Title: A Dual Theory of Death: Making Sense of Brain Death as Bodily Death and Death of Consciousness

Executive Summary: A process-ontological paper proposing a dual-criteria framework for defining death through bodily and conscious cessation.

Abstract:

How are we to make sense of brain death? It seems initially clear that the brain is *the* irreplaceable organ, the one that holds our memories, our personality, ***all of the characteristics*** (aside from those of the body) which we typically identify with as the self. However, we can imagine that our memories have been erased, that our personality has flipped completely, that our characteristics are simply no more what they once were. These are the cases of amnesia, of frontal lobe lesions, of the split-brain patient. These cases are perceived as massive damages and distortions of the brain. And yet, in most of those cases we would generally consider ourselves to be *alive*. In contrast to the concept that the brain is the thing of importance in death, in edge cases, where either the brain survives but brain *processes* do not, or cases where some subset of brain processes survive but substantial parts of the brain do not, it becomes evident that there is more to the concept that brain is the self. Perhaps that there is another thing which we care about in considering what life is, in the way that is important to us. So, what is that thing that we so care about, which seemingly disappears in brain death, if any individual part of the brain is not of the utmost importance?

In this paper, we propose that what we care about and what is lost in brain death is not the brain itself, nor its entire set of functionalities, but rather is the ability and processes of experiencing, of being a consciousness, an experiencer. More specifically, we propose that brain death, and death in general, are best understood as the cessation of two processes, bodily processes, and the processes of consciousness.

We delineate a process-ontological view of life, consciousness, and death, and justify that view, as well as delineate some potential benefits of such a view over the more widely used concepts of life as a substance or being. We then go over some important implications, the first of which is that consciousness, as delineated in this general conceptualization of death, is an ongoing process; a system with certain dynamics, which much like a flame can be extinguished and restarted. This has implications for the coherence of the idea of death at the short and larger scale. We draw parallels and substantiate the similarities between sleep and death by appealing to clinical measures of brain death, a minimal neural-systems theory of consciousness, and some deductive arguments. We then review the mechanisms of select anesthetics, demonstrating that the effects of anesthetics are best described as a cessation of the dynamic we propose is identical to consciousness, and that, despite the various mechanisms of action of anesthetics, and the varying neural and global propagation dynamics induced by general anesthetics, all function to ensure the lack of a particular dynamic identical to consciousness. We conclude by reviewing our dual conceptualization of death, position brain death as a specific semi-composite death of consciousness, and finally discuss how this dual theory of death applies to various kinds of unconsciousness, and finally redefine a composite, third "Final Death" from the composite.

We have the capacity to do this, but the machines are expensive, and there are not sufficient staff, and what of the resources? What of the shareholders of medical centers? Those are all terribly, astronomically, metaphysically important things, but I would argue that the death of a person which is otherwise preventable is likely more important, to the point that it probably outweighs those things. If one is of a mind that this is not so, I would urge said person to take a paralytic and have someone cut into their

body sans anesthesia, to remove a kidney, while a family member does or does not try and scramble up the money to pay the operator to cease doing so. For TJ Hoover, who would have likely not been failed by the medical system quite so fantastically as it did if he were wealthy, it felt like that. If one doesn't wish to do that (completely understandable, and terribly expensive and impractical anyways), then imagine the horror of being in that scenario. If you are wealthy and know that this would never happen to you, I encourage you to look at your stock portfolio, and recall that if your local large hospital makes half a million on a day, it is not just a bad day, it is *an astronomically* bad day, and that an average (read, plebian treatment that everyone receives) day at an inpatient facility costs around 2,800.00 USD, and that the average stay is not for just one day. If you are *still* unconvinced that better screening criteria ought to be implemented and balk at the potential cost, I encourage you to go skiing this spring on the half-dome in Yosemite[1], specifically off the flat face of half-dome.

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Introduction:

Death is not a static concept. As our understanding of cognition and the human body has grown more intricate, so too has our conception of what it means to die [1]. Although we have shifted from cardio-respiratory markers to neural criteria, replacing cardio-respiratory death for brain death [2], lingering concerns remain about the precision and reliability of these frameworks. Recent neuroscientific advances, and clinical reports of patients in likely permanent unconscious states who nonetheless exhibit some neuro-electrical activity [3], have called into question the precision and reliability of contemporary brain-death frameworks [4].

These disjoints, between current definitions of death and our current understanding of neural systems and clinical cases, are adequate cause for the re-evaluation of what it means to be "dead" in a clinically, morally, and ontologically significant sense. If certain neural processes persist, ought we regard an individual as living in a morally significant sense, or does a lack of consciousness, of having a "what it is like to be", present as a necessary and sufficient criterion for a person to be no more? The philosophical literature on these questions has challenged the idea that biological viability of the body or brain equates to continued existence of the self, often pointing to concepts such as psychological continuity [5] or narrative attributability [6]. More recently, sentientist views have proposed that the "what it is like"-ness of any system is that which provides it moral worth, and as such, any thing with a "what it is like", a phenomenology or rather experience, is worthy and deserving of moral consideration[2]. Consciousness becomes critically important under this conceptualization, in no small part because consciousness is taken to be a definitive marker of meriting moral consideration under sentientism, and because conditions often conflated with brain death like coma or permanent vegetative state are defined by a lack of consciousness, and are resolved by the return thereof [7].

This paper aims to expand upon and enrich the discourse on death by proposing a dual conception of death, where bodily death and the death of consciousness are considered to be the cessation of two distinct but tightly interconnected processes. One of the conceptualizations we will seek to delineate through the use

[1] Yosemite is wonderful in the spring.
[2] For brevity and coherence, we will primarily utilize the sentientist view.

of this framework is that of brain death itself, and its moral significance. This paper contends that it is the existence and continuation of a consciousness that is of moral and ethical concern, and seeks to substantiate this claim philosophically, and subsequently substantiate those philosophical claims empirically. This is a utile endeavor in that it resolves some pending issues in the clarity and conceptualization of brain death. At present we struggle with "edge cases", such as those where the self seems to vanish in ways that are not clearly defined within the whole-brain or brain death standards, and as such we require some principled framework by which to understand these cases.

It is argued that our conceptual difficulties stem from the conflation between the integrity and continuity of the organ (the brain), and the continual presence of an experiential process, of a consciousness, and that a disabusal[3] of this conceptualization enables a more precise and well-structured definition of death. We substantiate this argument through the use of thought experiments, and by examination of various cases, including those of coma, sleep, and anesthesia. By taking this distinction between bodily processes and conscious processes, we can gain a clearer understanding of what is lost when a person dies in a morally and existentially significant sense, and begin to tie that directly to clinical and functional measures.

In what follows, we will first revisit conventional accounts of brain death and some of their limitations in explaining the continuation or lack thereof of life in some well examined cases, and discuss some of the neurological considerations there. We will then outline a process-ontological perspective, again utilizing contemporary neuroscience as well as arguments from philosophy of science to substantiate and illustrate our perspective. From there, we substantiate the idea that consciousness is the thing that matters in determining death, and further propose a revised conceptualization of death that incorporates both the cessation of essential bodily processes and the termination of consciousness as a dynamic system. This dual model, we propose, better accommodates edge cases, permits for a more nuanced empirically comprehensible definition of death, and provides further scaffolding for clinical measures of consciousness. A key clinical measure we take to indicate non-consciousness is the lack of TMS induced perturbational responsiveness of the brain, which would be reflective of the lack of an integrated neural dynamic and of consciousness. Finally, we consider the implications of this viewpoint for the examination of consciousness in medical practice and ethical decision-making, in particular, we emphasize the necessity for more robust and empirically sound neurological measures, and an increase in the availability of equipment and personnel necessary to implement those measures, to best guide clinical interventions and end of life decisions. Furthermore, we emphasize the necessity and permissibility for interventions in cases where a person is not at the moment able to instantiate a conscious dynamic, but could, through the right treatment or through time, come to be able to instantiate a consciousness.

Existing Definitions and Shortcomings

Early medical conceptions of death were guided by the loss of heartbeat and spontaneous respiration. Once the heart and lungs ceased to function, it was assumed that a person was dead. In the mid-twentieth century, the advent of cardiopulmonary resuscitation and mechanical ventilation introduced the possibility of extending or restoring basic cardiopulmonary function beyond what was previously naturally feasible. This challenged traditional markers of life and prompted clinicians and ethicists to look elsewhere for clearer criteria. By 1968, a landmark report from the Harvard Medical School Ad Hoc Committee introduced the idea of "irreversible coma" as a new clinical measure of death [8]. Subsequent guidelines, including the Uniform Determination of Death Act (UDDA) in the United States, formalized the notion that the irreversible cessation of all functions of the entire brain constitutes death [9]. These standards remain widely

[3] To disabuse is to free from misconception.

influential, but have been superseded by the 2023 Guidelines for the Determination of Brain Death/Death by Neurologic Criteria, attributable to the American Academy of Neurology, Society of Critical Care Medicine, American Academy of Pediatrics, and Child Neurology Society (2023 DNC Guidelines).

The shift from heart-lung criteria to the concept of brain death was a practical advance towards a more clinically stringent and ethically utile criteria of death. Under brain-death-based criteria, a patient without certain brain-stem reflexes, or who is unable to show organized neurological activity can be declared dead by neurologic criteria. In many respects, this standard has worked quite well, since the permanent loss of integral brain functions will coincide with the permanent end of any meaningful conscious awareness. Yet in some cases, residual neural activity or other signs of life persist, making it unclear whether the person has truly ceased to exist in the sense that most of us care about. The dissonance between complete "brain failure" and subtle indications of ongoing physiological or neurological processes have prompted renewed debate about the sensitivity of these criteria to potential nuances [10]. These are especially apparent and salient in the face of outlier cases.

While the present brain-death standards have served us well for decades, especially in the clinical context, a number of controversial or borderline clinical cases suggest that the existing criteria are at the very least incomplete. In the next section, we briefly go over two such edge cases and demonstrate how those make evident the necessity for a more nuanced conception of death.

The Problem of Edge Cases

The most famous case of contested consciousness is that of Jahi McMath, a 13-year-old girl from California who was declared brain dead after a somewhat routine oral operation led to massive blood loss, and subsequent (allegedly) inadequate treatment of said bleeding led to sizeable brain damage [11]. While we do not take a position on the Jahi case within *this* section, the physiological information available in the literature provides some evidence that the brain death criteria utilized in Jahi's case were less than adequate, and that her treatment may have been handled differently with a more thorough examination early on [12]. We will return to this Jahi in the case study section. But it should be noted that the McMath case was not a flash in the pan debate primarily between a family and the doctors, attorneys, and representatives of UCSF Oakland Children's Hospital. There were real scientific debates and conflicts of evidence there, which had moral and clinical implications of utmost importance for both Jahi herself and for the McMath family.

Some more recent and perhaps less controversial cases that demonstrate the issues with current measures are those cases where a person is declared brain dead, and where the person nonetheless manages to regain consciousness, indicating that the robust integrity of brain death criteria [13] may not be so robust. The less controversial but no less striking case of a misdiagnosis of consciousness is that of Anthony Thomas "TJ" Hoover II of Kentucky, who was declared brain dead after an overdose and whose registration as an organ donor meant he was slated to donate his organs upon his death. As such, after a series of steps, he was rolled into the operating room, where he began thrashing about and, according to those present in the operating room, crying. He was subsequently sedated. Staff were allegedly pressured to proceed with the organ donation procedure, and they did not. Hoover did not remain brain dead, and whether it was the case that TJ was misdiagnosed, or was adequately diagnosed and still made a recovery, is of substantial concern. TJ, as of writing, has not died, and has made a partial recovery from his overdose relearning to walk and managing to verbally communicate.

These borderline outcomes highlight a deeper conceptual gap between two preexisting standards of death, those of the Uniform Determination of Death Act, and those of the Harvard Ad Hoc Committee, and the current standards, the 2023 DNC Guidelines. The "whole-brain" criterion focuses on whether the organ

(the brain) is *irreversibly* nonfunctional, the criteria used in practice, the Harvard criteria, tends to prioritize the *immediate* existence of a functional or functioning neural system, and its successor, the 2023 DNC Guidelines, have come to value reflexive capacity. Yet what most patients, families, and clinicians truly worry about is whether there is any chance of regaining consciousness or more broadly, whether "someone is still in there", not if their loved one is *at present* incapable of regaining consciousness. That someone may have recovered if they had received treatment is not just a concern for patients, but it is a concern for people on organ waiting lists, since episodes like these have had real effects on donations.

The disparity between whole-brain criterion and the Ad Hoc Harvard and 2023 Guidelines is somewhat severe. The current tests for brain death often involve reflex checks, apnea trials, and imaging to verify that metabolic or electrical activity is essentially absent. In practice, it appears that the brain stem reflex checks and apnea examinations are the most popular, with one prominent physician ethicist seemingly asserting that brainstem reflexes and/or apnea tests ought to be the final decisive factor in determining brain death [13]. As we will demonstrate later on, these criteria are inadequate, especially for donors. In McMath's case, the first brain death examination demonstrated an absence of all of these. And it ought to be noted that, in most cases, these tests are reliable. However, the rare but significant misdiagnoses point toward the necessity for more precise measures aimed at identifying either the existence of ongoing integrative processes in the brain, or the capacity to instantiate these, rather than simply verifying that the organ is no longer working as a whole at this moment.

This is not an impossible request by any means. Indeed, some newer neuroscientific tools, such as transcranial magnetic stimulation combined with high-density electroencephalography can detect patterns of integrated neural dynamics that may remain in spite of severe injury. Familiar tools like fMRI scans are also a gold standard in evaluating recovery capacity. In cases such as TJ's, a patient might show minimal but definite responsiveness to TMS pulses, demonstrating that parts of the cortex and larger neural integrative circuits are still capable of organized communication. While not every patient in such a borderline state will recover, the possibility of residual or re-emergent consciousness complicates our sense of whether we have truly reached a "point of no return", and even by brain death criteria renders said person ineligible for a determination of death. That complication in turn raises fundamental questions about what it is, exactly, that we seek to preserve when we consider keeping a patient on life support, along with everything else that comes along with it.

As mentioned prior, the shift from cardio-respiratory criteria to the "irreversible cessation of brain function" standard was a logical evolution in medical science. Still, real-world edge cases have revealed cracks in the assumption that the entire brain must either be entirely dead or otherwise be alive. These cases suggest that we might need to go beyond the current criteria and begin to examine other factors, and reconsider the concept of brain death. We propose that in deciding whether to declare death in the clinic, that we consider principally whether the process of consciousness exists, even if minimally, and alternatively if it could be restored granted certain interventions. This is not a rejection of the brain death criteria in and of itself, but is a refinement on it. Where the current criteria center the immediate presence of certain reflexes, we center retained capacity and the *possibility* of recovering capacity given certain interventions. This would of course require fMRI or structural measures, and would require a uniform standard for non-recoverability.

Such a framework enables the differentiation, in principle, of brain structures which are more or less necessary for conscious existence[4], which will make some areas especially relevant, and others less so. Such a

[4] For instance, it is likely not controversial that you can lose your visual cortex and remain alive and a conscious human being. Indeed, this has happened. What we propose in refining the criteria for death is that we enable ourselves to consider both that consciousness can exist without certain functions, for instance some brain stem functions or visual cortical functions, and that consciousness cannot exist without certain functions. In determining brain death, it is then of principal importance not whether there is or is not any neural activity, but whether or not the correct type of neural activity is possible.

view is supported by a growing consensus in neuroscience that consciousness depends on specific dynamic interactions within the brain, and that the occurrence of gross structural failures may not always reflect the moment in which a person ceases to be.

This is in many ways a continuation of the sentiments and work of the Defining Death report, which itself stated that, "Now… certain organic processes in these bodies can be maintained through artificial means, and though they will never recover the capacity for spontaneous breathing or sustained integration of bodily functions, for consciousness, or for other human experiences." And we similarly agree, "That recent developments in medical treatment necessitate a restatement of the standards traditionally recognized for determining that death has occurred." [14]

In the next section, I will introduce a process ontological framework to better engage with our current understanding of the brain, and will redefine death, especially brain death, through the lens of ongoing or extinguished processes. This approach can resolve some of the conceptual and clinical tensions which afflict determinations of death. By treating consciousness as an active process rather than a fixed property of an organ, we can explain why borderline cases confound existing theories, set clear empirical targets that experiments can succeed or fail to meet, and develop more nuanced, clinically testable models grounded in contemporary neuroscience.

Process Ontology and Its Relevance to Death and Consciousness

To refine our notions as they relate to the brain and death, and to explain why certain borderline cases pose deep conceptual challenges, it is helpful to turn to process ontology. Although process ontology as relevant to our concerns is substantively explores in the work of Alfred North Whitehead and other philosophers of science, we will not dwell extensively on historical or doctrinal details here. Instead, our goal is to offer a functional account of why viewing life and consciousness as processes rather than as properties of objects, substances, or states can clarify the thresholds for life and death, and further helps us apply definitive (if somewhat complex) criteria to discerning the potential and existence of life and consciousness in biological systems.

The Core Idea of Process Ontology

At its most basic, process ontology, or systems ontology holds that reality is best understood in terms of active, ongoing processes rather than discrete objects carrying or exhibiting specific properties. Within the context of biology, this perspective is often seen as more intuitively aligned with how living systems behave. A living organism is not quite a collection of parts, it is an ensemble of dynamic, interlocking processes, metabolic regulation, cellular turnover, and what have you. Each of these processes depends on a set of underlying structures, but cannot be wholly reduced to them. A cell requires a membrane, organelles, and genetic material to perform its functions, yet the cell's life is a matter of continuous transformations and exchanges with its environment. Once these transformations cease entirely, the cell is no longer alive, even if the structures of the membrane, organelles, and genetic material remain. As such, it is quite a bit more elegant to see a biological thing as constituting a process of some kind[5]; as opposed to the thing being in a certain state or having a property of being alive. Moreover, describing it as a process allows one to more definitively evaluate whether or not a thing is alive. To do so, one simply evaluates whether or not the processes in question are occurring or not. If they are, then the thing is alive. If not, then the thing is not alive, or in other words is dead.

[5] Being a process, or instantiating a process, or taking a part of a process, the verbiage is variable.

Extending this process-based framework to life, especially life as it relates to the mind follows naturally. The mind is a mind insofar as it goes about doing mind-y things, and the brain *has* a mind insofar as it creates through its operation the mind-y things that make a mind. Broadly speaking, this logic can also apply to our existing or being alive as experiencing beings, which is our goal here. More specifically, the phenomenon we typically don't want to end ending when talking about brain death, consciousness[6], appears to hinge on a specific kind of global, integrative dynamic or process within the brain [7]. If that dynamic ends, one might say the process of being a conscious being disappears, even if every neuron and iota of neural tissue remains viable; even if there is some brain activity, it matters that it is the *right kind* of brain activity. The organ that gives rise to our experience, the brain, is then a necessary *but not sufficient* condition to determine the presence of a conscious process. The question in our edge cases is whether enough of the integrative neural process has halted *irreversibly*[8]. If so, then the subject is effectively gone in the sense that we value morally and existentially. There will never again be a consciousness that originates from that body or brain, and there will never again be an interaction with what that person was understood to be.

Contrasting Objects & Processes, Ascertaining Permanence

The more standard object-property view might look at the brain as an organ, note its structural integrity, and then conclude that if the organ fails to exhibit some property[9] or perhaps to function in certain ways, the person is gone. In most clinical scenarios, this aligns well with reality, which is why whole-brain death standards have proven workable in a vast majority of cases. However, edge cases reveal that organ-level assessments can sometimes miss sub-detection threshold signals for processes that may still be meaningful. The critical question is not just whether the organ is in disrepair, but whether the integrative process of experience is *wholly and irrevocably* extinguished. A process ontology framework thus invites us to look for active or potentially *reactivatable* neural dynamics and structures, rather than confirm that the organ, in broad terms, has ceased to function as a unified entity. This is not a novel view [15], but it is an important, if overlooked one.

Whitehead's broader philosophy of organism similarly proposed that living beings are best conceptualized as flows of activity, an equivalent, if more poetic, description of a process. The same is generally true[10] for consciousness as relates to being a conscious being; we can either focus on structural things, (like specific brain regions) or we can track the dynamic, that is, the changing patterns of neural information flow that themselves constitute conscious awareness [11]. In general, I propose, it is this dynamic that we care about. That is partly why families in borderline coma cases ask, "Is there still any chance she is in there?" rather than whether the physical brain as an object remains intact, or if it exhibits *at present* the properties of being conscious.

This is all quite abstract when applied to consciousness and the brain, so it may be useful to think of it via an analogue. A good metaphor for systems as opposed to objects as understood in process ontology is that of a flame. A flame depends on a fuel source (such as wax, wood, or gas) and on an available oxidizer (oxygen in the air). It also depends on a spark that begins, and heat that sustains, a chain reaction of oxidation. Although the flame is dependent on these underlying conditions and substances, it is not *identical* to

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[6] Consciousness here being quite simply a "what it is like"-ness.
[7] One which we will examine more in depth in the section on scientific theories of consciousness
[8] The reversibility pre-requisite will become important later, as we will demonstrate that consciousness tends to end relatively frequently.
[9] It is important to note that the ontology of things being primarily objects and properties is not opposed to the concepts we outline here. One could, for instance translate the idea of a property to be related to whether or not we evaluate a given thing to fall under a given category. The property of a thing can be understood as our having an evaluation of the thing that it does the same things or leads to the same interactions or results that we typically ascribe to other things which exhibit that thing-iness, lets say "wetness". That being said, process ontology allows us to bypass the more verbose conceptualizations necessary to coherently talk about things in this particular context, and additionally does not encumber by needless complications the understanding we are seeking to build.
[10] Arguably. Relighting the candle will produce a flame that appears similar in shape and brightness to the previous one, however, it is not the same flame. The process that existed in a prior instance is distinct of that one begun anew in the subsequent instance. This becomes important later.
[11] Unfortunately, you do have to do both, but the point stands. Assuming there isn't more neuroscience to do , then the above holds.

them. Extinguishing a flame does not require removing the wood or oxygen or even heat from existence (although doing so will similarly end the process) what it requires is terminating the *process* by which the oxygen and heat and fuel react, the pattern. As such, when you snuff out a candle, you have ended or "killed" that flame[12].

A more apt comparison, particularly for those who prefer a higher-energy analogy, is that of a fusion reactor. Consider a hypothetical[13] energetically self-perpetuating controlled fusion reaction, occurring in a specialized reactor chamber. The physical apparatus includes electromagnets, complex coolant systems, and so on, yet the key phenomenon of fusion is *not* those specific things. It is a dynamic process, atomic nuclei colliding and fusing under extreme conditions in a positive self-reinforcing process and feedback loop[14]. One can shut down this reaction by adjusting the reactor's confinement field or reducing plasma temperature, effectively stopping the fusion process while leaving the reactor structure the same. If, at some later time, one reintroduces the conditions for fusion and ignition, a ***new*** reaction begins. The machinery is the same, but a ***new*** process has been created, it is *similar* to that of the prior session because the same underlying system gave rise to the new process, but it is ***different nonetheless***.[15]

This distinction is critical in that it permits us to parse apart 1. The possibility of a process being instantiated. 2. The process itself existing, and 3. The medium of the process existing. These are interrelated but separate questions. Whether the process of fusion is instantiable is dependent on there being certain underlying structures present, namely a reaction chamber, containment field generator, and the like. Whether the process exists is dependent on those things operating in a certain manner and the dynamic of atomic fusion actually occurring. This is verified by the appearance of a toroidal plasma ring, and of an energetic output. Whether the medium of the thing exists is verified simply by ascertaining that the containment chamber and other such accoutrements exist.

From a neuroscientific perspective, utilizing process ontology means looking at how the brain's electrochemical signals flow, integrate, and reconfigure over time and across neural ensembles. Rather than labeling the brain as having the property of being "alive" or "dead," we ask whether the global neural dynamic that underlies subjective awareness is operating, or is able to be instantiated. If the structure generating the dynamic has collapsed beyond any feasible self-repair or medical repair, then from a process-ontological standpoint, the conscious system, what a person would identify as the self, is irreparably lost. Death has come to it. There is a permanent death there, even if some brain cells fire occasionally. Conversely, if the dynamic remains there, it is overwhelmingly likely that there is still a "somebody" in there, granted it is the right kind of dynamic. Final death, the death we are concerned about in these cases, comes about when the system instantiating consciousness is incapable of doing so any longer, in other words, it occurs when the brain or instantiating system ends its last run of consciousness.

This shift in emphasis, while perhaps seemingly minor, may help us explain why borderline cases are genuinely controversial. Standard clinical tests for brain death often look for reflexive brain stem function, spontaneous respiration drive, or EEG activity. Yet these might neglect cases where consciousness is not presently there, yet could emerge given the right conditions. Similarly, these criteria may neglect more subtle integrative phenomena detectable through advanced measures like transcranial magnetic stimulation (TMS) employed alongside high-density electroencephalography (EEG) measures. In principle, such measures can probe functional connectivity, the actual interplay of signals across cortical and subcortical regions which give

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[12] That *specific* flame.

[13] There have been self-sustaining fusion reactions, but they are not stable. Here we imagine a stable one.

[14] The fusion reactions occurring are a result of the plasma conditions (temperature and density) in the immediately prior instant. The energy released by these reactions then maintains those conditions for the next instant.

[15] This is analogous to consciousness, to awareness. The underlying neural "machinery" may remain, but if the global integrative dynamic ceases, that particular run of consciousness is over, never to exist again. If the brain reboots and reestablishes integrative connectivity, a new iteration of consciousness arises, one that reflects changes in the instantiating stratum made by the previous iteration, but is not identical to it.

rise to consciousness. A purely structural lens (object-property ontology) might evaluate whether or not a neural system has the property of being conscious, while a process-based lens (process ontology) might specifically check whether global processing integration is permanently halted or if some emergent dynamic could still reorganize and bring about consciousness under the right conditions.

This process-based lens allows us to further account for various stages of consciousness and lack thereof. A systems outlook can account for temporary cessations of consciousness, such as deep sleep or anesthesia. In those cases, the physical substrate (the brain) remains sufficiently intact to re-initiate the global integrative loop. The same substrate, if properly maintained, can spawn multiple instances (or "runs") of consciousness over time, just like a fusion reactor can be cycled on and off, and therefore is not necessarily "dead" as a structure, even if the fusion reaction within it does fit the criteria of being "dead" at any given moment. By viewing consciousness as a dynamic, we further recognize that once it ends, that particular run is finished, but a new one can be ignited upon waking or resuming normal neural integration.

Some might worry that "process" is a vague term, or that process ontology veers into metaphysical territory. However, process is already a central concept in contemporary systems biology [16], theoretical neuroscience [17], and dynamical systems theory [18] [19]. When we say a neural ensemble is engaged in a "pattern of activity," we are treating it as a process, an event or sequence that unfolds across time. This framing meshes well with some topics which we will explore in our section on clinical measures, namely experimental work on empirical measures of consciousness in locked-in coma [20], current understanding of neural computation [21], and complexity indices in neuroscience such as the perturbational complexity index, or PCI [22]. The more that a theoretical framework aligns with empirical approaches in neuroscience, the stronger its footing is in guiding clinical decisions[16].

In philosophy of biology, a similar shift has occurred away from viewing organisms as collections of matter with properties and toward viewing them as self-maintaining, self-organizing systems [23]. These living processes exhibit functional unity that comes and goes depending on conditions like nutrient availability, internal feedback loops, and environmental factors. Death, in that context, is the irreversible breakdown of these coordinated processes, leaving behind a non-dynamic set of tissues or remains [24]. Extending that same logic to consciousness suggests that if the core neural dynamics disintegrate past a point of no return, we say the conscious being has died, even if the body is partially kept alive. Ultimately, a process perspective aligns with the reality that "life" and "mind" are not just properties glommed onto otherwise inert substrates, but rather are emergent, perpetually unfolding systems dependent on their substrates.

By adopting a process ontology, we lay the groundwork for more precise definitions of death, especially brain death. We do not need to solely rely on what is in essence a measure of organ failure, which can be quite partial and unclear as we will see in our case examinations, and may instead examine the termination of the relevant process, and the integrity and existence of the underlying infrastructure giving rise to said process. This leads naturally to the question of which neural process we ought identify with consciousness, and how we may measure its ongoing presence or absence. In the next sections, we will draw on contemporary empirically founded theories of consciousness, most notably Cortical Self-Referential (CSR) theory, to illustrate how a globally integrated neural dynamic arises, why it matters morally and clinically, and how it may cease under various conditions such as coma, anesthesia, or catastrophic brain injury.

[16] It is important to note that indices like TMS-EEG perturbational complexity indices are a measure of consciousness existing, and as such if one tests for it, we absolutely cannot pull the plug, and additional considerations must be had. Whether or not to discontinue care for someone who is not currently conscious will require that we evaluate the integrity of certain circuits and their capacity to be rebuilt or repaired enough, either though medicine, to again instantiate a consciousness. That second evaluation is much more sensitive to developments in medicine, and is much more of an empirical question than a philosophical one.

Relevance and Utility of Neuro-Physiological Accounts of Consciousness.

The question of when someone is dead and concurrently of what it means to be alive, is at its most consequential in the clinical context. As such we want to address these more abstract philosophical concerns with as much empirical structure and validity as possible. For that reason, we utilize neurophysiologically amenable accounts of consciousness, which, while quite divergent in some aspects, mostly cohere in their requirements for the existence of consciousness, and determinations of lack thereof. Neuro-physiological accounts of consciousness are naturally important in brain death cases and are similarly important in determinations of death overall, especially because they provide a theoretical scaffold by which we can determine death along physiological lines.

The key measure of consciousness we will discuss throughout was originally proposed by way of a theory of consciousness called Integrated Information Theory, or IIT [25]. The perturbational complexity index (PCI) is a measure of the complexity of the brain's response to transcranial magnetic stimulation (TMS), and doubles as a measure of consciousness [26]. A more simple measure is that of Mean State Shift or State Variance in response to a TMS pulse, as measured by various characterizations of TMS-evoked electroencephalographic potentials [27] [28]. While PCI and related measures were principally developed by IIT proponents, the theories of consciousness we have previously mentioned have explanations and mechanisms through which these same mechanisms can be understood and justified [29]. There is, in short, a tentative scientific consensus on what measures indicate one to be brain dead or unconscious, even if the explanations for what consciousness itself actually *is* differ substantially [30].

CSR, GWT, IIT, and HOT all determine some kind of global network connectivity and information flow to be necessary to consciousness and the determination of the presence thereof. CSR delineates a cortical and deep brain circuit which instantiates a computational dynamic isomorphic with consciousness [31]. It further determines all systems which are affected by and affect that overall dynamic to be a part of consciousness, and as such any perturbation of one sub-system dynamic will necessarily affect the larger dynamic, and this perturbation will reverberate in the dynamics over time. GWT, through the global workspace paradigm, states that all things which are conscious are a part of the global workspace, centering global integration and global broadcasting [32], which is parallel to CSR's requirement of a globally integrated computational dynamic. IIT defines consciousness as the integration of information, hence the name, and as such the brain's ability to integrate information can be utilized as a proper measure of consciousness directly, through measurement of what IIT calls "Phi", which is the measure of integrated information and therefore the measure of consciousness of a given system [33]. HOT requires that cortex come to a determination of there being a self and further go about essentially evaluating the self [34]. As such, HOT requires at the minimum that cortical activity be present in certain areas required for self-reference, namely the frontal and parietal cortices.

It is important to note the consensus of these theories in the requirement for what is essentially that there be a global integration of signals or some set of dynamics, for the existence of consciousness[17]. This consensus is key to this paper as it means that, while one can disagree on what consciousness itself is, we have surprisingly little disagreement on what factors indicate consciousness, and can therefore speak broadly about consciousness and unconsciousness.

A Cortical Self-Referential Theory of Consciousness

[17] Although HOT places more emphasis on the system's higher-order representation of itself, and IIT focuses on the quantitative measure of integrated information (Φ), both still require that separate brain areas form a unified, globally integrated whole in order for a conscious dynamic to exist.

While our discussion is critically applicable across all of the aforementioned theories of consciousness, here we utilize primarily the Cortical Self-Referential theory of consciousness, (CSR). It is therefore useful to outline CSR and why it is especially utile in this context. CSR is a theory of consciousness which proposes that consciousness is best understood as a specific dynamic process arising from the operation of a central cortico-basal ganglia-thalamo-cortical circuit [18]. The set of computations instantiated by this dynamic create what is in essence a simulation of the exterior world and of a self[19] within that simulated world. The process of the entire simulation is then proposed to be identical to consciousness. Under CSR, consciousness is fundamentally you, and you are fundamentally your experience (consciousness). This is best illustrated through example. Let's imagine that your nerves are suddenly exposed to open air, which would lead to indescribable pain and an inability to think about or perceive very much of anything else. At that moment, according to CSR*, all you are* ***is*** *that pain.* Your perception or experience of that pain, and nothing else, is you at that moment. When you read this paper, what you *are* is the experience of reading this paper, of feeling the paper or computer in your hands, of an inner monologue describing the paper, of the percept of the paper itself. That you are identical to your experience is an important assertion in our capacity to ascertain the existence of a consciousness in a given system, as it allows us to determine consciousness in terms of dynamics and neural structures which are understood and can be definitively ascertained to be present or not present *in principle*[20]. This distinction further permits the nuanced understanding of death that we are trying to build, and in particular permits a precise understanding of the nature of brain death and further permits the construction of a more clinically precise, definite, and utile definition of death.

The general structure of the central circuit is important to note as it will form a central part of the overarching analysis going forward, as well as the case examinations, including those of McMath and Hoover. The central circuit for consciousness according to CSR is composed of the Anterior Temporal Lobe, Prefrontal Cortex, Basal Ganglia, and Thalamus:

Anterior Temporal Lobe (ATL) & Prefrontal Cortex (PFC)

Under this view, the ATL holds semantic representations and relational structure between said representations [35] [36], and communicates the activation of those representations to the hippocampus and the PFC [37]. It maintains sense-invariant representations of objects, concepts, and relationships, linking various sensory features to an abstract, *modality-independent* identity, permitting abstract computational comparisons. In other words, the ATL distills the varied sights, sounds, and other features of an entity into a stable concept, for example, your ATL helps encode the idea of an "orange" (its shape, smell, taste, name) in a unified representation [38]. Lesion studies demonstrate that the ATL is critical for semantic memory, our knowledge of objects, people, words, and facts [39]

In the CSR circuit, the ATL provides the general abstract content of conscious experience, the "what" that is being represented in mind[21]. It is the repository of the current abstract percepts and thoughts,

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[18] CSR uses this particular circuit since it is the circuit which in healthy neurotypical humans instantiate the variety of complex tasks related to consciousness. This circuit is important for everything from visual processing to abstract cognition to language, in that those systems and functions have the circuit as a necessary pre-requisite in order to function. That being said, like most systems in the human brain, this circuit can be redirected through neuro-plastic adaptation, and it is very likely that there exist redundant deep brain systems which could take over in the absence of the central CRS circuit, instantiating a "base-level" consciousness. The computational and functional role of such a potential circuit is conjectured to instantiate a minimal version of the same general CSR circuit and dynamic, and likely further requires some (but not all) of the same neural structures. A potential example of a parallel multiply-redundant structure is the periaqueductal gray in decorticate infants, which we conjecture fulfills both the roles of the PFC and ATL, albeit at greatly diminished capacity [163]. This is important to note as the CSR framework is substrate neutral and quite comfortably non-dogmatic on the particular anatomical structures instantiating the dynamic of interest.

[19] As well as all of those cognitive percepts attributable to the self, smells, etc. Basically, everything that a person is aware of at a given moment is a part of consciousness. I feel that is relatively uncontroversial, if one disregards one's preconceptions and prior commitments.

[20] The reason I say in principle is that these systems are currently not entirely understood. The idea is that, if you understand the entire brain as well as we understand, say the visual cortex (which is quite well understood), then you are able to track dynamics through that neural architecture, and that the dynamics through the architecture are what matter in determining whether or not the particular process you care about is actually there.

[21] It is important to note that other parts can take part in sense invariant relational representation, and that the cortex is plastic enough to instantiate basically the same functions in areas other than the anterior temporal cortex. This is what happens in cases of recovery post-lesion.

encoded as richly interconnected semantic representations, with relational information being instantiated by the overlap between variably separable neural ensembles [40]. The ATL communicates the activation of these representations to other nodes. Specific to our purposes, it sends the active conceptual patterns to the hippocampus and prefrontal cortex. For example, perceiving or recalling a dog activates an ATL ensemble for "dog," which then broadcasts this semantic content to the PFC. Top-down signals from PFC (and modulatory input from the thalamus and hippocampus) can bias ATL activity, strengthening certain features or associations while damping others. Through this bidirectional exchange, the ATL's conceptual content is continuously fine-tuned through interactions with other ensembles. Phenomenologically, the ATL's contribution is the general abstract content of consciousness which when elicited, creates the recognizable people, objects, words, and relationships that populate our inner experience[22]. CSR views these ATL-held representations as the relational-computational building blocks of "what it's like" to experience something, what is termed in CSR as the "semantic subjectivity" of the cognitive-computational system, referring to the idiosyncratic relations and interpretations of perceived content.

The prefrontal cortex (PFC) acts as the brain's affective evaluator and is guidance mechanism for dynamics within the CSR loop. The PFC integrates the information coming from the ATL with context about goals, expectations, preferences, and task-relevance. It performs what is in essence a higher-order appraisal, essentially asking of the current pattern of representations, "What does this mean for me? Is this good, bad, or neutral?". Neuronal activity in medial and orbital regions of PFC evaluates[23] the value and emotional significance of stimuli [41], while lateral PFC regions incorporate rule-based and goal-oriented processing. The PFC is richly interconnected with sensory, limbic, and memory systems, permitting the PFC to compute a composite evaluation of the current state of the corresponding system, drawing on both the factual content (from ATL) and afferent evaluations from limbic systems [42] [43] [44].

In sum, **t**he PFC functionally binds representations and evaluates the pattern of representations in the ATL through further processing of the same computational kind as the ATL. The PFC then modulates the representations in the ATL and additionally communicates the determination of the overall valence of the active pattern of representations in the ATL to the BG.

Basal Ganglia (BG)

The BG, treated as a whole, integrates information sent from the PFC along with inputs from the ATL and many other systems to create a measure of how the system currently is, in other words, the valence of the system, and the trajectory of the valence, both projected and actual, to the end of responding to the current state of the system. The BG communicates that measure to the thalamus and hippocampus, which subsequently communicate and modulate systems including the BG itself, the ATL, and the PFC, thereby completing the loop.

Striatum (Part of Basal Ganglia)

The Striatum is the primary sub-system of interest in the BG in CSR[24]. The striatum according to CSR serves partially to instantiate a population code for the amount of activity and change in the circuit at any given time. Conceptually, the striatum can be seen as constituting the evaluation of the incoming information from the PFC, and creating a linear evaluation of the overall incoming information. The linear

[22] The elicitation of these neuro-representational ensembles leads to activation of the relevant pattern for the particular representation corresponding to the ensemble in sensory areas such as the visual cortex. The pattern is "projected" (elicited in a top-down fashion) into the relevant area, in such a manner as to similarly elicit the activation of the relevant representational ensemble upstream. This reciprocal activation creates a semi-stable and self-correcting percept, with the entirety of the (in this case) stable dynamic constituting the entirety of the relevant percept as perceived.
[23] Based on prior experience and training.
[24] In the basal ganglia there are quite a few structures which can partially perform the role of the striatum, and in the service of brevity we are lumping in some of the afferent or downstream effects of the striatum within the basal ganglia with the striatum-exclusive functions.

measure is something that is akin to valence and salience[25], from which the trajectory of valence can be additionally determined. Valence here is basically a measure, instantiated via population code, of "is this good or bad? And by how much?"

The striatum, especially the ventral striatum, is known to be primarily involved in reward and aversion evaluation and communication [45]. Via extensive reciprocal interaction with the cortex, thalamus and other parts of the BG, the striatum instantiates and heavily influences sensory gating, selective attention, and temporal structure [46]. In the CSR framework, the striatal circuits provide a general affective "weighting" to concurrent, functionally bound, ensembles and dynamics. The striatum serves to guide action, and more importantly serves to change the existing dynamics through the overall neural system in order to concur with its evaluation of the overall condition of the system as evaluated by itself. In other words, in order to adapt and concur with the evaluated valence of the system. That the striatum affects the overall circuit dynamics itself is quite important in CSR for understanding the substantial variations in moment to moment salience and subjective rate of time evaluatiojns [47]. In general, the striatum's modification of existing dynamics is necessary for the creation of dynamic, non-stereotyped behavior. The idea being that a valence or basic modulatory system very akin to valence of the kind instantiated by the striatum is necessary for the dynamic running through the circuit to affect change upon itself and guide its own activity across time.

The self-modification of the dynamic is critical to the determination of consciousness in CSR, as a complete lack of change or self-modification by the overall dynamic across the circuit by definition constitutes the complete cessation of consciousness[26]. That consciousness is dependent on these structures is borne out through the existing literature. Recovery of consciousness from coma patients is substantially determined by the persistence or recovery of striatal function [48], upon ablation of the striatum, stereotyped repetitive behaviors emerge [49], as does the complete cessation of motivated behavior, including basic survival behavior [50].

Thalamus

The Thalamus modulates the cortex at large, including the ATL and PFC and further modulates a great number of other neural systems, changing the dynamics of the overall system to reflect the BG's evaluation of the current state of events, as well as the concurrent computations of a wide swath of neural structures [51] [52].

The thalamus is often described as the brain's central relay station. In CSR the thalamus plays an active role in regulating, directing, coordinating, and synchronizing global neural activity [53], and as such is a necessary prerequisite for the existence and determination of consciousness [54] [55]. In the general CSR circuit, the thalamus functions as the global context regulator and broadcaster of the valence evaluation computed by the basal ganglia. Once the BG have evaluated the current condition of the system, the thalamus propagates this evaluative result broadly to the cortex [56], ensuring that the entire network updates

[25] From here on out we will simply refer to valence-salience as valence. According to CSR, both salience and valence as different functional applications of the same striatal measure. Whereas salience is typically attributed to sub-systems related to reward, and valence is typically taken to be primarily limbic, CSR proposes that both valence and salience are the utilization of the same striatal measure, of valence-salience, by different neural system modalities, namely those of the limbic and reward centered sub-systems respectively.

[26] That the lack of self-correcting dynamics is conjectured to definitively lead to the complete cessation of consciousness is one of the edge-cases and falsifiability criteria for CSR. If, for instance, the ablation of self-modifying structures does not lead to a lack of consciousness equivalent to irreversible coma or untreatable stereotyped behavior, CSR would be effectively disproven. Clinically, the conjectured role of the striatum and the basal ganglia as a whole are crucial in CSR's capacity to explain and guide treatment of cases of unconsciousness or minimal consciousness, presenting alongside high neural activity and significant integrative dynamics. Such cases are rare [159] [160], but constitute the absolute edge of consciousness in humans as understood through CSR. Such cases additionally motivate this examination of death, as akinetic mutism of the kind accompanying complete or near complete negation of striatal processing are difficult to conceptualize under the current determination criteria, and are further demonstrations of non-consciousness demonstrating the need for a "permanent irreversibility" criterion for determining death of consciousness of the morally relevant kind. Because consciousness is not an ever-present dynamic, its ability to be instantiated or not by a given neural infra-system is what determines if the relevant system is properly understood to have undergone a "total" death as constructed in this paper.

according to the new information in a context specific manner[27]. Anatomically, certain thalamic nuclei (like the intralaminar and mediodorsal nuclei) have diffuse projections to widespread cortical areas enabling them to enact modulation across the system [52]. Under CSR, the thalamus receives the "verdict" from the basal ganglia, a signal that "this is important" or "this is not rewarding", and then broadcasts that signal to influence processing in sensory, associative, and motor regions accordingly [57], taking in information from those modalities and using that information in concert with the basal ganglia's evaluative information to modulate the corresponding system accordingly [31]. Essentially, the thalamus helps align the subsequent time-step of the system with evaluative feedback, so that what the cortex represents and computes in the next moment reflects the outcome of the previous moment's appraisal.

Empirically, the thalamus is also known to help the neural system decide what in particular to focus on among vast sensory input. Here it is biasing the overall dynamic in accordance with the latest value assessment and context. Under typical conditions, the thalamus works in tight reciprocal loops with the cortex, where cortical feedback to thalamus helps refine what is broadcast [58], creating a dynamic interplay that sustains and modulates percepts. For consciousness, the thalamus's contribution is to maintain a space for the coherent integration of global signals [58], a global workspace of sorts[28]. It ensures that when one part of the brain "decides something is important," the rest of the brain's processing reflects this or is able to integrate and interact with that decision in the next time-step. This underlies the unified, cohesive nature of conscious experience. We don't experience disjointed bits of processing. Thanks in part to thalamic coordination, we experience an integrated whole. If the ATL's role was content and the BG's role was value, the thalamus helps weave those together into a unified perspective, updating the entirety of consciousness continuously. Notably, without proper thalamic function, consciousness is greatly disrupted [59]. For instance, bilateral thalamic injuries can cause coma or vegetative state [59]. In CSR, the thalamus is the central structure for binding[29] the various cortical and subcortical processes into a single, conscious process.

Integration and Significance

Each of the above components performs a distinct computation, and consciousness in CSR arises from their integrated activity. The ATL provides the semantic[30] and relational content of "what is represented," the PFC attaches interpretive and goal-oriented meaning ("what it means for me"), the BG assigns an affective valence ("good or bad"), and the thalamus and hippocampus feed those results back to modulate and contextualize the subsequent timestep of consciousness. This looping architecture is self-

[27] The context specificity of the regulation by thalamus is due to the modality specific computations undertaken at dedicated modality nodes in thalamus.

[28] Notably, CSR is compatible with GWT, in that an equivalent neuro-computational structure exists within CSR. However, CSR identifies its "global workspace" as being a constitutive and necessary part of the overall process and dynamic of consciousness, and critically does not identify it's GWT equivalent structure as itself being consciousness.

[29] Binding of processes and neural ensembles in CSR is twofold. On the local neural level, neural ensembles are bound insofar as there is obligate reciprocal activation of those ensembles. On a global level, systems are bound or integrated into consciousness insofar as their processing is both affected by and affects the overall processing of the central conscious dynamic loop. The idea being that this network of interactions enables the all to all communication of the relevant modalities. This is a desirable thing since you want to be able to compute and integrate information across modalities, and you want to be able to do that because doing so enables the extrapolation of information that would be difficult or impossible to ascertain using a single modality or a set of modalities which are only moderately integrated. For instance, imagine that you are deathly allergic to grapefruit. The feeling of the fruit may be indistinguishable from an orange, yet the smell, when combined with the texture will enable one to identify and avoid the offending fruit. Similarly, associative processing, which is undisputably utile, is enabled by the vast integration of modalities into a single overarching system.

[30] Semantic content is here defined quite simply as the sensory or experiential content of a representation, combined with the representation's relational content. This may seem initially counterintuitive or tautological, however a thorough examination of informational content in CSR, especially as determined by the neural architecture of the relevant system, clearly demonstrates that relational content is fundamental to the kind of processing we typically describe as being semantic. According to CSR, a representation of something is a representation of that thing insofar as that thing elicits the activation of that same representation, and where that representation additionally enables the manipulation of that thing and the prediction of that things' behavior. My representation of coffee is a representation of coffee insofar as coffee elicits that specific representative ensemble, and the ensemble additionally elicits information that enables me to manipulate or predict the consequence of manipulation of the coffee.

referential and continuously updating. The brain is constantly evaluating its own representations and then modulating itself based on that evaluation. The result is a unified, ever-evolving stream of consciousness that has content (objects, thoughts, etc.), qualitative tone (emotion, importance), and perspective (a "for me"). By understanding the roles of each node, ATL (representation), PFC (evaluation), BG (valuation), and Thalamus (global broadcasting), CSR theory offers a mechanistic[31] account of how the brain might generate the subjective all-encompassing tapestry of experience from purely empirical processes. Each region's contribution is indispensable in this view, as together they form the "core circuit", the operation of which is identical under this view for consciousness. While this model continues to be refined, it is grounded in well understood neurobiological and neuroanatomical systems and provides a clear framework for our purposes: consciousness is a process of creating, evaluating, modulating, and representing a unified dynamic percept, which is identical to the totality of an individual's experience. What could otherwise be identified as exterior or interior states is instead understood as a unified internally created simulation of the surrounding environment, the self within that environment, and all of the cognitive processes typically attached to the self. Said more poetically, under CSR, you, as a consciousness, are simultaneously and inexorably the perceiver, all that you perceive, and the process of perceiving. By simplifying the vast complexity of brain activity into this circuit of "key players", CSR attempts to demystify subjective experience in terms of concrete interactions among well-known brain systems. More importantly, the theoretically delineated circuit maps closely to our current understanding of the necessary clinical structures for consciousness as is generally, if somewhat hazily, defined.

That CSR provides a conceptual model and more importantly an empirically accountable framework to work with moving forward. It additionally identifies very specific structures and dynamics for consciousness with a degree of detail that is both notable and generally utile. Each node in the proposed circuit corresponds to concrete, well-studied brain structures with known roles, as we have highlighted. It is no coincidence that anterior temporal lobes are indispensable for conceptual semantics [60], that prefrontal networks support and underlie directed purposeful planning [61] and affective evaluations [62], that basal ganglia (striatal) circuitry mediates reward, salience, and the enactment of context-appropriate actions [50], and that thalamo-cortical networks enable global integration and continual modulation of cognitive processing [54]. That damage to all but the most plastic of these structures[32] leads to comatose or coma-like states is similarly non-coincidental.

From here we will examine why these particular structures and why the conscious dynamic in particular is important in determining the death of a person, and further evaluate what consequences taking a process ontological approach has on our conceptions of death.

Why Consciousness and Not Something Else: An Examination of Personal Identity and The End Thereof

Personal identity, on the view presented here, is grounded exclusively in the continuity of one's conscious experience, in what we will call a single "run of consciousness", consciousness being the unified, globally integrated process of experiencing. We argue that consciousness is not a thing one *has* but rather an ongoing activity or process that one *is*, and that the self is strictly identical to this process. The argument is structured in formal steps, with premises and conclusions, meant to argue for the position that only an uninterrupted run of consciousness qualifies as having a persistence of personal identity. When the conscious

[31] Appendix includes a more specific neural circuit for mental imagery, which contains a more granular examination of these computations.
[32] The cortical areas are the most malleable, as the architecture of the cortex is fairly standardized across the entire cortex, and is generally quite adaptable. It is also important to again emphasize that there are other structures which seem to be similarly capable of instantiating a loop of the same general sort as the one outlined here due to their connectivity, computational complexity, and general functional roles [161] [162].

process ceases, even temporarily, as in dreamless sleep or anesthesia, that particular run, and thus that personal "self", ends. In short, continuity of consciousness is necessary and sufficient for the continuation of personal identity, and a break in consciousness marks the "death" of that identity in a meaningful metaphysical sense. As is evident, there are many premises which are themselves in need of validation, including empirical validation.

1. Consciousness as an Ongoing Process of Experience

a. **Consciousness is an active process:** Consciousness is fundamentally an ongoing process, an operating system of generation and modulation of experience. Quite critically it is not a static object or entity that could subsist on its own. It exists only as experience is happening, since it is itself experience.

b. **A process exists only while it operates:** By definition [63], a given process occurs only so long as it is actively unfolding. When the activity of a process ceases, the process itself no longer exists[33].

c. **Therefore, by [1a] and [1b], consciousness exists only while experience is ongoing, and it ends when that particular process ceases.** In other words, a given consciousness (stream of experience) is present only *so long as* the process of experiencing is running. When the experiencing stops, when consciousness is not operating, that *particular* consciousness is no more.

Explanation | Elaboration: This is meant to emphasize that consciousness has no independent existence apart from the activity of experience. There is no dormant "consciousness entity" sitting about when experience halts; the conscious process **is** the experiencing itself. Just as a dance exists only while dancing is occurring (and of course ceases when the dancing stops), a conscious experience exists only while it is an ongoing experience. When the process of experience ends, the consciousness in question vanishes with it, since it is, it.

2. The Self is Identical to the Consciousness Process

a. **Consciousness is the entirety of experience:** Central to CSR as well as the Predictive Processing [64], Self Model [65], and Attention-Schema [66] theories of consciousness, is the idea that reality is internally generated. That what we experience as daily reality is an internal reconstruction of the exterior world. One that is, by evolutionary necessity[34], quite close to external reality, but which is not equivalent to it. The arguments for this are varied and beyond the scope of the paper, but there is an intuitive argument for the internal generation of reality upon consideration of exceptionally vivid dreams, or strong mental imagery.

b. **The self is the subjective aspect of experience:** We ordinarily use the term "self" to refer to the personal subject of experience, the "I" or "Self" that perceives, thinks, and wills. It is the sense of being an observer or agent from a first-person perspective within one's conscious perception.

c. **All features of the self appear within consciousness.** Every attribute by which we identify the self is a segment of the overall dynamic of consciousness itself. One's thoughts and intentions, feelings of

[33] Once could imagine a scenario where a consciousness is "paused", say by the creation of some fictional time-stopping machine. The idea is that the dynamic be continuous with itself. So, if one was able to, for instance create a drug which would ensure that the activation or lack thereof of all given neurons were retained until instantiated anew with a separate "activating" drug, then that would indeed be a "paused" consciousness. A not entirely dissimilar scenario is entertained with the anesthesia-induced coma example a bit further down.

[34] Imagine that you are walking down the street, and you perceive that all is well, and then a car spontaneously appears and kills you. That would be bad, evolutionarily speaking, and whatever ended up making it so that your internal generation was broken in such a way as to kill you in this way would be evolutionarily selected against. You want to be able to know that the car or lion in front of you is real, or that the femur spinning in midair towards your head is indeed moving towards you and not towards some other entity.

ownership over one's body, memories, personality traits, and even the basic sense of being an "I" or observer, all of these are content or aspects of one's particular conscious experience, by virtue of being experienced. There is no observed feature of the self that lies outside one's conscious awareness; whenever we introspect to find "the self," we find thoughts, feelings, or perceptions, elements of the conscious process. If we take the position that the conscious "observer" can delineate what the self is, by identifying itself, then anything the observer points to, by virtue of having been observed, will be a part of consciousness, since the index will be to the experienced representation of the thing. Concurrently, if we say that the *observer* itself is the self, regardless of what it itself identifies as the self, then consciousness is again the self. [35]

d. **No extra-self exists beyond the conscious process.** There is no evidence of a separate "owner" of experiences standing outside the experiences themselves. The supposed inner observer, the entity that is said to "have" the experiences, is itself an idea or evaluation *within* experience. In short, we have no grounds to posit a homuncular self which is distinct from the stream of consciousness; what we need to identify as the self is inevitably encompassed by the process of experiencing.

e. **Therefore, the self is identical to the ongoing consciousness process.** The person *as a self* is the continuous run of conscious experience, rather than something apart from it. There isn't a two-tier system of an inner observer plus the experiences, the only comprehensible mapping to the "self" is the integrated conscious process itself, including the experience of being an "I".

Explanation | Elaboration: In reaching this conclusion, we clarify that the "I" or self is nothing over and above the processes of consciousness. The sense of an inner observer is an *aspect of the experience*, and is not evidence of an independent entity [67]. As such, to talk about the self is simply to talk about the unified process of consciousness (with all its content and the first-person perspective). There is no separate soul or ego or entity behind the stream of experience, watching a cartesian theatre; the self is instead the active experience process in its entirety.

3. Each Run of Consciousness is a Unique, Unrepeatable Identity

a. **Definition of a "run of consciousness":** A run of consciousness refers to one particular contiguous, causally connected, and uninterrupted stream of conscious experience, a single continuous operation of the consciousness process from start to finish. For example, the period from waking up to falling into dreamless sleep can be considered one run of consciousness, granted that there are no interruptions which interrupt the continuity of the process.

b. **Each run is defined by a *unique* continuous sequence of experiences:** Because a run of consciousness is an integrated process through time, each such run is characterized by its particular sequence of thoughts, sensations, and experiences, and by its continuous progression without gaps. No two runs occur in the same time and state; each has a distinct historical sequence.

c. **A run, once ended, cannot resume, & any restart creates a new run:** Once a given run of consciousness comes to an end (once the process is halted), that exact run cannot be literally

[35] The arguments for this need to be worked out extensively, but the idea is that anything that a conscious dynamic can point to is going to be within experience, and since the individual, consciousness, and experience are directly equivalent under this view, it is not possible for a system to identify something as the self without pointing to itself in a direct manner. The second idea is that, if we ignore what a system perceives or determines to be the self, and instead take the subjective observer to be the self, then we are again directly pointing to experience or consciousness.

restarted. Any subsequent "revival"[36] of consciousness (after an interruption) constitutes a *new* run, and *not* a continuation or instantiation of the old one, as continuity was broken.

d. **Therefore, the self is a run of consciousness [2e] which is a single [3a], unique [3b], un-reinstatable [3c] dynamic:** A particular run of consciousness, with its specific, uninterrupted flow of experience is a one-time, unique occurrence. It defines one personal identity for its duration, an identity that cannot be reproduced or continued once that conscious stream has ceased.

Explanation | Elaboration: This means that a person's identity, understood as a specific stream of consciousness, is bound to one continuous dynamic. Once that stream ends, the personal identity tied to it is finished and cannot be duplicated. Even if the subsequent conscious experience is very similar, it would still be a numerically distinct instance. In other words, each person's consciousness (considered as one uninterrupted run) is a one-of-a-kind structure and dynamic in time, a unique self that arises and exists only over the course of that particular stream of experience.

4. Cessation of Consciousness as Personal Death

a. **Self = consciousness [2e]:** From the above, the self is identical to the ongoing conscious process (the run of consciousness). One's personal identity *is* the continuous stream of experience and nothing else.

b. **If a process stops, the entity identical to the process stops [3d]:** Generally, if some entity Y is identical to a process M, then Y exists only as long as M is occurring. When M ceases, Y ceases to exist as well (because Y *is nothing but* that process).

c. **A run of consciousness ends when experiencing ceases [1c]:** When the process of experience ends, say due to brain activity stopping or going completely dark, then that particular run of consciousness is over. There is no continuing consciousness in waiting or stasis once experience has fully stopped.

d. **Definition of "Death":** For an entity to have died, it must have ceased to exist as that entity. For an entity that is a process to have died, it must have ceased to operate [4b].

e. **Therefore, when a run of consciousness ends the self ceases to exist [4c], and effectively the conscious entity, the person, dies [4d]:** Because the self *is* the conscious process, the end of that process is the end of the self. The termination of an individual's continuous run of consciousness marks the **death of that person as a conscious being**. In other words, when your run of consciousness shuts down and does not resume, *you* [2e|3d], as that conscious entity, are gone.

Explanation | Elaboration: This conclusion defines death in terms of the end of the particular process we take to be central. As such, the end of consciousness is the end of *the person or entity*. Even if a body remains, biologically active, after consciousness irreversibly ceases (imagine an irreversible coma, for instance), the person associated with that consciousness is no longer existent. What we consider "you" does not outlive your conscious process. In a meaningful sense, to lose the consciousness that constitutes your self is to lose your existence as a specific being. This is a bit more intuitive as a concept if we were to imagine a person who was put under anesthesia at age 20. The anesthesia is, in theory, reversible, but in our imagined scenario, the

[36] This is a point where the view being expressed here is somewhat unwieldly using the English vocabulary. Typically, we would assume that any time that a machine or system begins to instantiate a process anew, that the system is restarting the process in question, meaning that the system is starting anew the *same* process. This is opposed to the view that the system is instantiating a new iteration of a similar but *non-identical* dynamic, and since the opposing view is our view, we find ourselves in the unfortunate position of having to explain this.

person is never taken out from anesthesia, and his organs fail one day and the body of the person is no more. Where before, in theory, the person could have been made conscious, now that is no longer possible. Suppose we are asked when the person ceased to exist. His body ceased functioning in 2025, aged 65. But if we are to take a view of the mind or brain being the person, then he died in 1980, at 20 years of age. If we believe that such a thing as brain death exists, and that it has an importance at least somewhat equivalent to that of complete bodily death, then this should not be an entirely foreign concept to us. That the man stopped experiencing anything at all at 20 is somewhat, if not exactly, equivalent to the scenario of a person with severe brain damage that may, with the right intervention, come back again. But despite this possibility they never do. There are arguments that this is not possible definitionally [13], but those are relatively simple assertions to shoot down, and as such we will deal with them later.

5. Bodily or Memory Continuity Does Not Preserve a Consciousness-Run's Identity

a. **The body can persist without consciousness:** One's bodily organism (including the brain) often continues to live and function during periods when consciousness is absent. For example, in dreamless deep sleep or under general anesthesia, the body is still present and alive while conscious experience is entirely gone. Thus, mere bodily continuity (the same physical organism existing over time) does not guarantee an unbroken continuation of consciousness.

b. **Memories can bridge gaps that consciousness cannot, but these are not the same kinds of continuity:** Psychological continuity, such as having memories of past events, can exist even if consciousness was interrupted in the interim, that is factually and trivially true. For instance, you can remember what happened yesterday despite having been completely unconscious during the night. This means memory continuity can span across a break in consciousness. That being said, it is not the same as uninterrupted experience, but rather forms a sort of *connection* between separate conscious periods. More specifically, if we are to return to our nuclear fusion reactor, the formation of a memory is equivalent to a panel being shifted, or a switch being turned, by the activity of the fusion reactor, such that when fusion ceases, the shifted panel or switch remains changed. The next time the fusion reactor starts up a fusion reaction, that reaction may reflect the changed switch or shifted panel, and that will be an effect of the previous run on the current run, but that still isn't the same run, because the original run already ended.

c. **A new run of consciousness in the same body (with memories) is still a new run [3d]:** If the continuous stream of consciousness is broken (say, by a period of unconsciousness), then any later revival of consciousness, even in the same body and with retained memories, constitutes a numerically distinct stream. The original run ended at the point of interruption, and what comes after is a new instance of consciousness, it may inherit memories and run similarly, but it is *not* literally the same continuous run that was halted.

d. **Therefore, since memory continuity and | or bodily continuity cannot preserve the identity of a consciousness run once it stops, each run of consciousness is still a new run:** Only uninterrupted consciousness can preserve that identity, since identity is directly dependent on experience | conscious processing. If the conscious process has ceased (even temporarily), the personal identity tied to that original run is gone; the fact that the same body survives or that memories are later accessible does not change this. A later consciousness in the same body, even if it recalls the earlier life, is a **successor** to the original identity, not a direct continuation of it.

Explanation | Elaboration: This conclusion is meant to bring forth that personal identity, understood as being *the very same thing as it consciousness*, is not saved by keeping the body alive or by retaining information (memory)

through a gap. These factors can make the new consciousness very *similar* to the old one (which is why, in everyday life, we treat a person before and after sleep as the same individual). However, similarity or continuity of body and memory is not the same as continuity of a single experiential dynamic. If there was a true break in the stream of experience, then from a strict metaphysical standpoint, the original conscious self has ended, and a new one has begun, no matter how much the new one may resemble the old.

6. Unconsciousness and a Daily Death (Breaks in Consciousness and Identity)

a. **Total unconsciousness is a halt of the conscious process:** States of deep unconsciousness, for example, dreamless sleep or deep general anesthesia, are periods in which the person has no conscious experiences at all. In such a state, the process of consciousness is completely *offline* or halted, and equivalently, there is an end of that particular stream of experience.

b. **Entering sleep is entering unconsciousness, which ends the current run of consciousness:** By the earlier reasoning, when consciousness ceases, the run of consciousness, and the self identity with it, ends. So, falling into a state of dreamless sleep or any complete unconsciousness marks the termination of the *previous* conscious run. The continuous stream is broken at that point, effectively, that conscious self has come to an end.

c. **Awakening initiates a new run of consciousness:** When the person later wakes up or regains consciousness, a conscious process resumes. However, because there was an intervening gap of total unconsciousness, this resumed consciousness is in fact the **start of a new run**. It may occur in the same body and draw upon memories from before (thanks to the brain's memory retention), but it is a new continuous process, not a seamless continuation of the old one.

d. **Therefore, each episode of complete unconsciousness creates a break in personal identity, essentially a death of the prior self, with a new self arising upon awakening:** In everyday terms, the *you* that existed during yesterday's conscious period ceased upon entering dreamless sleep, and the *you* that awakens today, while physically and psychologically continuous with the prior you, is a new instantiation of a conscious self. This phenomenon can be understood as a "daily death", each night's deep sleep, or any period of total unconsciousness, is the end of one personal identity, and the subsequent awakening is the beginning of another.

Explanation | Elaboration: We use the term "daily death" to stress that, under this view, the break of consciousness that occurs in dreamless sleep *is not fundamentally different from death*, as it is the end of the particular conscious self that was alive before the break. Of course, the *new* conscious self upon waking "inherits" the same body and brain, with memories and personality intact, which is why practically we consider the new run to be "the same person." But strictly speaking, that continuity is deceptive as what has "resumed" is a new run of consciousness, one that is only historically connected to the prior run via memory and physical infra-structure. In a strict sense, the original stream of consciousness and thus the original person as a subject of experience was discontinued and is gone. In this way, every total interruption of consciousness is like a small death of the self that was, followed by the birth of a new self when consciousness returns.

In sum, the continuity of consciousness is the sole basis for the persistence of personal identity over time. A person is a specific run of conscious experience, and only an unbroken run constitutes the same person continuing to exist. When the conscious process stops, the personal identity defined by it terminates, this is death in the most direct personal sense. Neither the survival of one's body nor the retention of one's memories can by themselves carry a consciousness over a genuine break, and their existence does not provide

the same kind of moral consideration that they do when there is a consciousness attached to them. Personal identity lives and dies with the run of consciousness; continuous consciousness means the self endures, and the cessation of consciousness means that self has ended. This distinction is utile as it primes our intuitions on the subject, and additionally will become important in examining the cases of Jahi McMath and TJ Hoover which follows our examination of non-pathological states of unconsciousness.

The Little Deaths: The Neuroscience of Unconsciousness in Anesthesia and Sleep

Anesthesia and Sleep are the two primary non-pathological instantiations of unconsciousness. As opposed to a coma or unconsciousness due to traumatic brain injury, these are not seen as atypical or detrimental losses of consciousness, and as such merit consideration due to their typicality. In the following sections we will first look at why and how various anesthetics produce unconsciousness and in doing so address some potential questions on the characterization of anesthesia as unconsciousness and a death through the thorough examination of neural dynamics under anesthesia. We will then characterize sleep, its similarities to anesthesia, and evaluate the neural characteristics of sleep which motivate the determination of sleep as unconsciousness and a complete end to the dynamic of consciousness.

We will ultimately demonstrate that Nas, who once said "I never sleep, 'cause sleep is the cousin of death", was empirically justified in his decision, by neurologic criterion.

Anesthesia: Distinct Mechanisms and Global Effects Resulting in Equivalent Neurophenomenological Dynamics

There are many kinds of anesthetics, but here we will cover two of the most well understood anesthetics, propofol, the most common, and Xenon, which has a different mechanism of action globally.

Molecular Mechanisms to Global Dynamics: Propofol

To understand Propofol, it is important to understand the processes it potentiates, namely the effects of GABA on the corresponding GABA-A receptor. Neurons function by the propagation of an electrical gradient through itself. There are voltage gated ion receptors which open when there is a sufficiently large difference between the interior and exterior charge, where the interior of the cell is sufficiently positive, and the exterior negative. As such, its "resting state", when it is closed, is when the interior of the cell is more negative than the exterior of the cell. That is called its hyperpolarized state. When a neuron receives sufficient electrical signal, as measured by a sufficient increase in internal positive charge relative to the exterior, a voltage gated ion channel opens, allowing more positive ions into the cell, thereby creating a more positive interior local environment. That the area has more positive ions in it causes neighboring voltage gated ion

channels to open, making *their* local environment more positive, and causing other neighboring voltage gated ion channels to open, and so on and so forth until the signal reaches the end of the line. The cell then works to increase the interior negativity, bringing the cell back to hyperpolarization, its "resting state", thereby preparing the neuron to go again.

GABA-A receptors are ligand activated receptors, meaning that they requires a "key", in this case GABA, to open. Once open, that channel permits chorine ions, which are negative, to enter the cell, thereby making the interior of the cell more negative. As the exterior is typically[37] relatively more negative than the interior, the open GABA-A receptor increases the internal negativity of the neuron. This is the inhibitory effect. The reason this is an inhibitory effect is that there must be an overall net positive charge on the interior, and the increased negativity caused by the GABA-A receptor activation means that the neuron requires more stimulation, more positive ions and signaling, in order to reach the necessary interior positive gradient which would allow the voltage gated ion channels to open and subsequently propagate the signal. The result is that GABA, by opening the GABA-A receptor, makes a given neuron less likely to fire, thereby inhibiting it.

Propofol is a small molecule, which when injected at high doses leads to unconsciousness. At the molecular level, propofol molecules nestle into nooks at the GABA-A receptor. Because propofol exerts its effects by binding at a non-standard site and potentiating the effects of the mechanism it interacts with, it is considered a positive allosteric modulator. Propofol, in binding to those nooks, helps "wedge" open the receptor when it is present at low doses and GABA is bound to its site on the GABA-A receptor. Because there are *multiple* sites for propofol to wedge itself into on a single GABA-A receptor, at high doses, the wedges exert a combined conformational effect, which is capable of opening the GABA-A channel without the presence of GABA at the receptor site. This dual mechanism is important to understanding the different EEG signatures that arise in accordance to propofol administration, with both constituting unconsciousness, but in different manners.

One may expect that the above explanation is sufficient, and that when propofol is administered, the entire brain, or at least the important parts, go silent, and of course this leads to unconsciousness. Were that true, you would expect to see a EEG flatline or minimal activity. What we see instead is the emergence of slow waves across the cortex, and slower than normal but still not slow waves in certain parts of the cortex. So, the brain is still active. What makes propofol lead to a lack of consciousness is not that it turns off the neurons exactly, but that it disrupts, and negates the emergence of, an integrative continuous dynamic across the entire cortico-basal ganglia-thalamo-cortical network. It does this in multiple ways [68], but the most apparent and effective is that it prevents the emergence of any complex responsive dynamics by creating localized waves of propagation across discrete sections of cortex. The reason this happens is that the inhibition caused by propofol ensures that the normal informational processing of a given neuron does not occur. Where a neuron may have fired with two inputs under normal conditions, now it requires far more activation.

As such, only the activation of entire swaths of neighboring cells can cause any one given cell to fire. What that means is that signals do not propagate very much and as such are not a part of any larger dynamic, and that instead, simple waves of activation, which have no informational capacity, replace them. That the

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[37] This is not always the case. All children and some adults have alternative [164] active (meaning not passive, meaning they transport things one way only, regardless of the gradient, leading to an electrostatic gradient) mechanisms which introduce negatively charged chlorine ions into the neuron. Because these mechanisms make the interior more negative than the exterior, the opening of the passive GABA-A receptor by propofol and other GABA-A promoting drugs (barbiturates) is conjectured (by me) to lead to the paradoxical seizures seen in a rare subset of adults treated with propofol who develop seizures during propofol administration [165]. Despite the relative simplicity of the mechanisms in play, it is currently unclear whether the mistakes made by clinicians, especially in claiming that such effects are anesthetic-indifferent [165] are simply due to a lack of proper promotion of the idea that increased interior neural negativity is causally implicated, or if it is the case that such a connection has not been adequately proposed or directly presented in a coherent manner.

effects of propofol vary according to the regional distribution and also the particular GABA-A receptor subtype under consideration [69] has a further disruptive effect, where the lack of a coherent signal further ensures that a global integrated dynamic does not come to pass [70]. Together, the fragmentation of activation and prevention of complicated integrative dynamics [71], especially in the cortex and thalamus, caused by propofol [72] are what cause unconsciousness upon propofol administration.

Xenon

Xenon is an atypical general anesthetic in a number of ways. The most immediately apparent is that, where most anesthetics are molecules, Xenon is a chemically inert noble gas, an element; meaning that it exerts its anesthetic effects as a single atom. Xenon is able to exert effects as a single noble element[38] due to its stable but still active and deformable electron cloud [73]. Where most anesthetics like propofol and barbiturate anesthetics work via GABA receptor interactions and potentiation, Xenon has a relatively miniscule effect on GABA receptor function [74]. Xenon negates consciousness in that it primarily decreases excitatory signaling by blocking the binding to glutamate AMPA [75] [76] receptors and additionally takes residence in the spot typically reserved for its ligand "key", glycine[39]. It also inhibits HCN2 channels, which are a central part of the mechanism which produces "pacemaker" activations in thalamic "pacemaker" neurons [77].

At the network level, Xenon diffusely inhibits multiple mechanisms necessary for the formation of complex integrative dynamics of the kind relevant for consciousness. AMPA receptors, due to their quick deformation and signaling time [78], are responsible for a significant majority of the fast excitatory post synaptic signaling in the central nervous system [79]. Since they are especially present in and necessary for cortical pyramidal signaling, the blocking of AMPA receptors reduces the capacity of these neurons to signal quickly, and further reduces the amount of signaling that reaches the same cortical neurons. While they can and do still signal, it is not at the speed or fidelity necessary to coherently communicate with other cortical or global ensembles [76]. This slow messy signaling leads to the propagation of characteristically homogenous [80] slow waves of activation across the brain, especially the cortex. This is only part of the dysregulation involved.

HCN2 channels create an internal oscillatory signal by introducing positive ions into the neuron once it is hyperpolarized above a certain point. The introduction of positive ions by HCN2 brings the neuron closer to the positive internal gradient necessary for it to fire, and because the neuron has mechanisms for introducing repolarization, it ends up activating the neuron. Critically HCN2 receptors open and close on the scale of hundreds of milliseconds, and so are able to create pacemaker signals. HCN2 is additionally relevant here as it has another ligand, cAMP, which permits tonic rapid firing, by generally reducing the threshold for activation [81]. HCN2 receptors are used anywhere rhythmic activation is needed, in cardiac tissue to provide a pacemaker, in cortex, and in thalamus to provide regularity of signaling [82]. In thalamus, HCN2 oscillations drive the cortico-thalamic synchrony required to produce the low frequency oscillating waves which occur in sleep [83]. HCN2, when partially potentiated by cAMP increases excitability of a cell to a middle amount, permitting the transfer of signals through and within the thalamus. When Xenon exerts its effect on HCN2 by clogging up the HCN2 cAMP receptor area, it results in a decrease of binding by cAMP to HCN2, thereby disabling the excitability resulting from HCN2 potentiation.

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[38] The Krypton and Argon, also noble gasses, similarly seem to have anesthetic properties. They are not used because the dosages required are not feasible under typical atmospheric conditions [166]. Their decreased efficacy is presumably due to their decreased capacity for site interaction, due to the tighter binding and rigidity of their electron clouds.

[39] It is generally understood that glutamate is primarily excitatory and that glycine is primarily inhibitory, but it isn't in this context, where it is a co-agonist at NMDA receptors [167].

Sleep: A Necessary Dynamic for Continual Function Requiring Unconsciousness

Clinical Case Studies & Evaluations: Disorders of Consciousness, Brain Death, TJ Hoover, Jahi McMath, and How to Tell the Difference

In this section, we will examine various kinds of unconsciousness, starting with the most clearcut cases of coma with associated clearcut unconsciousness, then moving to the most clearcut cases of unconsciousness under anesthesia, then examine sleep, and its similarities with anesthesia on a neurological level, and finally we will examine the cases of TJ Hoover and Jahi McMath. For the Jahi McMath case we will go over the timeline and clinical evaluations given by employees and representatives of UCSF Oakland Children's Hospital and concurring ethicists as background, then we will go over the more empirically parsimonious timeline and clinical picture for accurate analysis, and then we will go through the clinical picture as understood through the dual theory of death and CSR framework, focusing mostly on the empirical picture in order to best ground the analysis.

Current Definitions and Clinical Criteria for Brain Death, Coma, Vegetative States | Unresponsive Wakefulness Syndrome, Minimal Consciousness: The Clinical Norm

The examination of coma, vegetative states, and minimal consciousness is well inside the domain of general neuroscience and the practice of neurology. As such it is utile to briefly go over the current definitions and criteria of those states from a clinical perspective.

Brain Death

The most recent authoritative definition of Brain Death or Death by Neurologic Criteria, the 2023 DNC Guidelines, abbreviated here as "BD/BNC" or "2023 Guidelines", defines death as the "permanent" cessation of neurologic functions [84]. The term "permanent" constitutes the most significant departure from previous definitions of brain death. This criteria differs substantially from the criteria of "irreversible" cessation of neurologic function. The irreversible criteria requires that the brain damage not be reversible, while the 2023 Guidelines *permit that the brain damage be reversible*. The term *permanent* in the context of brain function loss is that the brain functioning will not resume ***spontaneously***, and that medical interventions ***will not be used to attempt restoration of function***. The difference here is quite severe, as it introduces a much more subjective measure, which is dependent on the actions that the relevant authority will take, or not take, to recover functioning.

To demonstrate the issue in this definition, let us imagine that we used the same reasoning and logic in the cardio-pulmonary criteria for death. So, for me to be dead by cardio-pulmonary criteria, it must be determined that my cardio-pulmonary system will not resume ***spontaneously***, and it must be determined that medical interventions will not be used to attempt the restoration of function of my cardio-pulmonary system.

There are quite a few examples that can demonstrate how ill-conceived this is, but lets stick to the basics. Say that I take one too many expresso shots today at the WeWork on Wall Street, leading to pulseless ventricular tachycardia, That isn't good. Luckily for me, a doctor, my personal doctor from NYU Langone no

less, happens to be standing by, and is on duty, so no legal problems would arise from attempting treatment. My dear doctor, let's call them Dr. Huey Lewis[40], for whatever reason, is asked by a bystander, "is Aramis dead?" Dr. Lewis' response will depend entirely on whether or not he decides to intervene, and ***regardless of his determination he will be right***. If Dr. Lewis decides to intervene by pulling the automated external defibrillator off the wall and defibrillating me, the pulseless ventricular tachycardia will resolve, my heart will start beating normally again, and I will go on to do whatever I was liable to do in life.

However, if Dr. Lewis decides that he will not intervene, because for instance I once told him that I don't like Huey Lewis and The News, well, I will indeed be dead, before my brain goes dark and before my heart loses the possibility of being revived. That Dr. Lewis decided that there will be no rescue attempt made, has determined the fact that I am dead, and because of this decision, my heart will not restart, my brain and body will starve of oxygen, and after some time I will also be irreversibly dead. What should be painfully evident is that the criteria being dependent on whether or not it is ***determined*** that an intervention will be ***attempted*** introduces a self-fulfilling prophesy, where the "evaluation" or determination of my condition at T_0 ensures the truth or validity of that determination at T_2. As should be painfully evident, this is not a principled or properly considered way of going about determining death, both in the cardio-respiratory or neurological scenarios. This odd criterion, applied anywhere, is plainly and brazenly non-sensical.

> "Oh, you have a tension pneumothorax[41]?! Well, I am working on my work-life balance. I am on my break, and I have determined that you are cardio-respiratory-dead, and as such I will not do the thing I am trained to do, namely poke a hole in your chest cavity that will release the trapped air in your chest, allow you to breathe, and thereby save your life. I could do it, if I really felt like it, but my life coach taught me to center myself, and determining you are alive would make me have to intervene here, which isn't centering ***me***, now is it?" – Guy working on his work-life balance (probably).

The same 2923 Guidelines, authored in collaboration with the American Academy of Pediatrics, Child Neurology Society, and Society for Critical Care Medicine, the American Academy of Neurology, states an interpretation of the UDDA, or Uniform Determination of Death Act which is similarly distinct from prior practice. They interpret the UDDA requirement, that there be a "loss of all functions of the entire brain, including the brainstem", as requiring "loss of ***function*** of the brain ***as a whole***, including the brainstem, resulting in coma, brainstem areflexia and apnea in the setting of an adequate stimulus." (emphasis added)

It should be noted that the admittedly creative interpretation of the UDDA standard by the writers of the 2023 Guidelines is ***not*** a valid interpretation of said standard, and cannot be logically understood to be one. I am aware that the authors understand the differences introduced, so the choice to state this is an interpretation and not a re-definition is certainly a choice that was made, albeit one that was necessary to the entire endeavor.

I will now cover in depth the failure of the new definitions philosophically, specifically in discussing their blatant treatment of "death" as a definition to be re-written, and not a phenomenon to understand and treat accordingly. To be painfully explicit, there is a very substantial difference between "loss of all functions of the entire brain", which requires that the ***entire*** set of the various functions of the brain be lost, and the "loss of function of the ***brain as a whole***", which is not only a standard which is far more open to interpretation, but is also a standard that quite explicitly and egregiously contradicts the UDDA guidelines by

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[40] As in Huey Lewis and The News.

[41] Very common injury at crash sites, basically the pleural space, or rather the upper abdominal cavity, between the lungs and the chest, is filled with air, which doesn't allow the lungs to expand and as such leads to hypoxia, and, if left untreated, death. The immediate treatment is to take any sharp (or dull if you are in a pinch) object and stab through the chest wall through to the cavity, allowing the trapped air to escape and the lung to begin working again.

permitting that there remain active ***some set*** of the various functions of the entire brain in a canonical brain death determination.

The contradiction is as simple as it is irreconcilable, and continues into its treatment of the brainstem. Where the UDDA requires that "all functions of the entire brain, including the brainstem" be lost, the 2023 criteria require "loss of all functions of the entire brain, including the brainstem, resulting in coma, brainstem areflexia and apnea in the setting of an adequate stimulus." This parsing defines, in situ, the functions of the brainstem as being primarily, if not exclusively, its role in the reflexes, and further in its role in instantiating the breathing reflex. As will be made evident in the case studies, the lack of brainstem reflexes is by no means a conclusive determinant of death. This standard is made all the more problematic by the declaration within the 2023 standards that clinicians ***should not use neurophysiologic tests***, namely EEG, auditory evoked potentials, and somatosensory evoked potentials, because these provide "i***ncomplete information about the entire brainstem***" [85].

This restriction, meant to reduce inadequate determinations of brain death, has the adverse effect of making EEG an unacceptable test, period. While this is ideally intended to ensure that a flat EEG is not used to substantiate an incorrect diagnosis of brain death, it also has the clear adverse effect of contraindicating EEG in the determination of ***not*** brain death. This removes the only function-based ancillary tool that could detect residual cortical activity. Activity which, as we will discuss later, can be indicative of pending recovery of function. It is also important to note that the recommendations have already been interpreted in the clinical setting as stating that EEG is no longer acceptable as an ancillary test. At least in one hospital, medical staff policy has already been amended to reflect said guidance [86].

Clinically speaking, according to the 2023 standards, themselves coherent with the 2020 World Brain Death Project recommendations, it is generally required for the determination of brain death that the presence of catastrophic permanent brain injury be present, coma be present, and that the brain injury be additionally identified as having an etiology known to lead to BD/DNC without confounders. Additionally, BD/DNC determination requires that there be no preservation of brainstem reflexes, motor movements mediated by the brain or brainstem, and that there is no spontaneous breathing. Once that is the case, then it must be established that the brain injury is *permanent*, that function is lost and will not resume *spontaneously* and medical interventions *will not be used* to attempt to restore function.

For cases involving damage primarily to what is essentially the brainstem and posterior-inferior brain areas[42] (posterior fossa), there must also be neuroimaging demonstrating evidence of "catastrophic supratentorial injury", basically catastrophic damage to the non-brainstem and cerebellum parts of the brain. Optimal conditions must otherwise be ensured in terms of blood pressure, body temperature, confounding metabolic or endocrine issues, and confounding presence of a determined set of drugs, primarily paralytics or depressants.

Finally, the clinician or clinicians (in the case of pediatric DB/DNC evaluation) must have some credential or be adequately trained and competent in BD/DNC determination in accordance with local laws and institutional standards. There are some additional criteria and specifications, but these are the important ones. Although the new recommendations ameliorate many obvious confounders, the recommendations taken as a whole lower the evidentiary bar, replacing "irreversible" with "permanent," defined as a loss that will not resume spontaneously and will not be treated, and further loosens requirements by striking EEG from the list of acceptable ancillary tests. The first change converts a physiological impossibility into what could uncharitably be called a policy decision, and the second abandons the only functional modality capable

[42] We will cover this later, but do note that a substantial number of brain death cases will demonstrate brain stem damage of this sort, even without localized damage to the area.

of detecting residual cortico-thalamic dynamics in situ. Together, they make it easier, not harder, for a marginally salvageable patient to satisfy the requirements for BD/DNC.

Coma

Coma is defined as a state of "pathological unconsciousness", and is characterized by the "complete loss of spontaneous arousal, sustained eye closure despite the introduction of noxious stimulation, and total absence of behaviors associated with "normal" consciousness." Clinically, all of the following are required for a determination of coma: No following of commands, no intelligible speech or recognizable gesture, no volitional movement[43], No visual pursuit, fixation, saccade to stimuli, or eye opening or closing to command, no drugs or disorders which may confound, and no evidence of cognitive motor dissociation based on electrophysiological or functional imaging (if available) [87].

Vegetative State | Unresponsive Wakefulness Syndrome

Unresponsive Wakefulness Syndrome, previously known as Vegetative State and referred to here as UWS/VS, is generally defined to be a state where a person continues to exhibit sleep-wake cycles, autonomic functions such as spontaneous breathing, and other brainstem and reflexive functioning. The term unresponsive wakefulness is an adequate, if succinct, characterization.

Clinically, UWS/VS is characterized by intermittent arousal or wakefulness, either spontaneous or stimulus induced, which nonetheless lacks behavioral signs of conscious awareness. There must be; no evidence of awareness of self or environment, an inability to interact with others, no evidence of sustained reproducible purposeful or voluntary behavioral responses to visual auditory tactile or noxious stimuli, an absence of language comprehension or expression, intermittent wakefulness demonstrated by the presence of sleep-wake cycles, sufficient preservation of brain stem and hypothalamic functions to permit survival with medical care, incontinence, no confounders, and variably preserved cranial-nerve reflexes[44] [87].

Minimally Conscious State

A Minimally Conscious State is defined primarily by the presence of cognitively mediated behavior, even if minimally present. It is further characterized as a positive indicator for recovery of function.

Clinically, a determination of a minimally conscious state requires that the patient be able to follow simple commands, verbally or gesturally respond in the affirmative or negative regardless of accuracy, demonstrate intelligible verbalization, non-reflexive purposeful movement or vocalization or affective change in contingent relation to environmental stimuli. Similarly to the previous criteria, there must be no confounding issues or conditions.

Integration and Significance

The above definitions and clinical determination criteria generally constitute the accepted standard of determination for the above disorders of consciousness. It is important to note that the adoption and specific determination criteria vary substantially in practice, and that there are further deviations and sometimes outright inadherence to these standards. A principled reading of the timeline and clinical picture of the TJ Hoover case as alleged will demonstrate what could be characterized by some as blatant malpractice and substantial inadherence to minimal standards. The Jahi McMath case, as understood by a parsimonious reading of the clinical and neurological evidence, will demonstrate a case of substantial clinical adherence to

[43] Excludes reflexive movements.
[44] As differentiated from brainstem reflexes

standards which nonetheless failed to adequately treat or characterize said case, and additionally demonstrates clinical inadequacies of the sort that are relatively common in practice and could be reasonably committed by a clinician of average capacity, but which remain inadequacies nonetheless.

Jahi McMath: A Story of Mildly Difficult Neuroscience, Motivated Thinking, and Potential Failure in Practice Through Strict Adherence to Procedural Rules and Norms[45]

The First Death of Jahi McMath

On December 9th, 2013, Jahi McMath went to UCSF Benioff Children's Hospital Oakland to treat her obstructive sleep apnea. She underwent an adenotonsillectomy, uvulopalatopharyngoplasty, and submucous re-section of the bilateral inferior turbinates. Around 7 PM she was transferred the intensive care unit, and was alert. She promptly began bleeding "copiously" from the nose and mouth, which was treated by suctioning, gauze packing, oxymetazoline nasal spray[46], and tranexamic acid[47], which blocks the breakdown of blood clots. No cauterization took place. By 11:00 P.M. of the same day, she had lost at least 250 mL of blood. At 12:30 A.M. December 10th, 2013, Jahi sat up, breathed in blood, and went into cardiorespiratory arrest. She was given atropine[48] and epinephrine[49], but her "heartrate"[50] still dropped, and she became asystolic[51] at 12:43. Chest compressions were administered immediately, and her pulse returned at 12:53 A.M. There was an initial lack of chest rise with bag and mask ventilation, and at 12:55 A.M., an anesthesiologist intubated Jahi, after extracting around 300 mL of blood. Blood continued to flow from the suction, and after two large blood clots emerged, ventilation markedly improved. At 12:58 A.M., Jahi's blood oxygen saturation was at 98%. She was administered stabilizing IVs of sodium bicarbonate [52], saline [53], albumin [54], and O negative blood[55], this was meant to replace lost fluids and blood lost, a process known as reperfusion.

The surgeon arrived at 01:35 A.M. December 10th, 2013, at which point the blood loss had largely abated. A source for the blood loss was not identified, but given the vessel rich nature of the area that was

[45] The following is derived from reports and medical examinations of Jahi McMath [88], and basic clinical science [89] [90].
[46] Presumably to constrict blood vessels and therefore decrease loss of blood. May have had unintended effects.
[47] Anti-fibrillation drug. This is an odd thing to give, since by 2010 Atropine was no longer recommended for these kinds of cases, and was certainly not recommended for children [171]. ICU charts are notoriously unreliable, but the cardiac resuscitation timeline during this section is concerning [172] even taking that into account.
[48] Increases heart rate.
[49] Increases blood pressure through vasoconstriction.
[50] This presumably refers to pulse-less electrical signals. This is odd to mention, as in general, when you have called a code blue, you either have a pulse (ROSC), or you have VF/pVT, PEA, or asystole. You do not track "heart-rate".
[51] Meaning her heart completely stopped beating, and demonstrated no electrical or mechanical activity. This is characterized by an ECG flatline, or a droning beep like in the movies.
[52] Ph regulator.
[53] Increase vascular volume.
[54] Prevents fluid loss from blood vessels.
[55] Replaces lost blood.

operated upon, there was no paucity of potential rupture sites. A bronchoscopy found no blood or blood clots occluding what was perceivable. Between 2:00 and 2:30 A.M. of the same day, there was a spike in blood pressure (reaching 200-250/150-160) which likely increased cerebral inflammation and intracranial pressure. The increase in pressure necessitated the cessation of epinephrine and dopamine IV administration. Nicardipine and Labetalol, which relax blood vessels, thereby reducing blood pressure, were administered, in an attempt to control the jump in blood pressure.

24 hours passed before neurologic assessment, due to restrictions in diagnosis owing to sedation via fentanyl and dexmedetomidine and paralysis via vecuronium. Nonetheless, there were clear demonstrations of neural issues, most important of these was the gradual increase in blood and pulse pressure, concurrent with decreasing heart rate, which is a classic indication of Cushing reflex, a signal of substantially increased intercranial pressure. Had the neurological assessment been performed, the clear signs of CNS edema would have been ascertained, and with treatment, subsequent damage to the brainstem [91], white matter [92], basal ganglia, and thalamus [93] may have been avoided.

At 12:00 A.M. December 11, heart rate increased to 100 bpm, and pupils were dilated and non-reactive to light. Herniation was suspected at this point, so mannitol[56] was administered to reduce edema, and vecuronium[57] was discontinued. A CT scan demonstrated severe damage, loss of grey matter white matter differentiation, and severe pressure evidenced by the occlusion of sulci due to internal pressure pressing the brain into the skull and flattening the wrinkles of the brain. Basal ganglia were edematous, the base parts of the brain were so inflamed that the spaces which are normally fluid filled were brain filled instead, and finally that there was evidence that parts of the brain, especially the brainstem, had been pushed into the large opening at the base of the skull. Further attempts were made to reduce inflammation, and fentanyl and dexmedetomidine drips were discontinued (likely on presumption of catastrophic damage warranting DNC diagnosis). A neurosurgical consult determined that surgical intervention, including surgical measurement of continuing brain inflammation was not indicated. An EEG was performed according to the relevant guidelines for suspected cerebral death, which returned an isoelectric, or flatline, reading.

At some time in the morning of December 11 a neurology consult determined that there Jahi demonstrated no response to environmental or noxious stimuli. Reflexes were absent, including an apnea test. Sedatives and paralytics had not been given enough time to clear out, and there was no attempt to reverse sedation from fentanyl (via, say, naloxone), or to guarantee that the paralytic vecuronium had worn off. This was the first formal brain death examination, which indicated that if an examination after at least six hours found the same, those findings would be "compatible with irreversible brain cessation." At some point on December 11th, presumably after this first examination, the local organ procurement organization was notified.

Mid-afternoon of the same day, blood pressure and heart rate spiked dramatically, potentially attributable to a sympathetic storm from brainstem compression. Over the subsequent 20 hours cardiac measures were variable and were treated variably with nitroprusside and esmolol. After an additional 24 hours, blood pressure stabilized to around 120/79 and 100bpm.

December 12th at the morning, further neurological inspection found no brainstem reflexes, and rescued spinal reflexes. A pinch test did lead to an increase in blood pressure, and there was one spontaneous right arm jerk, both of which were not considered counterindications of brain death. A second EEG was performed to confirm to Jahi's family that there was no activity, and again resulted in flatline. A second formal brain death examination was performed in the afternoon, which found again an absence of brainstem reflexes, and a lack of arousal. An apnea test was performed again and failed again. Jahi was determined to

[56] A diuretic.
[57] Vecuronium is a paralytic, which also causes edema.

have fulfilled the criteria for pediatric brain death, and was pronounced dead at 3:00 P.M. the following day, December 12th, 2013. A death summary was entered into the chart and the coroner was notified.

The First Death of Jahi McMath: The Neurological Picture

This clinical picture is not an exceedingly uncommon one, and is quite explicable. The sequence of massive blood loss followed by hypoxia and reperfusion is a well-known, textbook, trigger for hypoxic-ischemic brain injury [95]. This is a kind of reperfusion injury where the sudden influx of oxygen leads to an increase in reactive oxygen species (a variety of reactive oxygen-bearing molecules), that leads to inflammatory cascades being activated, which induces massive inflammation and edema, and subsequent tissue damage. In this case, that is almost certainly what occurred[58], with the ensuing massive cerebral edema causing what is called a tonsillar herniation, where a part of the brain, usually the brainstem, is pushed down through the foramen magnum, which is the large opening at the base of the skill leading into the spinal canal**.**

This is an important, probably the most important, detail about Jahi's case. This precise kind of damage is known to cause severe brainstem damage [96] **and thalamic damage** [97] [98]. While this knowledge of this general pattern would have certainly been present in your average neurologist, that a neuroinflammatory response can continue to cause damage over weeks or even years would perhaps not have been known to clinicians of the era. Nonetheless, the resolution of massive edema of the sort that Jahi presented is generally understood to merit significant attention and treatment. That they did not intervene to ameliorate the inflammation, especially after the CT scan demonstrating a lack of perfusion or differentiation of ***any*** sort, does not break any guidelines as decompressive craniectomy is permissible but not required [92]. The reason why this treatment was considered futile was not provided.

The non-treatment and subsequent death of patients who undergo cardiac resuscitation, especially after a CT scan showing non-perfusion, is so low that it has been proposed to contribute to a self-fulfilling prophecy. One where patients are deemed to be unlikely to recover, and therefore no treatment is provided due to that determination, and the patient subsequently does not recover on account of having not been treated [100] [101]. This effect, at least on the statistical level, leads to deaths that may not have otherwise occurred[59], with something as simple as which doctor ends up looking at a scan indirectly determining the treatment course for patients, leading differential outcomes for said patients, most significantly, death[60]. These kinds of self-fulfilling prophesies are well known in medicine, and are of great concern to clinical neurologists [99].

Jahi had an additional complication in her case which likely had a detrimental effect, that being the pro-coagulant therapies which were used to stem the bleeding. Jahi was administered, over the course of seven hours, Jahi was administered tranexamic acid, multiple vasoconstrictors, tranexamic acid again, platelet

[58] Any other explanation would require astronomically unlikely odds and would require a reconsideration of the last 50 to 100 years of biological and clinical science.

[59] It is important to note that it is not a negligible number of patients who go through cardiac resuscitation and after undergoing imaging end up receiving different kinds of care. In one study, 60% of patients underwent a head CT scan within six hours of cardiac arrest, where 25% of those were abnormal (78% from hypoxic-ischemic brain injury). Of those cases, 46% resulted in different management of their case, with the most common being a de-escalation in care including transition to do not resuscitate status, withholding of targeted temperature management, and withdrawal of life sustaining therapy. In these cases, where hypoxic-ischemic brain injury was established by radiography, progression to brain death was higher, with 44% of those cases leading to brain death, as opposed to 2.9% of cases not diagnosed as such. P was 0.001 [100].

[60] It is also important to note that neuro-intensivists, those who, by virtue of their profession have the most exposure to these kinds of cases, demonstrate low interrater reliability. Meaning that what one intensivist says is hypoxic-ischemic brain injury does not necessarily match what another intensivist says is that same thing. It is also very important to note that neuro-intensivists diagnose hypoxic ischemic brain injury more often [100] and with less inter-rater reliability than critical care, emergency medicine, and general radiology physicians [170].

transfusions, and factor VII[61]. Factor VII, a part of the clotting pathway, was used off-license for the prevention or treatment of bleeding. By 2012, it was counter indicated for use in all patients except those with hemophilia, due to substantial increases in thromboembolic events above baseline, averaging 45%, sometimes reaching up to 105% higher than baseline [102]. This increased the existing post-trauma increases in coagulation factors, and likely led to a hyper-coagulatory state, as evidenced by the afferent findings of tissue damage associated with diffuse clotting-induced blockage of small blood vessels, what is called microthrombosis. Various types of microthrombosis are known to cause damage to the kidneys, lungs, liver, and brain [103]. While limited testing was performed[62] and a hematology consult indicated no evidence of coagulopathy, the tests performed do not preclude coagulopathy of this sort. Moreover, the pattern of end-organ injuries matches quite exceptionally to those caused by said coagulopathy [104]. While the liver and kidney damage could be attributable to both edema or microthrombosis, the pulmonary issues, low blood pressure, atypical pattern of neural damage, and pattern of diffuse damage could point more to micro-clot induced damage than edema-induced damage, but could similarly arise from both. However, edema-induced damage ***cannot*** explain the bleeding of uncertain clinical origin and the sparing of ***grey matter concurrent with white matter degradation and demyelination, which are clear indicators of microthrombosis***, and will become important in our examination of Jahi's MRI.

The Resurrection and Second Death of Jahi McMath

Jahi's mother did not accept that Jahi died, and requested that treatment be continued so that close relatives could arrive. Jahi's cardiovascular function was restored, and treatment for the diabetes insipidus, namely the reintroduction of vasopressin, was initiated. Jahi's kidneys remained insufficient and were observed without treatment. Methylprednisolone was administered in order to reduce inflammation, in order to increase viability of organs for transplantation. December 16th, the coroner requested that Jahi be removed from life support in order to facilitate autopsy. Jahi's mother found an attorney who then placed a temporary restraining order on the hospital, until the issue was resolved in court.

Another EEG was requested by the family, and on the 17th of December, it came back flat. An outside neurologist reviewed the records and imaging and confirmed brain death. On December 20th, it was noted by a nutritionist that there was no nourishment provided, save a 5% dextrose solution intermixed with saline. The same nutritionist, after discussion with the medical team, wrote that Jahi required no supportive care, and so Jahi continued to receive 5% dextrose.

The court appointed the chief of child neurology at Stanford University Medical Center as a consultant. A fourth EEG was performed before the consultant's visit on December 13th, which was again isoelectric. Blood flow scans and other imaging techniques were used and found no intracranial flow. Apnea tests were again failed, and a diagnosis of Brain Death was confirmed anew. After the visit, no more blood tests were obtained. There was no adjustment of the vasopressin nor the IV fluid. Jahi gradually deteriorated," She continued to exhibit gross anasarca. Bloody secretions began to emerge from the endotracheal tube, and tan, blood-tinged, malodorous secretions came from the mouth. After three weeks with no bowel sounds, on January 2 her first bowel movement consisted of a small amount of sloughed intestinal lining."

It was reported that the deterioration was "inevitable the moment she died," [94]. At this point it is important to note again that self-fulfilling prophesies are a thing, especially when one provides no care. These

[61] The text uses " factor VII" throughout, so I use it here. Factor VII refers to recombinant factor VIIa, which is different in that it is the activated form of factor VII which is used to induce clotting.
[62] Parameters prothrombin time, partial thromboplastin time, and international normalized ratio were noted to have been "always normal".

are especially prevalent in the neurological context, where timely, well-considered care is critical to both recovery and retention of function. The brains well documented plasticity is additional factor worthy of consideration. If the underlying system is not adequately maintained, then there will of course be very little if any recovery. It is difficult to get over a fungal infection if you also have a viral and bacterial infection. The conditions at the hospital were not adequate conditions for recovery. That will become important later.

A hospital willing to take Jahi was found, where a tracheostomy and gastrostomy would be performed. Jahi was transported there and was admitted January 6th. On January 7th, 2014, Jahi McMath was resurrected in the eyes of the law upon crossing the border into New Jersy. On January 8th, 2014, the two aforementioned procedures were performed without complications. August 25th, 2014, Jahi was discharged into her mother's care. No neurological tests took place, despite repeated demonstrations of brain function, including hormonal gland functions as demonstrated by menarche, and most importantly by repeatable motions upon command, corroborated by family and medical staff. As Jahi was in the family's care, the possibility of outside consultation came to be, and she was evaluated on three separate occasions in September 2014, April 2016, and May 2016.

As is perhaps to be expected from a brain able to respond to commands, an EEG of the brain did not demonstrate a flat line, and instead demonstrated diffuse delta-theta activity. Amplitudes increased in the final EEG, which is coherent with a continual recovery, which ought perhaps be unsurprising given her age. Heart rate showed clear increases in variability dependent on the mothers voice, even when compared to an age-matched female control voice, indicating the existence of complex auditory discrimination, and further integration with the central sympathetic and parasympathetic regulatory centers. Another test was completed at a hospital, which demonstrated less activity, taken to be potentially indicative of hypotension sensitivity, with hypotension being attributed to movement and dehydration attributable to changing venues beforehand.

An MRI was performed, which showed quite clearly grossly intact cortex, basal ganglia, thalamus, upper brainstem, and cerebellum, with signal changes suggesting extensive demyelination and cystic degeneration of the corpus callosum and bilateral centrum semiovale, and a lytic cleft in the lower brainstem. Sporadic responsivity to commands was continually documented by family through video, but did not present on an attempted reproduction date. Amantadine and Modafinil trials were unsuccessful in eliciting arousal. A chance visit had Jahi respond with what could be described as painfully deliberate motion to a request to raise her right arm, to which she responded by deliberately and slowly moving her right arm. Jahi remained stable, and would be in and out of hospitals for various treatments for gastrointestinal and hepatic issues. Jahi died of multi-organ failure and cardiac arrest a little more than four and a half years after her first determination of death via neurological criteria.

The Resurrection and Second Death of Jahi McMath: The Neurological Picture

If it were not for the scans taken months after Jahi's discharge from the hospital, the Jahi McMath case would be entirely uncontroversial. A clear case of brain death. However, on December 16th, 2013, Jahi's mother procured an attorney and the saga we are familiar with began. Recall that on December 17th, a third EEG and outside consult was requested by the family. The EEG, which was again isoelectric, was reviewed alongside the CT scan and records, and resulted in a confirmation of brain death. December 23rd, a fourth EEG was performed, *which was again isoelectric*, and a radionuclide CT blood flow scan was obtained. Both planar and single photon emission CT scans demonstrated no discernable intracranial flow and no uptake of radionucleotide tracer, and therefore no uptake of blood. Another apnea test was performed and failed. The fourth examination determined brain death yet again. Due to a lack of medical care (vasopressin and 5% dextrose IV flows and solutions remained unchanged since administration thereof) Jahi began to deteriorate, and no interventions to stabilize were taken.

On January 6th Jahi was transported to a hospital in New Jersey, where Jahi would be considered legally alive and therefore eligible for treatment. Her condition improved over her stay and the extreme edema was ameliorated. August 25th, 2014, Jahi was discharged into her mothers care at her apartment.

On September 2014, April 2016, and May 2016, neurologists selected by the Jahi family evaluated Jahi using EEG and heart rate monitors. All three EEG's were notably and quite evidently non-isotonic, with diffuse delta-theta activity invariably present, and the final EEG demonstrating substantially greater amplitudes, implying recovery. Heart rate demonstrated variability well outside the normal range in brain dead patients. While brain dead patients tend to demonstrate variability corresponding to ventilator rate, Jahi demonstrated a wide span. Most importantly, Jahi demonstrated autonomic reactivity dependent on her mother's voice, as distinguished from similar stimuli from an age-matched female control. This complex auditory discrimination and integration with central sympathetic and parasympathetic regulatory centers is, again, incompatible with brain death. A subsequent EEG on September 26th, 2014, demonstrated minimal activity, finding only "infrequent isolated frontal slowing" and being otherwise basically flat and irresponsive. This implies better and worse days, in some of which Jahi was present, and others in which she was not.

That same day, September 26th, 2014, MRA (magnetic resonance angiogram), MRV (magnetic resonance venogram), and MRI (magnetic resonance imaging) examinations were performed. Of these, the MRI is the most salient. MRI of Jahi's brain demonstrated quite evidently an intact cortex, basal ganglia, thalamus, upper brainstem, and cerebellum. Damage included extensive demyelination, cyst-producing degeneration of the corpus callosum and bilateral centrum semiovale, and open CSF-filled cavities in the lower brainstem.

Most impressively, cortical and deep-brain grey matter were spared and demonstrated no evidence of atrophy. This is considered paradoxical since hypoxia tends to kill the sets of cells requiring a lot of oxygen, which grey matter neurons are a part of. The authors of the article examining Jahi's MRI correctly note that microangiopathy and post-hypoxic effects may have been at play in the preferential destruction of white matter in the McMath case, however they do not arrive at a specific cause or kind of microangiopathy, instead indicating hypertension-induced ischemic demyelination, hypotension-related post-cardiac arrest demyelination, post-hypoxic leukoencephalopathy, or some combination of those may be causally related.

Here I propose that Jahi's seemingly paradoxical grey-matter preservation concurrent with white matter demyelination and destruction is most likely attributable to microvascular thrombosis, or microthrombosis, induced damage. The substantial use of pro-clotting factors, including tranexamic acid, platelet transfusions, and recombinant factor VIIa. The combined effect of all of these would have led to a hyper-coagulatory state leading to diffuse clot complexes to develop and subsequently clog blood vessels. As previously noted, recombinant factor VIIa [105] is exceptionally good at creating such diffuse micro-clotting[63] and similarly leads to the aforementioned white matted damage due to the low vascularization of white matter tracts. Because of the low vascularity, small clots can be introduced and not be pushed out or be overcome by increases in blood pressure [106].

Grey matter, with its higher vascularization, was able to gain the oxygen necessary to survive despite those clots due to the extance of multiple channels for blood to go through, ensuring some oxygen made its way in. The grey matter survival was likely also because of the lowered temperature and further lowered metabolic needs induced by the sedatives administered, as the authors mention [12]. This condition, of a

[63] It should be noted that recombinant factor VIIa is factor VII combined with an activating factor like thrombin, and recombinant factor VIIa is the only factor VII that is used in the clinic. In the literature, thrombin is referred to as the most potent coagulatory factor leading to coagulopathy, and in this case I have given a document which only explicitly notes thrombin's role, which may cause some confusion. As such we note that recombinant factor VIIa can be best thought of as thrombin plus an effector or a general thrombin or coagulation inducing factor, since it ultimately leads to the same effects, and is variously derived from thrombin and converted to thrombin through the famously complex clotting cascade [90].

transient global ischemic neuropathy[64], which can be defined as a transient state with massive edema, little to no discernable cranial blood flow, lack of EEG activity, low grey-white matter CT differentiation, and absent brainstem reflexes, is liable to be relatively common place in those who undergo cardiac resuscitation and subsequently exhibit massive cranial edema and coma, especially those who are determined to be dead by neurologic criteria. As we will examine in our discussion of the implication's that this case has on the current standards of brain death, this is a substantial amount of people, and there is a linear correlation between one's likelihood of presenting in this manner and youth, with perinatal patients at substantial risk of being inadequately treated and/or being incorrectly determined to be dead by neurologic criteria.

Jahi died of multi-organ failure and cardiac arrest a little more than four and a half years after her first determination of death via neurological criteria.

TJ Hoover: A Case of Institutional Failure, Clinical Inadequacy, Adherence to Routine, and Malpractice[65]

According to reports, On October 25th, 2021, Anthony Thomas Hoover II (AKA TJ Hoover) arrived and was admitted to an emergency room at Baptist Health Richmond in Richmond, Kentucky, USA, presenting with symptoms of an overdose [141], presumably attributable to opiates[66], and his family was told that he suffered from cardiac arrest. On October 26th, TJ's family was told that TJ demonstrated no reflexes, no brain activity, and no "brain waves" or EEG activity. As such, on October 27th, TJ's family decided to remove him from life support. They were informed that he was registered as an organ donor. October 28th to October 29th, TJ's organs were assessed for viability, and in the morning of the 29th, a cardiac catheterization was performed, reportedly to evaluate his cardiac viability [142]. It was reported by medical staff[67] speaking on record that the case notes for TJ noted that he had woken up during the aforementioned cardiac procedure, and was "thrashing around the table" [143].

That same day, in the afternoon, an "honor walk" or ceremonial demonstration of gratitude or honor for the organ donor, for said organ donation [144], occurred. During the ceremony, TJ opened his eyes and began visually tracking his family members. The family was allegedly told that his tracking of them was a reflexive act, which is blatantly incorrect[68]. TJ was brought into the surgical room, at which point it is reported by another medical staff member speaking on the record that TJ began moving around, "like thrashing around on the bed… and then when we went over there, you could see he had tears coming down. He was crying visibly" [143]. It was additionally reported that the patient was "shaking his head 'No'." [145]. It is further alleged that an officer of Kentucky Organ Donor Affiliates Inc., the corporation responsible for

[64] The authors call this "Global Ischemic Penumbra" since that was the term used by the neurologist who hypothesized it in 1999 [12].
[65] Allegedly. Many people are saying it. **Including me.**
[66] This is not confirmed explicitly available documentation, however considering the age of the individual, the significant presence of the opiate trade at the time and place of the events, and that the patient suffered cardiac arrest, which can manifest either separately from or as a result of opiate-induced respiratory depression, all indicate that an opiate overdose was what caused admission. Additionally, that no news source indicates that opiates were used is, due to the culture relevant to the case, also an indicator. Finally, had it been another drug type, it is not immediately evident that this particular course of events would have taken place.
[67] The staff member, Nyckoletta Martin, left Kentucky Organ Donor Affiliates (KODA) and was fired from a subsequent employer Paragonix, two days after testifying to congress on these same issues. KODA requested that Martin not be assigned to any KODA organ transplant cases, after which Paragonix fired Martin [168].
[68] There are two options in this scenario. Three if we are charitable. 1. The staff purposefully misinformed the family. 2. The staff were themselves misinformed, and in their ignorance disseminated their misinformation to the family. 3. They did not believe the family when they said that he was visually tracking people, and as such decided to interpret their perceptions as being something acceptable to them instead of denying it outright. All of these are inadequate.

organ procurement and delivery [69], pressured at least one staff member to continue with the operation, and find staff willing to do so, to the point of bringing said staff member to tears. Ultimately all staff and physicians refused to proceed, and the family was notified that TJ had woken up, wouldn't live long, and should be taken home and made comfortable.

TJ has survived as of writing, and regained the capacity to walk, dance, and speak [143]. It should be noted that Julie Bergin, the CEO of Kentucky Organ Donor Affiliates Inc., and President and COO of Network for Hope Inc, the post-merger successor of Kentucky Organ Donor Affiliates Inc and another Organ Procurement Organization, has made a statement to the effect that the TJ Hoover case had "not been accurately represented.", and the Association of Organ Procurement Organizations stated that they, "obviously want to ensure that individuals are, in fact, dead when organ donation is proceeding,." [143] Dr. Robert Truog, professor of medical ethics, anesthesia, and pediatrics at Harvard Medical School stated that, "I really would not want the public to believe that this is a serious problem. I believe that these are really one-offs that hopefully we'll be able to get to the bottom of and prevent from ever happening again…" Dr. Seth Karp, Surgeon-in-Chief at Vanderbilt University Medical Center stated, "It's not infrequent that something comes up around the donor and whether or not the donor is dead. The problem is we've had 40 years where there has been no oversight at all of OPOs. (Organ Procurement Organizations)" [142].

Malpractice

That the hospital in question does not seem to have implemented adequate measures is quite frankly unquestionable. Indeed, if they had implemented adequate measures, by definition, they would have not gone about wheeling the patient into the operating room. Moreover, they would not have initiated and completed a cardiac surgery, seemingly without any paralytics (the patient did flail around and require sedation for the surgery to be completed) had they properly ascertained that the patient was conclusively not there. Yet, I have no doubt that the physicians at the hospital completed at least some subset of the recommended minimum checks, at least some of those required by the 2023 standards we previously mentioned. Clearly not enough of these were completed, but still, I am sure that this was not a purposeful attempted slaying. I am also not certain that this was the failure of any individual staff member. To determine blame here we will have to wait for the investigations to be completed, and for ancillary factors to be elucidated, such as the role of hospital administrators and outside organizations, both those employing staff to assist in the medical aspects of organ transfer, and those responsible for the procurement of organs.

I will now review the clinical evidence and try to be charitable to the relevant parties. A patient who has presented with severe heart damage, after being brought in for what is assumed to be an opiate overdose, could certainly fail an apnea test. After all, the main cause of death in opiate overdose is respiratory depression. The additional amount of sedation and other interventions necessary to resuscitate a patient with cardiac arrest could similarly result in a case where the patient would fail some or maybe even all brainstem reflex tests. After all, opiates on their own are known to be confounders in the determination of Death by Neurologic Criteria [146].

Allegations that TJ Hoover was not yet *technically* declared brain dead are negated by the fact that there were no attempts to rescue neural function or to assess function before performing surgery on TJ's heart. Even if this is true, then we are left with a hospital which did not treat a patient during the critical moments where neural processing is salvageable, and further decided to operate on a patient without

[69] Kentucky Organ Donor Affiliates Inc. (KODA) has since merged with another organization to form "Network for Hope Inc", a Louisville KY based non-profit organization. Before the merger, KODA, itself a non-profit corporation made over 50 million dollars in 2023 as per their 2023 tax filings [169].

anesthesia or paralytics, which any neuroscientist worth their smock and overpriced pen will tell you is a faux-pas. That hospital then managed to cripple the man for life, failed to treat him neurologically ***at all***, spectacularly failed to adequately diagnose the guy, and finally managed to traumatize the medical staff is a true accomplishment.

Returning to the potential examinations, an MRI would have clearly been sufficient to prevent this from having occurred, and this particular case demonstrates with exceptional clarity the necessity for ancillary imaging in diagnosis. A TMS plus high density EEG perturbational test would be able to indicate in this case that, not only was there a significant activity (precluding prior standards of brain death), but that it was of the integrative, meaningful sort, of the kind that is regularly indicative of experience occurring at that moment [20], and as such requires that we have moral consideration for the experiencing person that is present there. At the very least, one would hope that such a demonstration would preclude starting a surgery without anesthesia, and in that manner such a tool would also be utile in the determination of where and when to administer anesthetics.

Finally. I leave you with this. Either one or the other happened. Option one, the hospital slated a man in a coma, a man understood to be alive, to have his organs removed, bringing the relevant staff together, preparing a room for surgery, and having transplant transportation staff at the ready, and then did not plan to actually go through with the organ removal, and were instead going to go about doing an extensive hour long evaluation of the patient, paying the surgeons, transplant staff, nurses, and other staff the entire time it would take the neurologist (one hopes it would be a neurologist) to complete their thorough evaluation. That is option one. Option two is that the hospital and staff slated a man who they inadequately determined to be brain dead to have his organs removed and were fully prepared to go through with it, had the patient not woken up.[70]

I present this case less as a neurological argument, and more of a demonstration of how low the standards can drop. If standards are falling through the floor, the adequate response is not to lower the standards further, especially in the medical field.

What the Jahi McMath and TJ Hoover Case Tells Us About the Current Brain Death Criteria.

> Reports have shown that cardiac arrest and resuscitation leads to death in 68% patients, with those deaths being attributable to brain injury 65% of the time [111].

It has been previously noted that, as medical interventions are introduced and improve, the methods by which we determine death must follow. It is therefore concerning that our current criteria for determining brain death, the 2023 Consensus Guidelines, eschew our best diagnostic methods for reflex-based criteria over half a century old. According to the current authoritative guidelines, death or death by neurologic criteria can be diagnosed by a lack of brain stem reflexes after a single evaluation in uncomplicated adult cases. As we will demonstrate, these criteria are faulty, regressive, and unacceptable in contemporary practice.

Hypoxic-Ischemic coma has changed thanks to the introduction of therapeutic hypothermia, which has changed the time-course of recovery and lack thereof, extending the recovery time while reducing damage and changing the damage profile in patients [112]. Severe cerebral edema is a significant clinical feature of

[70] It is important to recall that death by neurologic criteria only requires one physician's examination in adult cases, and that examination can consist of only reflex tests if the case is deemed to be without complications. To be fair, the brain is a famously simple organ, so these are most cases.

cardiac arrest, presenting itself in 22% of cases in a recent study [113]. In that cohort, only 2% of patients with severe edema survived, vs. 36% of patients with non-severe-edema. Severe edema increased with epinephrine administration, younger age, and non-cardio-pathological cardiac arrest etiology.

Withdrawal of life support treatment for neurological reasons in the same study accounted for the majority of deaths in those with central edema who died, at 57%. Withdrawal of life support treatment for non-neurological reasons was higher again for edema at 15%, vs. 5% for patients without edema. The same study demonstrated that those with edema died at a median time of 1 day, with an interquartile range of 1-2 days, where non-edema presented people died after 3 days, with an IQE of 2-6 days. In those who died from withdrawal of life supporting treatment for neurological reasons, the median time to death was 1.5 days, with an interquartile range of 1-3 days, as opposed to those without, who returned to baseline of 3 days [112].

All of the people in that study presented were patients who were resuscitated, and for those who presented with edema and were taken off of life support or diagnosed with brain death, hypoxic ischemic coma was definitionally a factor in their diagnosis and outcome. All of the edematous patients shared markers present in the Jahi case. This should not be surprising since, if Jahi had been taken off of life support during the timeframe requested by the coroner, she would have fallen into the category of people with cranial edema acquired post cardiac resuscitation. All of those people with edema would have presented with the same kind of brainstem damage, permanent or otherwise, that Jahi did, where the brainstem was pushed through the foramen magnum, causing damage to the reflexes that are now primarily used to determine brain death.

This is an issue, because at least at one hospital, people presenting with this profile are being diagnosed as being dead through neurological criteria, after 1.5 days on average, with an interquartile range of 1 to 3 days [112]. Jahi took months to recover, and this kind of inflammation can take days or weeks to resolve without aggressive treatment [71]. Moreover, that edema is common is an issue since edema is not considered a base of brain confounder for brain death diagnosis. The most current guidelines for determination of brain death do not list cranial edema as being a confounder, despite ample evidence that it can significantly confound diagnosis. As such, no testing is required to ensure that a lack of brainstem reflexes in a brain death determination is not attributable to that as opposed to whole-brain death.

We must now state again, that brainstem reflexes are the primary tests for brain death under new standards. Brain stem reflex failure is necessary ***and sufficient*** to diagnose brain death, unless there are confounding issues, ***and cranial edema is not considered a confounding issue*** [108] [109].

Children, likely due to their large brains and comparatively small skulls, are very liable to present with the transient global ischemic neuropathy sequence [116]. Interestingly, Chinese criteria for brain death are apparently more stringent than those of the United States, leading to decreased incidences of brain death and decreased eligibility for brain death determination. At this point we must recall what percentage of patients are diagnosed dead by neurologic criteria, and then are removed from life support. In a cohort of 37 patients at a large intensive care hospital in Beijing, a total of 33 patients were diagnosed as being brain dead by our criteria. Of those same 37 patients, only 9 were diagnosed as being brain dead using Chinese criteria [114][72].

[71] It is likely that TJ Hoover also went through the same post-cardiac resuscitation-induced hypoxic neuropathy on account of his absent brainstem function following cardiac resuscitation and hypoxia. However, because no testing was done, this cannot be determined to be the case.

[72] There was a notable difference in the time and money required to diagnose the patients by Chinese standards. Where our standards were regimented and simple to execute (any neurologist will be able to perform the reflex tests), theirs took significantly more time and resources. Where our standards took 52 minutes on average to execute, the Chinese standards took an average of 990 minutes to execute.

These are the statistics in the United States between January 1st, 2012, and June 30th, 2017.

"Of the 15 344 patients who died, 3170 (20.7%) were declared brain dead; 1861 of these patients (58.7%) were male, and 1401 (44.2%) were between 2 and 12 years of age… The most common causative mechanisms of brain death were hypoxic-ischemic injury owing to cardiac arrest (1672 of 3170 [52.7%]), shock and/or respiratory arrest without cardiac arrest (399 of 3170 [12.6%]), and traumatic brain injury (634 of 3170 [20.0%]). Most patients declared brain dead (681 of 807 [84.4%]) did not have preexisting neurological dysfunction…" [116]

Brainstem criteria are inadequate in meaningfully determining brain death, and the fact that they are being used to declare death by neurologic criteria, and are further being used to withhold care [108] is a failure of the highest order.

Discussion

The cases of Jahi and TJ make evident that our current standards for determining brain death are not sufficient, and that we must adopt more specific criteria and provide more resources to this end. We have noted that some clinical tools already exist and are in use, and how those, if used in these cases would have led to different outcomes for those patients. It has been noted by some that resources are finite and as such greater standards of care are not possible. It suffices to state that the use of high standards of proof in determining brain death is very possible, as evidenced by the fact that is occurs, and that general adoption of an existing medical technology does take time and effort, and excepting those objecting, expending time and effort is not the kind of thing that falls under the category of "impossible".

Returning to brain death criteria, the current framework has forced clinicians and ethicists to take some increasingly contorted views, including views that the lower brainstem is what determines life, that the capacity of a being to keep itself going (without medical intervention) is what determines life[73], and other such interestingly convoluted frameworks. The dual framework enables clinicians to more cleanly delineate life and death, and the capacity for that, and enables philosophers and ethicists to make sense of edge cases which have variously fascinated and confounded them. That the CSR framework overlaps with the clinical standard for treatment targets for disorders of consciousness like coma and vegetative state is also an important thing to note, as one can take the importance of this entire section without CSR as a driving theory, but instead taking up the medical literature and appropriate frameworks. The important thing here is that there is a need to adopt these standards as best practice, and then standard practice, in order to avoid cases like those of Jahi and TJ, and the others which haven't made the news, or were not known to be conscious or capable of instantiating consciousness.

There are many potential treatments available. In reviewing the cases of Jahi and TJ, some may wish to push forward the idea that our medical system is incapable of treating people with the standard of care which is implicitly endorsed by this examination and made necessary by the adoption of the general dual death framework. Indeed, such arguments have been made.

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[73] The one guy with the iron lung who lost the ability to breathe because of polio is in shambles, and likely doesn't know he is dead, unless of course someone sends him an email with the article. He is in for a shock, that dead guy, when he reads the article, *which is a thing he can do*

That something is expensive or currently unavailable is hardly a response for whether or not someone ought to receive it. That obligations exist that a system cannot meet is not evidence for the non-existence of the obligation, but is rather evidence of a necessity of the relevant system to improve. Hemodialysis was a terribly expensive and uncommon thing in the 1960s, and there were not enough centers nor machines to care for the sheer number of people who needed it. As such, the allocation of these was considered an "ethical dilemma", and there was always a decision to be made about whether or not someone ought to receive said treatment.

The Seattle Artificial Kidney Center quite famously centralized and formalized this practice through the Admissions and Policy Committee. Those who would benefit from hemodialysis far exceeded the number of spots, and other criteria were generated, including marital status, occupation, and perceived social worth. During that time, clinicians and ethicists pontificated about whether society could afford the treatment for everyone who required it to live, and how one ought to choose who to receive it. Out of all of these, costs, to us, are the most grotesque of justifications for the inability to provide a certain standard of care. In 1972, public law 92-603 was passed, which, among other things, expanded Medicare coverage to include hemodialysis, and nearly overnight those questions and all of the pontification and ethics around the thing were dissolved, almost as if never uttered, almost as if those people who were denied care did not die of a preventable condition, as if they could not have been saved up until the magical moment when suddenly they could. ECMO now is much like hemodialysis then, or penicillin during WWII, or ICU treatments in the 1950s, and the standard of care required in the neurological cases we are speaking about, and stating are necessary in order to evaluate true brain death is certainly similar to all of those.

Conclusion and Recommendations

Where some organizations and practitioners look upon our current paucity of resources and neurologists [125], on our deficient outcomes and see the need to reduce standards, there are intensivists, neurologists, and a myriad of other researchers and practitioners who see the opposite.

One of the primary recommendations here is one that is repeated across contexts, that being the necessity for prompt neurological evaluation for treatment, not brain death, and empirical measures, key among these EEG post-stabilization, to ascertain integrated processing, and MRI early on, to differentiate the kind of damage done. MRI prognostication is understood to provide substantial benefits over CT, especially in ascertaining white-grey matter differentiation and the preservation of structure. Additionally, patterns of damage after global brain injury are characterizable much more cleanly and reliably into pattered development sequences associated with the etiology of damage [120].

More recent clinical guidance stresses the importance of reducing secondary injury and not withdrawing care too early, as 86% of patients with TBI who die at the ICU do so due to withdrawal of life sustaining measures. Advanced EEG, MRI, and other such measures, heavily informed by neurophysiological specificities are further stressed as is precision in determination of lesions and their effects. Promising advanced therapies such cell therapy, gene therapy, and acellular scaffolds and the like are to be embraced and must be understood to be what they are, necessary [124]. Our current capacity for high resolution imaging, combined with our developing capacity to simulate and reconstruct system neural propagational dynamics from neural data [21] indicate the possibility of prognostication that is principled, empirical, and above all is amenable to the highest standards of proof for brain death.

The movement towards brainstem and other such measures is a step backwards, one that is not only incompatible with the Uniform Determination of Death Act, but is also incompatible with human progress and the spirit of advancement which has defined the neurosciences and led to our evolution from a field that

sits and diagnoses [126] to one that has seen an explosion of knowledge and ability to treat over the last 20 years. That we would stop now, not because we don't know how to improve, but because we don't feel the need or impetus to do so, would be to squander and nullify all that we have been handed, and further fail the people who depend practitioners to enjoy lives free from the pain and hardship that neurological damage can cause.

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