

CURRENT OPINION/ARGUMENTS



Truly Reconciling the Case of Jahi McMath

D. Alan Shewmon^{1,2*}

© 2018 Springer Science+Business Media, LLC, part of Springer Nature and Neurocritical Care Society

Abstract

This article clarifies some issues raised by Dr. Ariane Lewis in her recent “Current Opinion/Arguments” article on the case of Jahi McMath. Review of case materials. Jahi’s case most likely represents an instance of global ischemic penumbra (GIP) mimicking brain death (BD), with intracranial blood flow too low to support neuronal function or to be detected by radionuclide scan but sufficient to prevent widespread necrosis. Her MRI scan 9 months after the ischemic insult showed gross preservation of cortical and internal structures, incompatible with there ever having been a period of completely absent blood flow. Regarding Jahi’s alleged intermittent responsiveness, the set of videos, unsystematic as they are, constitutes convincing evidence that her movements in seeming response to command are not of spinal cord origin and are indeed voluntary responses, placing her in the category of minimally conscious state (MCS). In the absence of serial examinations by experts in MCS, the benefit of the doubt should be given. Unfortunately, her death on June 22, 2018, 4½ years after the diagnosis of BD, precludes such examinations. During those 4½ years, Jahi underwent menarche, with three documented menstrual periods, and ongoing pubertal development. Her case is an important example of false-positive diagnosis of BD, demonstrating the inability of current diagnostic standards to distinguish true BD from potentially reversible brain nonfunction due to GIP. The incidence of such mimicry is impossible to determine, because in most cases a BD diagnosis becomes a self-fulfilling prophecy.

Keywords: Brain death, Ischemic penumbra, Minimally conscious state, Ethics, Medicolegal

The recent piece in “Current Opinion/Arguments” by Dr. Ariane Lewis calls for a counterpoint [1]. To begin with, Lewis conflates the diagnosis of brain death (BD) with BD itself. Since, by definition there is no recovery from BD or death, it makes no sense to ask, even rhetorically, whether Jahi “could be the first person to recover from brain death” or whether “there is something unique about her brain that would allow it to become the only brain that could recover from death.”

The three possibilities on which Lewis’s article is based are not logically parsed. She states,

Because brain death is an irreversible coma, one of three conclusions must be drawn: (1) Jahi was never dead; (2) Jahi met the criteria for brain death, but she isn’t dead now; or (3) Jahi’s movements are not purposeful responses, and she has been brain dead since 2013.

Choices (1) and (2) are not mutually exclusive. The logical alternative to (3) is actually a merger of (1) and (2): Jahi was never brain dead; although she met the criteria for BD in 2013, she doesn’t now. In other words, her case represents a false-positive diagnosis of BD.

Jahi’s diagnosis would not be the first false positive made according to the adult or pediatric Guidelines [2, 3]. (I shall refer to the adult Practice Parameters [4] and the pediatric Guidelines [5] together as “the Guidelines,” since for Jahi’s age they are nearly identical.) After all, the Guidelines have never been validated as possessing 100%

*Correspondence: ashewmon@mednet.ucla.edu

¹ Professor Emeritus of Pediatrics and Neurology, David Geffen School of Medicine at UCLA, Los Angeles, CA, USA

Full list of author information is available at the end of the article

specificity for the Uniform Determination of Death Act's (UDDA's) criterion of "irreversible cessation of all functions of the entire brain" [6, p. 73]; they merely represent the consensus of a group of experts who uniformly did not consider that global ischemic penumbra (GIP) could in principle mimic BD in every way [7]. The Guidelines require that, prior to embarking on a BD diagnosis, confounding mimics must be excluded. That particular mimic is never excluded.

Apart from its mathematical necessity of occurrence during the progression from normal flow to no flow in the pathogenesis of BD, indirect evidence that GIP can masquerade as BD comes from several directions. The first of two major autopsy studies was the NINDS Collaborative Study, in which 60% of the 226 cases that met the study's criteria for BD did *not* have total brain necrosis [8]. Molinari considered this discrepancy "one of the major and most disturbing findings" of the Collaborative Study [9, p. 63]. More recently, Wijdicks and Pfeifer retrospectively studied 41 patients diagnosed according to the 1995 Guidelines [10]. Minimally ischemic changes were found in the cortex in 30–40% of cases, and in the deep nuclei and brainstem in around 60%. Although the authors attributed this to early fixation, Nguyen makes a convincing case that it was more likely due to ischemic penumbra [11, pp. 99–101]. Early fixation was certainly not a factor in the Collaborative Study.

Additional evidence for GIP comes from a Japanese study of serum neuron-specific enolase (NSE) in 3 BD children supported indefinitely [12]. NSE was elevated for over 8 weeks following clinical BD, in contrast to non-BD, brain-damaged controls, indicating that neuronal cell death did not occur all at once throughout the brain at the time of diagnosis—in other words, for a long time many neurons were in ischemic penumbra until they finally succumbed [13].

Low blood flow sufficient to maintain neuronal viability may not be detectable on radionuclide scans, especially in the posterior fossa, occasioning a false impression of intracranial circulatory arrest [2]. The self-fulfilling nature of a BD diagnosis (nearly universally followed by organ retrieval or termination of support) eliminates the possibility of GIP mimicry ever coming to light in the great majority of cases. This is one reason why Jahi's case is of particular importance.

Her MRI scan, performed 9 months after the ischemic insult, showed remarkable preservation of cortical and internal gross anatomy, with surprisingly little atrophy, despite cortical laminar necrosis, demyelination, and cystic encephalomalacia in the centrum semiovale, corpus callosum, and posterior pons and medulla. Selected MRI images are viewable over the internet, in my April 11, 2018 presentation at the 50th anniversary conference

on BD at Harvard Medical School (<https://www.youtube.com/watch?v=tHD00UUFiR0>, from 1:00:51 to 1:03:08), and in a recent publication by Machado et al. [14]. The lower brainstem lesion explains her apnea and most of the brainstem areflexia, but the relative intactness of the upper brainstem, thalamus and cortex could possibly serve as a structural basis for intermittent consciousness. Such preservation implies that there was never a period of truly absent intracranial blood flow. Low flow, below the detection threshold of the 9/26/14 magnetic resonance angiogram and the 12/23/2013 radionuclide scan but sufficient to support neuronal viability, must have been present. The preserved structure means that in principle there could be potential for function, within the limitations of the severe disability caused by the damaged areas.

This brings us to the issue of Jahi's alleged intermittent responsiveness, which, if true, would place her in the category of minimally conscious state (MCS) [15]. Lewis proposes that

acceptance that Jahi is not brain dead... would require the brain death examinations be performed by prominent, trusted figures, such as Dr. Thomas Nakagawa and Dr. Stephen Ashwal, who wrote the 2011 Guidelines. (emphasis added)

That assertion makes no sense on a number of counts.

First, as a matter of logic, "acceptance that Jahi is *not* brain dead" would require examinations designed to diagnose MCS, not examinations designed to diagnose BD.

Second, the detail immediately following that block quote, that "apnea testing would need to be done in a hospital," makes no sense, because (1) her mother would (rightfully) never consent to either the transfer or the test, on account of their inherent risks, and (2) her lawyer is prepared to stipulate that Jahi would show no respiratory effort on a formal apnea test (personal communication, Bruce Brusavich).

Third, the proposal further makes no sense, because I already did a BD examination in Jahi's apartment on December 2, 2014 (minus a formal apnea test), and found unresponsiveness and brainstem areflexia. She was briefly taken off the ventilator to see whether any respiratory effort might occur that would contradict a BD diagnosis, but none did. (After about 20s, she began to desaturate and was immediately reconnected.) Does Dr. Lewis really expect Dr. Nakagawa or Ashwal to find something different?

Finally and most importantly, as emphasized by Giacino, serial assessment, not a single examination, is required for diagnosing MCS [15, p. 351, 16, p. 296]. That is precisely what the videos provide. Lewis states that

“video analysis is not scientifically rigorous and is neither accepted for determination of a minimally conscious state or brain death.” Video analysis for determining BD is not at issue. It goes without saying that the videos made by Jahi’s family are not scientifically rigorous. They have, however, been certified by a forensic video expert as unaltered since their original recording.

Obviously, in-person observation by experts in MCS would be far preferable to reliance on those videos. The practicality of that depends on the frequency and duration of the periods of claimed responsiveness. According to Jahi’s mother, they occur around three times per week on average, lasting for around half an hour at a time. To confirm or disprove that assertion in person would require a team of MCS experts to spend up to a week in Jahi’s apartment, taking shifts in order not to miss anything. This is clearly never going to happen, so the video evidence is the next best thing, despite its intrinsic limitations. Since a girl’s life versus death status hangs in the balance, we should take the videos very seriously, learn from them what we can, and not dismiss them out of hand merely on the grounds that they are not methodologically ideal.

I have spent countless hours studying the entire collection, playing the devil’s advocate at each step. From a total of 60 videos received, 48 were suitable for command–response analysis. Eleven representative videos were presented at the Harvard conference and are available for viewing over the internet (<https://www.youtube.com/watch?v=tHD0OUUfiR0>, from 00:46:32 to 00:55:43). Three of the 48 contained a discontinuity, due to pausing and restarting the recording; each discontinuity was treated as having split the video into two distinct videos, resulting in 51 videos for purposes of analysis. The sound tracks were transcribed, and every command and movement was timed at frame precision. Myoclonic jerks (mostly of thumbs and fingers), operationally defined as movements with a first-phase duration of < 400 ms, were excluded from consideration. The entire data set contained 189 commands to move various body parts and around 100 non-myoclonic movements (the exact number depending on various definitions and assumptions). Each body part with enough non-myoclonic movements to determine a temporal distribution displayed an approximately exponential distribution of inter-movement intervals, facilitating statistical comparison of movement frequency during command versus baseline conditions. Likewise, the approximately exponential distribution permitted a comparison of latency from command to next movement of the commanded part with expected latency given baseline frequency. A third kind of analysis, not dependent on temporal distribution, involved Chi-squared or Fisher’s exact tests on

contingency tables of commanded body part versus next-moved body part.

Further details of the statistical analyses would far exceed the scope of this paper and are the subject of a manuscript in preparation. Suffice it to say that I am convinced that the apparent responses to command cannot all be explained away as mere chance occurrences of spontaneous spinal movements. Reasons for this conclusion are multiple. *First*, although the videos do demonstrate spinal myoclonus (mainly of the fingers), the movements at issue are slower, and some are anatomically and/or temporally complex. More importantly, they are not any of the types of spontaneous movements known to be generated by the autonomous cord in patients with high spinal cord injury. *Second*, the movements occur much more frequently during periods of command and coaxing than at baseline. Body parts with sufficient numbers to power a comparison yielded high significance. *Third*, the latency from command to next movement of the requested part is significantly shorter than would be expected by chance. *Fourth*, following a command, the next non-myoclonic movement is of the requested body part much more often than could be explained by chance (with very high statistical significance, even after including imputed baseline movements of off-camera body parts, which the family states did not occur). *Fifth*, a study of Jahi’s heart rate variability by Machado and colleagues showed responsiveness to mother’s voice, reinforcing the video evidence for motoric responsiveness [14].

One might object that the family could have taken many videos and submitted only those that happened to support their claim. Several considerations argue against that. *First*, they had no idea a priori that any sort of analysis would be made of the videos, much less what types of analysis. *Second*, they did in fact submit videos containing no response and others containing movements of “wrong” body parts. *Third*, given the baseline frequency of movement of each body part, to obtain the submitted videos by chance would have required making many hundreds or even thousands of videos (depending on body part and type of analysis) and withholding the vast majority—an extremely implausible scenario, given both the sheer impracticality and the fact that it would have involved massive fraud, of which no one who has met the family can reasonably accuse them.

Thus, Lewis’s proposal of an independent evaluation by BD experts would fail to address the real issue, namely whether Jahi is in MCS, as the videos strongly suggest. A one-time finding of coma, brainstem areflexia and apnea would not disprove the claim of intermittent responsiveness.

Lewis expressed reservations regarding my objectivity in analyzing the videos. She stated that,

Because of [my “philosophical beliefs”] it is worth noting that even though Dr. Shewmon said Jahi met the criteria for brain death when he examined her in 2014, he did not accept that she was dead at that time.

This is a mischaracterization of my position in general and regarding Jahi in particular. *First*, my published critiques of the rationale for equating BD with death are based not on “philosophical beliefs” but on logic applied to medical facts [17]. Dr. Lewis is welcome to present counterarguments more convincing than listing specialty societies that endorsed the Guidelines. She conveniently omitted mention of the many other experts who have likewise criticized the concept of BD; I am hardly a lone outlier in that regard. *Second*, my conclusion in 2014 that Jahi was neither dead nor BD was based primarily on the video evidence of intermittent responsiveness, not on “philosophical beliefs” about BD. *Third*, my conclusion was reinforced by the fact that Jahi had had three menstrual periods that year (documented in hospital and home nursing records) and displayed ongoing pubertal development, with breast enlargement and all the secondary sexual characteristics (documented photographically and personally by her primary physician, Dr. Alieta Eck, and myself)—a phenomenon (a) that the Guidelines do not address, (b) that evinces hypothalamic functioning relevant to the “organism as a whole” and therefore contrary to the UDDA, and (c) that one does not need a medical degree to know that corpses do not manifest.

To the contrary, when I became involved in Jahi’s case in the spring of 2014, it was on the assumptions that she *was* brain dead and that the few videos of alleged responses posted by the family on YouTube showed only spinal movements. Jahi was defying all the predictions of imminent cardiovascular collapse based on multisystem deterioration (which in retrospect was due to lack of nutrition for 3 weeks and untreated hypothyroidism and hypoadrenalism); she had not merely survived for several months, but had actually improved in overall health. I thought she would fit perfectly in the series of chronic BD cases that I have continued to collect since my 1998 article on the subject [18]. Over the rest of 2014, however, more and more videos were submitted by the family—and much more impressive ones—convincing me that she was actually in MCS and did not belong in my chronic BD series after all.

Regarding Lewis’s proposed alternative to my alleged lack of objectivity, does she really think that two neurologists “who wrote the 2011 Guidelines” would be free of desire to vindicate those Guidelines? Or that one of them (Nakagawa), who was paid a hefty sum by defense lawyers for his declaration that Jahi was dead (personal

communication, Bruce Brusavich) [19], would be less biased than me, who have not been retained by any lawyer or paid anything for my work on the case, and who was open-minded enough to change my opinion about her BD status based on evidence? No one is entirely free of bias, myself included, and independent evaluation by experts in MCS who have no stake in either the validity of the Guidelines or the outcome of Jahi’s medicolegal case would certainly be welcome.

Lewis condescendingly dismisses my assertions that Jahi does not fulfill the California statutory definition of death and that the Guidelines do not comport with the UDDA. Apart from the specifics of Jahi’s case, the Guidelines explicitly allow for some retained brain functions (including some that would qualify as “critical” according to Bernat’s distinction [20], or as “clinical” according to the insistence of Bernat, Wijdicks and others [20, 21]) [3, 11, 22, 23]. Therefore, the Guidelines, by their own wording, do not identify the “irreversible cessation of all functions of the entire brain” required by the UDDA [6, p. 73], despite having become, through political decree within professional societies, the “accepted medical standards” that the UDDA defers to. The intrinsic potential for contradiction between those two parts of the UDDA, a potential that remained latent for over three decades, has now become a glaring reality, unforeseeable by its drafters in 1981. The new Nevada law, which specifies the Guidelines as the only acceptable means for diagnosing BD, only further confuses the already confused medicolegal scene surrounding BD [24, 25].

Jahi’s case fails to correspond not only to the wording of the UDDA but also to its spirit, insofar as the reason its drafters considered BD to instantiate the concept of death was that they considered the brain to be the master coordinator of the body, unifying the “organism as a whole.” The main evidence for that belief was that, in the absence of brain function, “Even with extraordinary medical care, these [vital] functions cannot be sustained indefinitely—typically, *no longer than several days.*” [6, p. 35] (emphasis added) I seriously doubt that the drafters of the UDDA would have considered a ventilator-dependent patient who overcame initial multisystem failure and survived in good health for over 4 years, not in an ICU but in an apartment, and who began menarche and underwent pubertal development, not to be an “organism as a whole” and to be dead.

For the record, there is no contradiction between my and Dr. Machado’s statements about “the” EEG, because there were four EEGs. I was referring to the report of the one at Rutgers University Hospital on September 26, 2014, while Jahi was probably hypotensive after transport. Dr. Machado was referring to three EEGs done

portably in her apartment between September, 2014 and May, 2016 [14]. Intermittency of electrocortical activity would concord with intermittency of responsiveness.

Conclusions

The true reconciliation between the diagnosis of BD in 2013 and Jahi's development of intermittent responsiveness in 2014 is the following. At the time of diagnosis, Jahi was in GIP, which mimicked BD; brain blood flow was below the detection threshold of the radionuclide scan but sufficient to prevent widespread necrosis. Months later, with resolution of cerebral edema, cardiovascular stabilization, provision of nutrition, and treatment of hypothyroidism and hypoadrenalism, intermittent increases in cerebral blood flow above the penumbra range permitted cerebral function to return intermittently, manifested by responsiveness to commands. There is no other possible explanation, except to dogmatically disregard the gross structural preservation on MRI and the behavioral and heart rate variability evidence for responsiveness.

There is no way to know what proportion of patients diagnosed BD might actually have been in GIP, nor is there any way to know the spectrum of degrees of potential recovery if maximum neuroprotection, treatment of endocrine dysfunction, adequate nutrition, and intensive care were provided despite apparent BD, and maintained indefinitely as is sometimes still done in Japan. Many such patients would no doubt eventually go on to cardiac arrest regardless of therapeutic interventions, particularly older adults and those with multisystem damage to begin with (e.g., from cardiorespiratory arrest, near drowning, massive trauma, etc.) [18]. That, of course, would not prove that they were already dead, but only that they were moribund. More importantly, younger patients and those with primary brain pathology (e.g., intracranial hemorrhage, and gunshot wound to the head) have statistically longer survival potential [18], and a subset of them are probably in GIP, with the potential to improve to a level of MCS like Jahi, or possibly even higher. We may never know, because of the rarity of motivation to support such patients indefinitely and the multiple powerful factors in western society militating against it.

The physicians who diagnosed Jahi as BD in 2013 indeed followed "accepted medical standards," but those standards themselves do not heed the Guidelines' own first requirement, namely to rule out confounding factors. Patients suspected of becoming or being BD are generally not tested for thyroid or adrenal function, nor are they treated with hormone supplementation, even though the adult Guidelines explicitly mention endocrine

dysfunction as a potentially confounding factor to be excluded. (After a diagnosis of brain death, however, organ donors are often treated with hormone supplementation in order to maximize organ viability.)

Likewise, the "accepted medical standards" do not include ruling out GIP as a confounding factor. The Guidelines make no mention of it, and there is no way to rule it out in a given case short of actual measurement of blood flow in every part of the brain, for which no practical test exists (an area ripe for urgent clinical research). Thus, the Guidelines are intrinsically self-contradictory, because their diagnostic algorithm fails to exclude the most deceptive BD mimic of all. That it has taken so long for this to come to light is attributable to the self-fulfilling nature of the BD diagnosis. That is why Jahi's case is so important.

Regarding the maintenance of public confidence in the diagnosis of BD for the sake of transplantation [26], that goal should be accomplished by making the diagnosis worthy of confidence. Counterexamples to the claimed infallibility of the Guidelines should be taken seriously and learned from.

Postscript

Jahi passed away on June 22, 2018, rendering moot all debate over which independent experts should examine her and which diagnostic protocols should be employed. At the times of my court declaration and the Harvard conference, I had not personally observed her responsiveness. However, 6 days before she died, I visited her in her hospital room and observed a (non-myoclonic) right arm movement in response to her mother's command to move that arm. (There had been no spontaneous movements of any kind up that point or for the rest of my visit, so it was clearly not a chance coincidence of a random baseline movement.) Her mother also showed me two recent brief videos on her cell phone, demonstrating convincing responses to command.

Jahi's New Jersey death certificate is dated June 22, 2018. Prior to that, both Jahi and her brain were alive. Corpses cannot die. The recent newspaper headlines got it right: "Jahi McMath, whose 2013 declaration of brain death sparked a legal fight to keep her on life support, dies" [27].

Author details

¹ Professor Emeritus of Pediatrics and Neurology, David Geffen School of Medicine at UCLA, Los Angeles, CA, USA. ² Pediatrics Department, Olive View-UCLA Medical Center, 14445 Olive View Drive, Sylmar, CA 91342-1495, USA.

Source of Support

No funding.

Compliance with Ethical Standards

Conflict of interest

The author declares that he has no conflict of interest.

Published online: 15 August 2018

References

- Lewis A. Reconciling the case of Jahi McMath. *Neurocrit Care*. 2018. <https://doi.org/10.1007/s12028-018-0561-5>.
- Shewmon DA. False-positive diagnosis of brain death following the Pediatric Guidelines: case report and discussion. *J Child Neurol*. 2017;32:1104–17.
- Dalle Ave AL, Bernat JL. Inconsistencies between the criterion and tests for brain death. *J Intensive Care Med*. 2018. <https://doi.org/10.1177/0885066618784268>.
- Wijdicks EFM, Varelas PN, Gronseth GS, Greer DM. Evidence-based guideline update: determining brain death in adults: report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology*. 2010;74:1911–8.
- Nakagawa TA, Ashwal S, Mathur M, Mysore M, Committee for Determination of Brain Death in Infants and Children. Guidelines for the determination of brain death in infants and children: an update of the 1987 Task Force recommendations—executive summary. *Ann Neurol*. 1987;2012(71):573–85.
- President's Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research. *Defining death: medical, legal, and ethical issues in the determination of death*. Washington: U.S. Government Printing Office; 1981.
- Coimbra CG. Implications of ischemic penumbra for the diagnosis of brain death. *Braz J Med Biol Res*. 1999;32:1479–87.
- Walker AE, Diamond EL, Moseley J. The neuropathological findings in irreversible coma: a critique of the "respirator brain". *J Neuropathol Exp Neurol*. 1975;34:295–323.
- Molinari GF. Review of clinical criteria of brain death. *Ann N Y Acad Sci*. 1978;315:62–9.
- Wijdicks EF, Pfeifer EA. Neuropathology of brain death in the modern transplant era. *Neurology*. 2008;70:1234–7.
- Nguyen D. The new definitions of death for organ donation. *Bern: Peter Lang*; 2018.
- Suzuki Y, Mogami Y, Toribe Y, et al. Prolonged elevation of serum neuron-specific enolase in children after clinical diagnosis of brain death. *J Child Neurol*. 2012;27:7–10.
- Shewmon DA. Brain death or brain dying? [editorial]. *J Child Neurol*. 2012;27:4–6.
- Machado C, DeFina PA, Estévez M, et al. A reason for care in the clinical evaluation of function on the spectrum of consciousness. *Funct Neurol Rehabil Ergon*. 2018;7:43–53.
- Giacino JT, Ashwal S, Childs N, et al. The minimally conscious state. Definition and diagnostic criteria. *Neurology*. 2002;58:349–53.
- Giacino JT. The vegetative and minimally conscious states: consensus-based criteria for establishing diagnosis and prognosis. *NeuroRehabilitation*. 2004;19:293–8.
- Shewmon DA. The brain and somatic integration: insights into the standard biological rationale for equating "brain death" with death. *J Med Philos*. 2001;26:457–78.
- Shewmon DA. Chronic "brain death": meta-analysis and conceptual consequences. *Neurology*. 1998;51:1538–45.
- Nakagawa TA. Declaration of Thomas A. Nakagawa, M.D., FAAP, FCCM in support of defendants' motion for summary adjudication of plaintiff Jahi McMath's first cause of action for personal injuries. Superior Court of California, Alameda County, R-1838158; 2017.
- Bernat JL. Refinements in the definition and criterion of death. In: Youngner SJ, Arnold RM, Schapiro R, editors. *The definition of death: contemporary controversies*. Baltimore: Johns Hopkins University Press; 1999. p. 83–92.
- Wijdicks EF. The case against confirmatory tests for determining brain death in adults. *Neurology*. 2010;75:77–83.
- Nair-Collins M, Northrup J, Olcese J. Hypothalamic-pituitary function in brain death: a review. *J Intensive Care Med*. 2016;31:41–50.
- Miller FG, Truog RD. *Death, dying, and organ transplantation. Reconstructing medical ethics at the end of life*. New York: Oxford University Press; 2012.
- Pope TM. Brain death forsaken: growing conflict and new legal challenges. *J Leg Med*. 2017;37:265–324.
- Yanke G, Rady MY, Verheijde JL. Ethical and legal concerns with Nevada's brain death amendments. *J Bioeth Inq*. 2018;15:193–198.
- Lewis A, Bernat JL, Blosser S, et al. An interdisciplinary response to contemporary concerns about brain death determination. *Neurology*. 2018;90:423–6.
- Tchekmedyan A. 'A girl with a brain injury.' Jahi McMath, whose 2013 declaration of brain death sparked a legal fight to keep her on life support, dies. *Los Angeles Times*, 2018; B1–B2.