”⁶² In addition, “in animal models of brain injury, once the intracranial pressure rises above systemic blood pressure, even transiently, there is often a no-reflow phenomenon with collapse of the cerebral vasculature that is often not reversible by lowering the intracranial pressure again.”⁶³

Taken together, the above information should raise serious concern that apnea testing can “further damage a brain under high intracranial pressure with tenuous blood flow. Just a slight decrease in BP, which is insufficient to consider a hemodynamic complication, or a hypercarbia-induced slight increase in intracranial pressure could reduce blood flow critically to areas of the brain that are not already infarcted, possibly even precipitating intracranial circulatory arrest.”⁴⁵ As a result, patients who were not already brain-dead (but in the stage of global ischemic penumbra) “might subsequently be made brain dead as a result of the testing.”⁶⁴

How Should the UDDA Be Ethically Revised?

The ULC Drafting Committee is undertaking the task of formulating a revision of the UDDA. This is an opportunity for physicians to play an important role in addressing the ethical issues raised by BD, especially the false claim that BD alone is sufficient to signify death of the human person. Although BD diagnoses comprise only a small fraction of the total number of human deaths, it is nevertheless a serious legal injustice to allow living individuals to be called dead and be treated as such. As noted at the beginning of this essay, BD is—in the words of the Harvard report—an “irreversible coma.” But, as Shewmon correctly pointed out, “coma, whether reversible or not, is not biological death. One cannot say with semantic correctness that a cadaver or corpse is comatose.”³⁹

Therefore, the question at stake is: how should the UDDA be revised? Any revision of the UDDA, if it is to be ethical, must take into consideration the fact that death is a biological phenomenon whereby the death of a human being is no different from that of any other warm-blooded mammal.⁸ A revision of the UDDA can fall into one of the following three possible categories:

(1) The revision contains only the first arm of the UDDA, that which corresponds to the traditional circulatory-respiratory standard for the determination of biological death.

(2) The revision maintains the whole BD paradigm (i.e., the UDDA formulation remains essentially unchanged), but there must be an accompanying explicit indication that BD is legal death and not biological death.

(3) The revision consists of the aforementioned RUDDA,

which is none other than the UK-BSD standard, a variation of a personhood-based definition of death.

Of the above three approaches, only the first corresponds to the reality of biological death and, as such, is also in accord with sound philosophical and biological principles.⁴³ Thus, a revision according to the first approach would be most ethically acceptable. If either the second or third approach were to be chosen, however, certain amendments would need to be included to render the revision ethically acceptable. As previously pointed out by both Doyen Nguyen and Alan Shewmon, the amendments should include: (a) the requirement of informed consent prior to BD determination, and (b) an exemption from the diagnosis of BD on the basis of personal convictions (including but not limited to religious beliefs or moral convictions, since there are people who reject BD on scientific grounds).^{39,65}

Conclusion

Serious conceptual and moral difficulties with brain death have so far been carefully kept out of the mainstream of medical and societal discussions. Physicians and other medical professionals of good will could be instrumental in correcting the state of affairs if informed of the issues.

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