



20294053

1 **AGNEW & BRUSAVICH**
 Bruce M. Brusavich, Esq. (SBN 93578)
 2 Terry S. Schneier, Esq. (SBN 118322)
 Alexander B. Boris, Esq. (SBN 313195)
 3 20355 Hawthorne Blvd., 2nd Floor
 Torrance, California 90503
 4 Telephone: (310) 793-1400

5 **ESNER, CHANG & BOYER**
 Andrew N. Chang, Esq. (SBN 84544)
 6 234 East Colorado Boulevard, Suite 750
 Pasadena, California 91101
 7 Telephone: (626) 535-9860
 8 Attorneys for Plaintiffs

FILED
 ALAMEDA COUNTY
 DEC 22 2017
 CLERK OF THE SUPERIOR COURT
 By *[Signature]* Deputy

10 SUPERIOR COURT OF THE STATE OF CALIFORNIA
 11 FOR THE COUNTY OF ALAMEDA

12 LATASHA NAILAH SPEARS WINKFIELD;
 13 MARVIN WINKFIELD; SANDRA
 CHATMAN; and JAHl McMATH, a minor,
 14 by and through her Guardian ad Litem,
 15 LATASHA NAILAH SPEARS
 WINKFIELD,,
 16 Plaintiffs,

Case No. RG15760730
 Assigned for All Purposes to the
 Hon. Stephen Pulido, Dept. 517

DECLARATION OF D. ALAN SHEWMON, M.D.

17 v.
 18 FREDERICK S. ROSEN, M.D.; UCSF
 BENIOFF CHILDREN'S HOSPITAL
 19 OAKLAND (formerly Children's Hospital &
 Research Center at Oakland); MILTON
 20 McMATH, a nominal defendant, and DOES 1
 THROUGH 100,
 21 Defendants.

[Filed Concurrently with Plaintiffs' Motion to
 Bifurcate the Issue of Whether AAN and AAP
 Guidelines Meet the Statutory Definition of
 "Dead" Under the Uniform Determination of
 Death Act]

DATE: March 8, 2018
 TIME: 3:00 p.m.
 DEPT: "517"

Complaint Filed: November 1, 2013

1 **DECLARATION OF D. ALAN SHEWMON, M.D.**

2 **I, D. Alan Shewmon, declare:**

3 1. I have been an academic pediatric neurologist since 1981 and am currently
4 Professor Emeritus of Pediatrics and Neurology at the David Geffen School of Medicine at
5 UCLA. My professional training includes a bachelor's degree from Harvard College in 1971, a
6 medical degree from New York University Medical School in 1975, two years of pediatric
7 residency at San Francisco Children's Hospital (now California Pacific Medical Center), three
8 years of neurology residency at Loyola University Chicago Stritch School of Medicine, and one
9 year of fellowship at UCLA in developmental disabilities and mental retardation. I am triply
10 board certified: in Pediatrics, Neurology (with special competence in Child Neurology), and
11 Clinical Neurophysiology. From 2003 to 2014 I was Chief of Neurology at Olive View-UCLA
12 Medical Center, a county hospital affiliated with UCLA, and Vice-Chair of the Neurology
13 Department at UCLA. Since retiring from county employment in 2014, I have remained
14 clinically active, maintaining my clinic at Olive View-UCLA and consulting for five other
15 hospitals in the Los Angeles area. I am a member in good standing of the American Academy of
16 Neurology, the Child Neurology Society, the American Epilepsy Society, and other professional
17 organizations detailed in my CV.

18 2. I have never charged for nor have I received any financial compensation from
19 Jahi McMath's family or from their lawyers for my professional time reviewing the
20 documentation and providing my expert opinions in this case. I have volunteered my time and
21 effort out of a combination of humanitarian, ethical, academic and research interests. This
22 declaration supplements my declaration dated December 10, 2014 filed in this case.

23 3. My two main areas of special expertise have been pediatric epilepsy and the
24 interface between neurology and bioethics, particularly brain death and vegetative state. A rough
25 estimate of the total number of brain death cases I have diagnosed in the course of my career,
26 according to accepted medical standards, is probably between 150 and 200. My philosophical
27 opinion about the concept of brain death (*vide infra*) has no impact on how I go about diagnosing
28 brain death in day-to-day clinical situations.

1 4. My expertise in brain death is internationally recognized. Related to that specific
2 topic alone, my CV lists 13 peer reviewed publications, 2 invited reviews, 1 book, 12 chapters,
3 36 invited lectures at the international level and 20 at the national level. Three of the peer-
4 reviewed publications were given pride of place in their respective journal issues. One was the
5 lead article in a major biostatistics journal.¹ Another was a feature article in the official journal
6 of the American Academy of Neurology,² accompanied by an invited editorial³ and selected for
7 mention in the "Highlights" section. The entire October 2001 issue of *Journal of Medicine and*
8 *Philosophy* was dedicated to commentaries on my lead article, none of which disputed my
9 arguments and conclusion about brain death with respect to the biological organism as a whole.⁴

10 5. In the mid-1980s I was a member of the Child Neurology Society's Ethics
11 Committee, when it was entrusted with the task of drafting the first diagnostic guidelines for
12 brain death in children (predecessor of the 1987 Task Force guidelines⁵). I was a consultant for
13 two Working Groups of the Pontifical Academy of Sciences on the determination of death in
14 1989⁶ and 2006⁷, and a member of the Task Force on Brain Death of the Pontifical Academy
15 for Life (1997-98). I was on the Organizing and Scientific Committees for the 3rd and 4th
16 International Symposia on Coma and Death in Havana (2000 and 2004), and together with the
17 conference organizer I was co-editor of the book "Brain Death and Disorders of Consciousness."
18 ⁸ In 2007 I was a consultant to the President's Council on Bioethics during the drafting phase of
19 their White Paper on brain death.⁹ In 2012 I was a consultant to the German Ethics Council in its
20 deliberations on brain death.

21 **A. Jahi McMath does not currently fulfill the accepted medical standards for**
22 **brain death.**

23 6. There is no question that in December 2013 at Oakland Children's Hospital, Jahi
24 McMath fulfilled the widely accepted pediatric guidelines for determining brain death
25 (hereinafter referred to simply as the Guidelines),¹⁰⁻¹² as well as the adult guidelines,¹³ both
26 regarded as the accepted medical standards. There is equally no question in my mind that she no
27 longer does, for the single reason that the first of the "three cardinal findings in brain death"¹⁴ –
28

1 coma, absence of brainstem reflexes, and apnea—is not fulfilled. Rather, she is intermittently
2 responsive, placing her in the category of “minimally conscious state.”^{15, 16}

3 7. The change took place around the spring of 2014, when Jahi’s family members
4 began to suspect that she sometimes seemed to respond to command. When I first heard of this
5 through the news media, I was as skeptical as everyone else, assuming that they were mistaking
6 spinal reflexes or myoclonus (involuntary quick jerks) for voluntary movements. Because of my
7 research interest in the phenomenon of chronic brain death, I contacted Jahi’s family through her
8 attorney, Christopher Dolan, and developed a rapport with them.

9 8. Realizing that no one was likely to believe them about Jahi’s intermittent
10 responsiveness, the family began making video recordings of what they believed to be motor
11 responses to simple commands. They gradually formed the impression that Jahi’s responsiveness
12 tended to occur when her heart rate was above 80 beats per minute, and hardly ever when it was
13 slower—suggesting the possibility of some sort of inner state differentiation, with responsiveness
14 more likely during the more aroused state. Therefore, they tended to wait for occasions when her
15 heart rate was over 80 to record command-response sessions.

16 9. The intermittency of the alleged responsiveness—as infrequent as weekly or less,
17 sometimes more—creates a particular challenge to either disprove or verify, because the
18 likelihood of Jahi being in a “responsive” state during a random examination is small. In fact,
19 when I had the opportunity to examine her in person on December 2, 2014, it was one of her less
20 “aroused” days, and she did not respond to command in my presence. (Neither did she exhibit
21 any cranial nerve reflexes or breathe spontaneously over the ventilator—all consistent *at that*
22 *moment* with continued fulfillment of the brain death Guidelines.)

23 10. This is why the video recordings, as crude and unsystematic as they are, represent
24 the only way at present to decide whether Jahi is permanently comatose or in a minimally
25 conscious state with intermittent responsiveness. During the time period from March 2014
26 through April 2016, Jahi’s family entrusted me with a total of 49 distinct digital video files (not
27 counting several duplicates with different file names), believed to constitute the entire collection
28 of existing command-response videos. These have all been made available to the court and to the

1 expert consultants for the defense, who both cite them as among the material received
2 [Nakagawa, p. 12; Schneider, p. 8] but make no other mention of them in their respective
3 declarations. Every video file has been subjected to expert forensic video analysis and certified
4 to contain no evidence of post-recording alteration.

5 11. File durations ranged from 13 to 732 seconds, with a median of 70 seconds. The
6 videos contain 193 commands and 668 elementary movements (counting individual components
7 of compound movements). Some movements, especially of the fingers, have the quality of
8 myoclonus (quick involuntary jerks, almost certainly originating in the spinal cord). Judging
9 from the sound track, most of the finger myoclonias were considered by family to be involuntary
10 and of no interest. The movements that they interpreted as responses to command were for the
11 most part slower, with durations ranging from around half a second to a few seconds for simple
12 movements and over 10 seconds for more complex movements.

13 12. *Most of the non-myoclonic movements bear no resemblance to any kind of reflex*
14 *or spontaneous spinal cord-generated movements ever reported to occur in spinal cord injury*
15 *patients below the level of the lesion.* The repertoire of endogenous spinal cord-generated
16 spontaneous movements (after resolution of spinal shock) includes: myoclonus (a brief, single
17 twitch), clonus (a rhythmically repetitive, hyperactive muscle stretch reflex), muscle spasms
18 including massive body spasms (often provoked by internal noxious stimuli such as constipation
19 or a full bladder), alternating flexion-extension leg movements, periodic limb movements and
20 restless legs syndrome.¹⁷⁻³⁰ The autonomous cord is not known among neurorehabilitation
21 experts to generate any other type of spontaneous (or apparently spontaneous) movement. Jahl
22 has manifested myoclonus, clonus, and massive spasms at various times, but only myoclonus
23 (almost entirely of the fingers) and clonus occurred during the videos.

24 13. An obvious objection is that these videos could have been cherry-picked from a
25 much larger set of recordings, and only the ones that supported the family's claim were released.
26 They did, in fact, discard a number of videos in which no post-command movements occurred,
27 until I asked them to keep and send everything. The set of 49 video files contains 5 with no
28

1 movements at all and a total of 38 commands followed by no movement of the requested body
2 part.

3 14. There are no videos of pure baseline without any command, at times when family
4 might have suspected responsiveness (on the basis of heart rate over 80) and could have
5 attempted a command-response trial but did not for the sake of establishing a baseline. If all of
6 Jahi's movements were of endogenous spinal origin and the "responses" were mere temporal
7 coincidences relative to commands, it is reasonable to assume that each body part had a
8 characteristic average rate on days when family suspected her to be most likely "responsive"
9 (heart rate above 80) and made a video (and a lower rate on days when they considered her
10 unresponsive and didn't bother). Therefore, a reasonable estimate of baseline non-myoclonic
11 movement frequency for each body part can be inferred from the periods when that particular
12 body part was not the subject of a command, averaged across all videos.

13 15. It would be completely implausible if, on a given day frequent endogenous
14 movements occurred in only the left arm, for example, while the other body parts had only rare
15 movements, so the family decided to make a video demonstrating "responsiveness" to left arm
16 commands; and then on another day only the right foot had frequent endogenous movements
17 while all other body parts had rare movements, so they decided to make a video on that day
18 demonstrating "responsiveness" to right foot commands; etc. On days with heart rate above 80,
19 when non-myoclonic movements are more likely to occur, it is much more plausible that the
20 average rate for each body part would be relatively homogenous from day to day, so that the
21 average across the whole set of videos during non-command periods should be a reasonable
22 approximation of the baseline movement frequency for each body part.

23 16. Careful examination of the video data leads to the following conclusions about the
24 non-myoclonic movements.

25 17. (1) The baseline frequency of non-myoclonic movements in a given body part is
26 very low, whereas it is much higher during periods of request for movement of that body part. It
27 is therefore extraordinarily unlikely that the movements during command times arose from the
28 same process as the baseline movements. As a related observation, movements occur much

1 sooner after commands than would be expected on the basis of random occurrence at baseline
2 frequency.

3 18. (2) There is a very strong correspondence between the body part requested and
4 the next body part that moves. This cannot reasonably be explained by chance.

5 19. (3) There is a very strong correspondence between the laterality of the body part
6 requested and the laterality of the next body part that moves. With thumb or finger commands,
7 the camera was usually focused close-up on the expected hand. Therefore, this laterality effect is
8 best demonstrated with those commands where both right and left sides were in camera range
9 simultaneously for the body part commanded.

10 20. (4) Some videos show qualitative aspects indicative of more complex
11 comprehension and volition.

12 21. For example, in "VIDEO0112.mp4," made on 3/17/14, Jahi's mother asks her to
13 move her right hand, and 6 seconds later the right arm extends at the elbow, passively moving
14 the right hand along with the forearm (total movement duration 4 seconds). Then mother asks her
15 to move the left hand, and 12 seconds later there is a pair of slight lateral twitches of the left
16 forearm (they resemble myoclonus, but similar movements of the left forearm never occurred
17 during a total of 37 minutes of baseline time when no arm was commanded). Then mother asks
18 her to move the left hand *harder*, and immediately there is another pair of lateral twitches of the
19 left forearm, *stronger than before*.

20 22. In "jahi thumbs up.3gp," made on 10/30/2014, Jahi's aunt asks her to put her
21 thumb up; 10 seconds later there is a slight myoclonic jerk of the left third finger and a pair of
22 slight myoclonic flexion jerks of the left thumb. Her aunt tries to encourage her by saying, "I see
23 you moving. Try to put it up," and a second later the left thumb makes a non-myoclonic (total
24 duration 1 second) flexion movement, with simultaneous slight pronation of the left forearm and
25 slight movement of the second finger toward thumb. The aunt says, "I see you trying, honey.
26 You just moved your thumb. Can you put it *up*?" With a bit of further coaxing, 14 seconds later
27 the left thumb extends *upward* with a non-myoclonic movement.

28

1 23. In "Jahi relax hand.mp4," filmed on 1/13/2015, Jahi's aunt had been asking her to
2 move her thumb prior to the start of the video (by implication from the sound track, the first
3 words of which were "I see you movin' it, Jahi. Could you put your thumb all the way up?") At
4 14 seconds into the video, between the words "thumb" and "all" of the repeat command, there is
5 a large, slow flexion/opposition movement of the right thumb while the second and third fingers
6 flex at the metacarpophalangeal joints. After praising Jahi, the aunt says *sotto voce* to someone
7 else in the room at 27 seconds: "She's not relaxing her hand; she's still trying." Jahi's fingers and
8 hand muscles are visibly tense on the video. Then the aunt says to Jahi: "*Relax, girlie. Relax your*
9 *fingers, Jahi.*" Four seconds after the first "Relax," the hand and fingers begin to visibly *relax*,
10 gradually returning to their pre-movement position over the next 2 seconds.

11 24. In "20160224_the bad finger lol.mp4," Jahi's mother asks her to move her third
12 finger, but without using the phrase "middle finger." Rather, the requests are made in terms of
13 circumlocutions, such as: "Which finger is the 'bad' finger?" "Which finger would I move, if'n I
14 get mad at somebody?" "Which finger is the 'f- you' finger?" "So when you get mad at
15 somebody, which finger you 'posed to move?" Two seconds after the first question, the left
16 middle finger flexes (non-myoclonic). One second after the second question, the left middle
17 finger flexes with a velocity making it arguably a myoclonic jerk. Two seconds after the third
18 question, the left middle finger does likewise again . Two seconds after the fourth question, the
19 fifth finger makes a small myoclonic jerk. Mother says, "Not *that* one," and 4 seconds later the
20 *third* finger makes a large, slow flexion (definitely not myoclonic). Even if the second and third
21 trials are excluded as possibly involuntary myoclonus (they could also have been quick voluntary
22 responses), the first and fourth trials involved slower, non-myoclonic movements (which never
23 occurred during 29 minutes of non-commanded left third finger baseline), suggesting a *level of*
24 *linguistic comprehension* more complex than the usual "move your X [body part]" type of
25 command.

26 25. *Taken together, the video evidence indicates, beyond any reasonable doubt, that*
27 *the slower, more deliberate-looking non-myoclonic movements are in fact not independent of the*
28 *commands, ruling out some hitherto unknown type of spinal automatism. There is clearly a*

1 causal relationship, indicating that at the times the videos were made, Jahi was in a responsive
2 state, capable of understanding a verbal command and barely capable of executing a simple
3 motor response.

4 26. The obvious question is: How is this possible, given that on September 26, 2014
5 at University Hospital, her EEG was flat, suggesting absolute unconsciousness; her
6 somatosensory evoked response test showed no response above the mid-cervical level,
7 suggesting “loss of neurological brain pathway function above this level;” [Schneider
8 declaration, p. 14, line 1] and her auditory evoked potential test showed no response, suggesting
9 that “she has no auditory pathways.” [Id. at p. 14, line 1] Dr. Schneider interprets the latter result
10 as “establish[ing] to a reasonable degree of medical certainty that J. McMath cannot respond to
11 verbal commands because she has no cerebral mechanism to hear sound.” [Id. at p. 14, lines 6-7]
12 I certainly agree that the tests would seem to imply these things, raising serious difficulties for
13 reconciling them with the video evidence of intermittent responsiveness to commands.

14 27. I do not pretend to know the explanation for the apparent discrepancies. But
15 instead of concluding that “It is a medical impossibility that J. McMath is moving in response to
16 verbal commands,” [Schneider declaration, p. 14, lines 2-3] regardless what the videos show, in
17 a matter as important as life or death I prefer to give the benefit of the doubt to the behavioral
18 evidence of responsiveness, which seems incontrovertible, and entertain the possibility that these
19 tests may not imply as much about the functioning of a severely damaged brain as we usually
20 assume. The following are some possible alternative explanations for the test results.

21 28. But first, let us put to rest a particular complaint regarding these tests, repeated by
22 Drs. Nakagawa and Schneider. Dr. Nakagawa states that “The tests performed on McMath at
23 University Hospital on September [MRI, MRA, MRV, evoked potentials] are not accepted,
24 validated ancillary studies and do not meet accepted diagnostic criteria for determining brain
25 death (i.e., the Guidelines) and are not a substitute for the accepted medical standards.”
26 [Nakagawa declaration, p. 22, lines 7-10] Dr. Schneider states the same: “Although these tests
27 are not the accepted diagnostic criteria for determining brain death, ...” [Schneider declaration,
28 p. 11, lines 15-16] “Brain MRI and MR angiography are not validated tests to assess brain death.

1 The Guidelines state: 'MRI-MR angiography, and perfusion MRI imaging have not been studies
2 sufficiently nor validated in infants and children and cannot be recommended as ancillary studies
3 to assist with the determination of brain death in children at this time.' (Ex. B, p. e729) The
4 above accepted medical standards for diagnosing pediatric brain death have not been applied to
5 J. McMath since Dr. Paul Fisher's examination performed at Children's Hospital Oakland on
6 December 23, 2013." [Schneider declaration, p. 13, lines 8-14]

7 29. Their insistence on this point is a *non sequitur*. The tests were not done in order to
8 "determin[e] brain death" or to "substitute for the accepted medical standards," but to evaluate,
9 out of interest, the structure and electrophysiological functioning of Jahi's brain 9 months after
10 the uncontroverted diagnosis of brain death according to the Guidelines. Regarding the
11 electrophysiological tests, I agree completely with Dr. Schneider that "the results are consistent
12 with J. McMath's diagnosis of brain death made in December 2013," and that "None of the
13 results would cause a reputable expert in pediatric or adult brain death to question or reconsider
14 the accepted brain death assessments of Dr. Robin Shanahan, Dr. Robert Heidersbach and Dr.
15 Paul Fisher performed in December 2013 at Children's Hospital Oakland." [Schneider
16 declaration, p. 11, lines 17-20] But they are also "consistent with" the possibility that Jahi is
17 *currently not* brain dead, even though that would go against the supposed infallibility of the
18 Guidelines.

19 30. First of all, the MRI scan on September 26, 2014 showed that Jahi's brain had
20 (and presumably still has) a surprising amount of preserved structure for a brain that was
21 supposedly totally destroyed 9 months previously. Brain scans on three cases of chronic brain
22 death that I have studied showed complete liquefactive necrosis (destruction) of the entire brain
23 months after the onset of brain death. In one case, the first MRI scan was performed 13.9 years
24 into brain death; an eventual autopsy showed no identifiable brain tissue.³¹ The other two had
25 scans performed closer to the same post-brain-death time frame as Jahi's MRI scan. One was a
26 15-year-old girl who became brain dead from a malignant brain tumor; a CT scan 10 months into
27 brain death showed replacement of most of the brain, especially the cerebral hemispheres, by
28

1 fluid.¹ The other was a boy who became brain dead at age 13 months from an overwhelming
2 presumed viral infection, whose MRI 31 days later showed advanced, widespread necrotic
3 changes; the next neuroimaging was a CT scan 1.7 years into brain death, showing the skull to be
4 filled with disorganized fluids and membranes, without identifiable brain structures.

5 31. If Jahi's MRI scan had shown similar findings, she could not possibly be
6 intermittently responsive, there would be no videos showing what these videos show, and I
7 would not be making this declaration. As it is, Jahi's MRI revealed a surprising extent of
8 relatively preserved brain tissue (albeit with abnormal signal properties). This tells us in
9 retrospect that in December 2013 when she was diagnosed brain dead, the lack of brain function
10 was due more to *low* rather than *absent* blood flow – low enough to abolish neuronal function
11 but not low enough to cause necrosis (tissue destruction) in much of the brain. This range of
12 cerebral blood flow is called the "ischemic penumbra." The goal of stroke therapy is to rescue
13 the (potentially reversibly) nonfunctioning brain tissue in the ischemic penumbra, since the
14 necrotic core of the stroke is already a lost cause. The Brazilian neurologist Coimbra insightfully
15 pointed out that as intracranial blood flow decreases from normal to zero during the
16 pathophysiological vicious cycle leading to brain death, it necessarily passes through a stage of
17 *global* ischemic penumbra.³² If the brain's nonfunction is due to ischemic penumbra, all
18 elements of the standard diagnostic Guidelines will be fulfilled, but there is still the potential for
19 recovery of function because the brain tissue is still viable; therefore, the critical element of
20 *irreversibility* in the statutory definition of death is not fulfilled.

21 32. I am convinced that Jahi's case proves Coimbra's thesis; her intracranial blood
22 flow evidently did *not* progress all the way to zero, which would have resulted in necrosis of the
23 entire brain, as in the three cases described above; much viable, though damaged, brain tissue
24 remains. The fact that her radionuclide blood flow test on December 23, 2013 showed no
25 identifiable brain blood flow proves only that the radionuclide test lacked the sensitivity to
26 distinguish penumbra-level flow from no flow, contrary to the assertions of Dr. Nakagawa that it
27

28 ¹ This and the previous case occurred prior to 1998 and were included in the data set of my article on chronic brain death.²

1 “conclusively demonstrates that there is no blood flow going to McMath’s brain,” [Nakagawa
2 declaration, p. 19, lines 27-28; p. 23, lines 12-14] that “The cerebral blood flow performed on
3 December 23, 2013 is conclusive evidence of McMath’s death,” [Id. at p. 24, lines 1-2] and that
4 “The cerebral blood flow study performed on December 23, 2013, confirmed that McMath had
5 no intracranial blood flow.” [Id. at p. 24, lines 5-7] Dr. Schneider makes the same kind of
6 assertion in his declaration: “The radionuclide cerebral blood study is diagnostic of J. McMath’s
7 brain death in that it conclusively demonstrates there is no blood flow going in J. McMath’s
8 brain.” [Schneider declaration, p. 10, lines 9-11] These statements assume that radionuclide
9 blood flow testing can distinguish no flow from penumbra-level flow in every part of the brain
10 with 100% specificity for no flow—an assumption that has never been validated and is even
11 unlikely, given that hypothalamic function as well as EEG activity can persist despite
12 radionuclide tests or angiography showing apparently no blood flow to the brain.³³⁻³⁸ Grigg et al.
13 described two patients who met all clinical criteria for brain death short of an apnea test, who had
14 flat EEGs and no apparent blood flow on radionuclide testing, yet breathed spontaneously during
15 the apnea test.³⁹

16 33. Jahi’s MRI scan shows severe damage especially to the *brainstem*, with
17 substantial parts of it missing (after the body’s removal of necrotic tissue over the prior 9
18 months), most likely due to brainstem herniation around the time of diagnosis. Thus, it is not at
19 all surprising that Jahi should still demonstrate absence of brainstem reflexes and apnea, and that
20 her motor abilities are so severely limited. By contrast, consciousness, language processing, and
21 initiation of voluntary movements are mediated by higher brain structures, which the MRI shows
22 to be partially preserved.

23 34. Regarding the flat EEG, it is well known that this test reflects the electrical
24 activity of only the part of the brain’s cortical surface directly below the skull. Midline cortex
25 (along the fissure separating the two hemispheres) and cortex at the base of the brain are not
26 sampled by an EEG, nor are deep midline structures such as basal ganglia and thalamus, to say
27 nothing of the brainstem. Thus, the EEG can be flat in cases of so-called “neocortical death”—an
28 extreme form of persistent vegetative state, where patients are unresponsive but spontaneously

1 breathing and manifesting sleep-wake cycles due to an intact brainstem.^{40,41} It can also be flat,
2 or nearly so, in cases of congenital absence of cortex known as hydranencephaly, despite
3 behavioral evidence of adaptive, purposeful interaction with the environment (i.e.,
4 consciousness).⁴² Such cases, together with animal data, suggest that in the context of severe
5 cortical damage or even cortical absence, consciousness can still be mediated subcortically by
6 deep midline structures such as thalamus and basal ganglia, and therefore not reflected in surface
7 EEG activity.^{43,44}

8 35. In Jahi's case, there is the additional element of temporal variability. Most of the
9 time she is not responsive, but sometimes she is. A random neurological examination would
10 most likely find her unresponsive, with no clue as to the latent potential for responsiveness. What
11 if her EEG behaved the same intermittent way? Who knows what her EEG might have looked
12 like on days when the videos demonstrated responsiveness?

13 36. The somatosensory evoked response reveals function of the somatosensory
14 pathways from peripheral nerve to cerebral cortex – and those pathways alone. It does not imply
15 anything about the myriad other ascending and descending pathways between the brain and the
16 spinal cord, such as motor pathways, which are located in different parts of the spinal cord and
17 brainstem from the somatosensory pathways. It is not at all surprising, given the damage to the
18 brainstem revealed on MRI, that there would be no somatosensory evoked responses above the
19 cervical level. But that does not imply that the descending motor pathways are necessarily also
20 nonfunctional. The brainstem is not completely destroyed, and it is totally conceivable that some
21 descending motor pathways have survived. The somatosensory evoked response test, in and of
22 itself, certainly does *not* establish a complete “loss of neurological brain pathway function above
23 this [cervical] level,” [Schneider declaration, p. 14, line 1] if the phrase “brain pathway” is
24 intended to mean *all* pathways.

25 37. The brainstem auditory evoked response (BAER) test is harder to reconcile with
26 responsiveness to commands. There was absence of all the main waves, including Wave I, which
27 is generated peripherally by the acoustic nerve (transporting auditory signals from the cochlea to
28 the brainstem). Wave I is often absent in brain death, in which case the absence of downstream

1 waves implies nothing about the integrity or lack thereof of the brainstem. Absence of Wave I
2 ordinarily indicates a profound peripheral hearing deficit, but it does not necessarily indicate
3 total deafness.^{45, 46} Hearing can be preserved after acoustic neuroma surgery, despite absence of
4 all waves on BAER.⁴⁷ Thus, it is possible that a partial disruption of the axons in the acoustic
5 nerve can suffice to abolish the averaged evoked response but still permit sufficient transmission
6 of auditory signals in the remaining axons to mediate hearing. Since BAER waves are computed
7 averages of the brain's response to click stimuli, absence of Wave I (and consequently of
8 subsequent waves) can also be due to imperfect synchrony of the signals within the acoustic
9 nerve, not necessarily to a complete lack of signals. Instead of reasoning "Jahi's evoked potential
10 test showed no waves; therefore, she absolutely cannot hear," it is preferable to reason "there is
11 behavioral evidence that Jahi hears; therefore, there is something about the evoked potential test
12 and the auditory pathways in her case that we do not completely understand."

13 38. Given the evidence of intermittent responsiveness, we should be all the more
14 willing to remain agnostic regarding her inner state of mind during periods of unresponsivity,
15 rather than automatically equate it with unconsciousness. Patients with severe brain damage can
16 have many other reasons for unresponsiveness besides unconsciousness. Failure to appreciate or
17 properly test for subtle signs of awareness results in a substantial incidence of misdiagnosis of
18 the vegetative state on the part of even experienced neurologists.^{48, 49} Recent advances in
19 technology have revealed that even some "vegetative state" patients who are truly unresponsive
20 can be inwardly conscious, understand what is said to them, and follow verbal commands with
21 their minds.⁵⁰⁻⁵⁶

22 39. Not only seemingly "vegetative" patients can be inwardly aware, but also
23 seemingly comatose patients, for example during general anesthesia,^{57, 58} or cases like Zack
24 Dunlap, who was diagnosed brain dead (whether the Guidelines were followed to the letter
25 remains undocumented) and eventually made an essentially complete recovery; he claims to
26 remember hearing the doctor declare him brain dead and being extremely upset about it.²

27
28

² <http://www.today.com/news/pronounced-dead-man-takes-miraculous-turn-2D80555113>

1 40. The brain has a remarkable capacity to reorganize itself over weeks to months
2 after injury in order to maximize function – a phenomenon called “plasticity.” The fact that it
3 took several months before Jahi first showed signs of intermittent responsiveness is consistent
4 with the time course of brain plasticity.

5 **B. Jahi McMath does not currently meet California’s statutory definition of death**
6 **by neurologic criteria on additional grounds.**

7 41. California’s Health and Safety Code, Section 7180 states that “An individual who
8 has sustained ... irreversible cessation of all functions of the entire brain, including the brain
9 stem, is dead.” The 1/7/14 Supplemental Declaration of Dr. Heidi Flori Opposing Petitioner’s
10 Request for Court Order Compelling Children’s Hospital to Perform Tracheostomy and Insert
11 Gastrointestinal Tube made a special point to underscore this definition by emphasizing the
12 importance of *totality* of brain nonfunction in diagnosing brain death: “The diagnosis of death by
13 neurological criteria is predicated not only on loss of higher cortical functions (emotions,
14 voluntary movements, vision, etc.) but also on complete cessation of *all* brain functions,
15 including those of the brain stem.” (¶6, emphasis in original)

16 42. The accepted medical standards for diagnosing brain death in both adults and
17 children (i.e., the Guidelines) give lip service to this definition, but in fact allow for certain
18 functions of the brain to occur in patients meeting their criteria for “brain death.” As mentioned
19 above, the functions that the guidelines care about are of three “cardinal” categories: coma,
20 cranial nerve reflexes, and apnea. But there are other categories of brain function, which
21 proponents of diagnostic algorithms such as the Guidelines tend to write off as mere “activity” of
22 a few residual neurons (nerve cells).

23 43. The distinction between “function” at the organ level and “activity” at the cellular
24 level is valid and important, as explained by the 1981 President’s Commission:

25 After an organ has lost the ability to *function* within the organism, electrical and
26 metabolic *activity* at the level of individual cells or even groups of cells may
27 continue for a period of time. Unless this cellular activity is organized and
28 directed, however, it cannot contribute to the operation of the organism as a

1 whole. Thus cellular activity alone is irrelevant in judging whether the organism,
2 as opposed to its components, is 'dead.'" (p. 75, emphasis in original)
3 The Commission makes clear that what distinguishes a brain "function" from irrelevant neuronal
4 "activity" is teleological. A function is not defined by how many cells carry it out (which could
5 be very few), but by its role in the organism. Compared to the entire brain, the hypothalamus (a
6 part of the brain that lies above and controls the pituitary gland, among many other functions)
7 contains relatively few neurons, but so does the medulla. Hormonal control of fluid balance, for
8 example, certainly "has significance for the organism as a whole" (p. 28) and "is organized and
9 directed,... contribut[ing] to the operation of the organism as a whole," (p. 75) and therefore
10 qualifies as a "function." If that control is mediated by a part of the brain—regardless how large or
11 small a part—it rightly qualifies as a "brain function" and not merely "cellular activity."

12 44. In discussing the concept of "organism as a whole," Bernat seconds the
13 President's Commission's distinction, listing some examples of "critical functions of the
14 organism as a whole, which include: "(1) ... the autonomic control of circulation; (2) integrating
15 functions that assure the homeostasis of the organism, such as ... neuroendocrine feedback
16 loops, and temperature control." ^{59, pp. 257-8} Nevertheless, three paragraphs later he belittles one of
17 the same functions if it occurs in the context of coma, absent brainstem reflexes, and apnea:
18 "After brain death, ... some hypothalamic neuroendocrine activity of cells producing antidiuretic
19 hormone can be assayed... In these instances, isolated nests of neurons have survived the global
20 insult and continue to function independently. But because the neurological examination reveals
21 an absence of clinical functions, these small, independent, multifocal areas of functioning cells
22 do not contribute materially to the organism's clinical functions and thus do not count as
23 evidence of functioning of the organism as a whole." ^{59, p. 258}

24 45. Bernat, Wijdicks and many others insist that the only functions that are important
25 for distinguishing life from death are "clinical," meaning "those functions that clinicians can
26 assess by bedside physical examination." ^{59, p. 258, 60} But this is completely *ad hoc*, contrary to the
27 explanation that Bernat himself gave of "critical functions" of the organism as a whole (some of
28 which are not assessed in the bedside physical examination), and contrary to the statutory

1 definition of death, which does not restrict the notion of “all brain functions” to the subset
2 assessable by bedside examination. Besides, secretion of antidiuretic hormone by the
3 hypothalamus is even a “clinical” function, if one waits at the bedside long enough to observe
4 the patient’s urinary pattern or looks at the intake and output charting by nurses who have been
5 at the bedside all day. So are blood pressure control and temperature maintenance “clinical
6 functions” (vital signs are part of the bedside examination).

7 46. Nevertheless, the 1995 Practice Parameters for Determining Brain Death in
8 Adults explicitly state that “Normal blood pressure without pharmacologic support” as well as
9 “absence of diabetes insipidus” (i.e., maintenance of fluid balance through secretion of
10 antidiuretic hormone by the hypothalamus) are “compatible with the diagnosis of brain death.”¹⁴
11 The 2010 update specifies normal systolic blood pressure as a diagnostic prerequisite, stating
12 “Hypotension... is common; vasopressors or vasopressin are often required,” implying that they
13 are not *always* required.¹³ There is no requirement that temperature regulation be absent. (In
14 fact, a core temperature ≥ 36.5 °C is a diagnostic prerequisite for the 1995 adult criteria, ≥ 36 °C
15 for the 2010 update, and > 35 °C for the pediatric Guidelines). Although temperature regulation is
16 indeed faulty in most patients diagnosed as brain dead, some maintain normal body temperature
17 without extraordinary warming measures beyond standard blankets. These functions are, by
18 Bernat’s account, “critical functions of the organism as a whole,” and they are “brain functions”
19 (parts of the hypothalamus and brainstem).³ In fact, they are more critical to the organism as a
20 whole than most, if not all, of the cranial nerve reflexes that the Guidelines require to be absent,
21 and which are mediated by “nests of neurons” no more extensive than those in the hypothalamus.

22 47. This discrepancy between what the Guidelines diagnose and what the statutory
23 definition of death specifies has been pointed out by many commentators.⁶¹⁻⁶⁸ Probably the main
24 reason why the Guidelines focus so much on cranial nerve reflexes, to the exclusion of other
25 types of clinically evident brain functions, is that they were drafted so as to correspond to the
26

27 ³ Cardiovascular stability can in principle be maintained by spinal cord function, as the chronic brain death cases
28 prove. But most brain death diagnoses are made in the very acute phase, when the spinal cord is relatively
nonfunctional due to “spinal shock;” cardiovascular stability without pressor medication during that phase is
therefore more likely attributable to brainstem function.

1 standard bedside neurological examination of a comatose patient.⁶⁹ If the brain death guidelines
2 had been drafted by neuroendocrinologists, hypothalamic functions might well have been
3 included in the list of brain functions required to be absent; and if they had been drafted by
4 neurocardiologists, autonomic control of heart rate and blood pressure might well have been
5 included among the brainstem functions required to be absent.

6 48. I shall expand briefly on neuroendocrine functions, because they are particularly
7 relevant in Jahi's case. They frequently persist in patients who fulfill the standard diagnostic
8 criteria for brain death. The most externally obvious neuroendocrine function commonly
9 encountered in clinically diagnosed (but not statutorily defined) brain death is regulation of fluid
10 balance through secretion of antidiuretic hormone (vasopressin) by the posterior pituitary gland,
11 which is an extension of the hypothalamus. Absence of this hypothalamic function is manifested
12 by a massive outpouring of dilute urine, a condition called diabetes insipidus. The reported
13 incidence of preservation of this brain function (i.e. lack of diabetes insipidus) in brain death
14 varies widely, but the average is around one-third of cases.^{34, 70-76} The 1995 adult guidelines
15 explicitly state that absence of diabetes insipidus is compatible with brain death, in flat
16 contradiction to the statutory definition.^{14, 77, p. 1007} The 2010 adult update¹³ and the 2011
17 pediatric update¹⁰⁻¹² do not specifically mention diabetes insipidus, implicitly continuing to
18 endorse the 1995 compatibility statement.

19 49. Regulation of anterior pituitary hormones by the hypothalamus is less clinically
20 obvious than the presence or absence of diabetes insipidus, but it is a no less physiologically
21 relevant *brain function* (actually multiple brain functions, one for each hormone regulated).^{34-37,}
22 ^{71, 72, 78, 79} This includes normal levels of the sex hormones involved in puberty and menstruation.
23 ^{35, 36, 72, 80} Thus, the statement by Dr. Schneider in his declaration—that in brain death “Hormones
24 normally secreted by the brain [thyroid, adrenocorticoid, vasopressin] have to be externally
25 supplied” [Schneider declaration, p. 6, lines 20-21]—is erroneous as a generalization.

26 50. Jahi McMath has diabetes insipidus, which is treated with hormone replacement.
27 But she has evidence of different hypothalamic functions, namely puberty and menstruation.
28 Menstruation occurred twice at St. Peter's hospital (physician progress notes, 8/6/14, 8/7/14, and

1 8/9/14, mentioning menstruation at that time and "a few months" prior) and a third time in her
2 apartment (nursing notes, 9/9/14). She has also had development of pubic and axillary hair and
3 breast enlargement since becoming brain dead. Neither the adult nor pediatric brain death
4 Guidelines make any mention of puberty or menstruation, but clearly these are evidence of
5 hypothalamic brain function, in contradiction to California's statutory definition of death.
6 Corpses do not menstruate or develop sexually.

7 51. Jahi's body certainly functions biologically as a unified living organism, severely
8 disabled and dependent on support to be sure. Loss of integrative unity was the rationale for why
9 the 1981 President's Commission considered brain death to be death, and why the Commission
10 felt confident in drafting the Uniform Determination of Death Act, after which most state death
11 statutes (including California's) are modeled. It is also why the physicians at Oakland Children's
12 hospital in December 2013-January 2014, and many other physician commentators at the time,
13 were so sure that the diagnosis of brain death was correct in Jahi's case, not only because she
14 fulfilled the diagnostic Guidelines but also because her biological organism was showing signs
15 of dis-integration, as artificially maintained corpses necessarily do.

16 52. The 1/7/14 Supplemental Declaration of Dr. Heidi Flori nicely summarized these
17 signs. It is worth quoting at length, in light of how Jahi's subsequent course defied all predictions
18 of what must happen to dead bodies maintained indefinitely on ventilators:

19 "6.... The brain stem provides vital regulatory control for critical bodily functions
20 such as maintenance of heart rate, temperature, and respiratory effort, as well as
21 regulation of nerve impulses that adjust the tone of blood vessels and nerves
22 throughout the body. Therefore, the body of Ms. McMath, unlike the bodies of
23 those patients with severe brain injury but with retained brain stem reflexes
24 (including Terry Schiavo and Ariel Sharon), simply cannot regulate these life-
25 sustaining functions over time.

26 7. The inability of Ms. McMath's body to regulate life-sustaining functions is
27 already being demonstrated in many ways, including as follows:

28

1 a. She has not had evidence of bowel functioning (sounds) for weeks.
2 Yesterday (January 2), she passed some stool that was clinically consistent with
3 defecation of the tissues lining the bowel (i.e., her body is sloughing her gut). In
4 living persons, absence of bowel sounds and sloughing of gut materials are both
5 indications that enteral nutrition, which would occur through the g-tube being
6 proposed, may be deleterious, particularly where, as here, there has been prior
7 cardiopulmonary arrest and regulation of blood flow to the gut has been or
8 continues to be compromised.

9 b. Although the medical team has done an excellent job of maintaining the
10 body's external appearance (the hair is done, nails manicured, etc.), the tissues
11 beneath the skin (subcutaneous tissues and muscles) are showing gradual signs of
12 deterioration including change in skin "turgor" (in essence, its elasticity) and
13 increase in muscle contraction (due to the loss of nervous system regulation).

14 c. The body also does not exhibit airway protective reflexes such as cough
15 which are initiated by the brainstem. Although we are applying inhaled therapy
16 twice daily to improve the body's "pulmonary toilet" (its clearance of pulmonary
17 respiratory secretions), its secretions are continuing to change adversely with
18 time. They are now more malodorous, changed in color (sometimes tan, creamy
19 or bloody) and thicker in consistency.

20 d. Without nervous system control to adjust blood vessel tone with
21 changes in body movement (as would normally need to occur to allow living
22 persons to move from lying to sitting and sitting to standing), the body
23 occasionally exhibits precipitous, although so far temporary, changes in blood
24 pressure and oxygenation levels when staff are moving the torso up or down or
25 side to side in order to complete daily care routines.

26 e. The body is unable to regulate temperature. Blankets are needed to
27 maintain a temperature of greater than 35 degrees Celsius (95 degrees
28 Fahrenheit).

1 f. Finally, the body has had gradually deteriorating blood pressures over
2 the last three weeks, with blood pressures often half of what they were at the time
3 Ms. McMath was first declared deceased. This again, is a testament to the body's
4 long post-mortem course.

5 **8. The medical team and I believe that additional and more dramatic signs of**
6 **the body's deterioration will continue to manifest over time regardless of any**
7 **procedures and regardless of any heroic measures that any facility in the**
8 **country might attempt. This deterioration became inevitable the moment she**
9 **died. Mechanical support and other measures taken to maintain an illusion**
10 **of life where none exists cannot maintain that illusion indefinitely.**

11 9. The additional medical interventions Petitioner proposes are unprecedented.
12 They simply will not bring her back to life nor enable others to do so. **Nor can**
13 **they correct or even improve the above-described manifestations of the post-**
14 **mortem deterioration of Ms. McMath's body.** Indeed, such measures may well
15 be counterproductive, perhaps even resulting in expedited cardiopulmonary
16 cessation." (emphasis added)

17 53. Every other physician commentator at the time seconded this opinion, as the news
18 media documented. To take just one example:

19 "The bodies of brain dead patients kept on ventilators gradually deteriorate,
20 eventually causing blood pressure to plummet and the heart to stop, said Dr. Paul
21 Vespa, director of neurocritical care at the University of California, Los Angeles,
22 who has no role in McMath's care. The process usually takes only days but can
23 sometimes continue for months, medical experts say."⁸¹

24 54. Thus, Jahi's deterioration in late December 2013 and early January 2014 was held
25 up as proof that she was most certainly a corpse being artificially maintained with the appearance
26 of life. What then happened was that, upon transfer to St. Peter's Hospital in New Jersey, she
27 received the tracheostomy and gastrostomy feeding tube that were refused in Oakland. She
28 received the enteral feedings that her gut was supposedly unable to handle and that would only

1 be deleterious. With proper nutrition and other treatments appropriate for a patient requiring
2 intensive care, her intestines healed, her skin turgor and pulmonary status recovered to normal,
3 and she regained spontaneous maintenance of blood pressure without pressor medications. She
4 still requires blankets to maintain temperature, but for the past 3+ years she has remained
5 remarkably healthy, apart from being severely neurologically disabled. Most of that time she has
6 not even been in a hospital, but in an apartment with the assistance of nothing more than a
7 ventilator, excellent nursing care, hormone supplementation, and nutrition. Such recovery from
8 impending multisystem failure and such improvement in overall health, as Jahi exhibited in the
9 early months of 2014, is not possible for a ventilated corpse.

10 55. Dr. Schneider is certainly correct that "There is absolutely no medical possibility
11 that J. McMath has recovered, or will someday recover, from death." [declaration, p. 14, lines
12 10-11] Short of biblical miracles, there is, by definition, absolutely no possibility that *anyone* can
13 recover from death. What the above lines of evidence and reasoning show is rather that Jahi
14 McMath was never truly dead, even though she fulfilled the accepted medical criteria for death
15 in December 2013. She exhibited no brain function at the time, but the cessation of at least two
16 functions—consciousness and hypothalamic regulation of menstruation and sexual development—
17 has proved *not* to be *irreversible*. Hence she represents an example of a false positive (erroneous)
18 diagnosis of brain death following the Guidelines.

19 56. The Guidelines permit the persistence of some brain functions (neuroendocrine,
20 autonomic); therefore, they do not establish cessation of *all* brain functions, as California's
21 statutory definition of death requires. Moreover, Jahi's case demonstrates that neither do they
22 establish *irreversibility* of cessation of function, given that there is evidence, **to a reasonable**
23 **degree of medical certainty**, of return of consciousness intermittently and recovery of some
24 hypothalamic function.

25 57. Jahi McMath is a living, severely disabled young lady, who currently fulfills
26 neither the standard diagnostic Guidelines for brain death nor California's statutory definition of
27 death. At the very least, in a matter of life versus death, the compelling evidence of
28 responsiveness to commands and of puberty warrants giving life the benefit of the doubt.

1 **C. Opinions about the concept of brain death are irrelevant to whether Jahi**
2 **McMath fulfills the accepted medical standards for brain death or whether she**
3 **meets California's statutory definition of death**

4 58. Dr. Schneider states in his declaration: "I understand that plaintiff's allegation that
5 J. McMath is not dead is based on the opinion of D. Alan Shewmon, M.D. The dissenting theory
6 proposed by Dr. Shewmon is that death is not a neurological phenomena [sic] and death only
7 occurs after total cessation of the systemic circulation. This theory is contrary to the accepted
8 medical and legal standards that brain death is a legal criterion for death. Dr. Shewmon's opinion
9 is a philosophical minority opinion that denies and conflicts with the accepted medical standards
10 in the Guidelines as well as California law." [Schneider declaration, p. 14, lines 12-17]

11 59. I feel obliged to respond before the court to this *ad hominem* remark. First, my
12 opinion about the *conceptual rationale* for brain death is completely irrelevant to my competence
13 as a pediatric neurologist and to my clinical judgment whether Jahi McMath fulfills or does not
14 fulfill the accepted medical standards (the pediatric Guidelines) for brain death or whether she
15 meets California's statutory definition of death. The "plaintiff's allegation that J. McMath is not
16 dead" is not in any way whatsoever "based on" my opinion about the philosophical nature of
17 death. That ought to be enough said, but the implication that I am some sort of lone outlier
18 among my professional colleagues as regards this topic, and that my "minority opinion" should
19 in essence be disregarded on account of conflict with "accepted medical standards... as well as
20 California law," cannot be left unaddressed.

21 60. After completion of my training, for the next 11 years I accepted the mainstream
22 understanding of brain death, that it was merely an alternative way of diagnosing the same
23 physiological state as traditional death after cardiorespiratory arrest. I published and lectured to
24 that effect, seconding the 1981 President's Commission's rationale that brain death was death by
25 virtue of loss of integrative unity of the organism as a whole.⁸² Then, in 1992 I consulted on a
26 case that convinced me that at least some, perhaps many, cases of brain death were nevertheless
27 human organisms as a whole, and therefore permanently comatose yet still living human beings.
28 Subsequent research and clinical experience has only served to reinforce that conclusion.

1 61. Busy clinicians generally pay little attention to the philosophical, conceptual
2 debates surrounding brain death, being content to follow the officially endorsed diagnostic
3 algorithm and move on to the next patient. If one asks them whether they think brain death is
4 death, the vast majority will say yes. In that superficial respect, my opinion that brain death is
5 not true death is very much in the minority among clinical neurologists. But if one probes deeper
6 and asks *why* they think brain death is death, one finds that about half of them actually think that
7 brain-dead patients are biologically living human organisms—which is exactly my position—but
8 that they are “dead” purely by virtue of irreversible loss of consciousness (contrary to my
9 position and to every statutory definition of death).⁸³⁻⁸⁵

10 62. The editorial reinforcing my 1998 feature article in *Neurology* was written by one
11 of the most prominent experts on and defenders of brain death at the time, the late Dr. Ronald
12 Cranford.³ Under the catchy title “Even the dead are not terminally ill anymore,” he stated:
13 “Alan Shewmon, MD, in this issue of *Neurology*, has accumulated convincing data that, among
14 other things, undermine this somatic disintegration hypothesis.... Shewmon’s article and the
15 extensive case documentation, along with thoughtful concerns raised by scholars in recent years,
16 create serious questions about the validity of the somatic disintegration basis for brain death as
17 death and justify continued exploration of the issue.” This is the same Dr. Cranford who wrote
18 on another occasion: “It seems then that permanently unconscious patients have characteristics of
19 both the living and the dead. It would be tempting to call them dead and then retrospectively
20 apply the principles of death, *as society has done with brain death.*” (emphasis added)^{86, p. 243}

21 63. My presentation to the President’s Council on Bioethics was instrumental in the
22 Council’s abandoning the integrative unity rationale for brain death, held by the 1981 President’s
23 Commission and mainstream neuroethics thereafter.⁵⁹ The Council’s white paper cited my
24 publications more frequently than those of any other author and seconded my critique of the
25 mainstream rationale: “If being alive as a biological organism requires being a whole that is
26 more than the mere sum of its parts, then it would be difficult to deny that the body of a patient
27 with total brain failure can still be alive, at least in some cases.”^{9, p. 57} Two of the three personal
28

1 statements at the end of the white paper took my position, including that of Council Chairman
2 Dr. Edmund Pellegrino.^{87, 88}

3 64. Dr. Allan Ropper, Professor of Neurology at Harvard Medical School and
4 Executive Vice Chair of Neurology at Brigham and Women's Hospital evidently implicitly
5 accepts that, from a biological perspective, at least some brain-dead patients are comatose, living
6 human organisms: "In exceptional cases [of brain death], however, the provision of adequate
7 fluid, vasopressor, and respiratory support allows preservation of the *somatic organism in a*
8 *comatose state* for longer periods."^{89, p. 962} (emphasis added) The term "comatose state" applies
9 only to living organisms that are normally conscious, not to corpses.

10 65. The late Dr. Fred Plum, one of the great luminaries of neurology regarding coma
11 and brain death, during the question-and-answer session after my keynote address at the 3rd
12 International Symposium on Coma and Death, Havana, Feb. 22-25, 2000, interjected: "OK, I'll
13 grant you that the brain-dead body is a living human organism, but is it a human *person*?" –
14 thereby shifting the death debate from biology to philosophy. At which he proceeded to
15 propound person/mind/brain reductionism as the *real* reason why brain death is death, insisting
16 that the biological vital status of the body is philosophically and ethically irrelevant – another
17 example of conflict with California law and every other state law, by a neurologist with much
18 more prestige than myself, Dr. Nakagawa or Dr. Schneider, and an ardent proponent of brain
19 death.

20 66. Dr. James Bernat is Professor of Neurology at Dartmouth Medical School, a
21 highly respected expert in neuroethics, and undoubtedly the most important defender of the
22 mainstream rationale for brain death. Although he and I hold differing views about brain death,
23 we regard each other's work with great esteem and mutual respect. In the chapter on brain death
24 in the most recent edition of his book "Ethical Issues in Neurology," after discussing critiques of
25 brain death theory by myself and others, he wrote with remarkable open-mindedness and
26 humility: "I concede that the doctrine of whole brain death remains imperfect and that my
27 attempts and those of others to respond to its shortcomings noted by critics remain inadequate."
28 ^{59, p. 266} So as not to take this quotation out of context, I should add that it is hard to abandon a

1 life-long conceptual momentum, so he continued, almost ignoring what he had just written, "Yet,
2 its conceptual soundness, intuitive appeal, universal acceptance by medical societies and
3 lawmakers, and widespread societal acceptance mean that it is coherent biologically and has
4 succeeded as public policy."

5 67. Freudian slips of various expert defenders of brain death also reveal that, at a deep
6 level, they actually agree with me that brain-dead patients are biologically alive. To quote a few
7 of the most striking examples:

8 68. In an article on a pregnant brain-dead woman supported for 107 days until
9 delivery of the fetus, the mother was said to have died upon discontinuing support post-delivery,
10 not when she became brain dead.⁹⁰ In the discussion section, regarding a related case the authors
11 stated, "The [brain dead] mother died of spontaneous cardiac arrest 2 days after the delivery."


12 69. The neurosurgeon Albrecht Harders wrote: "Transcranial Doppler findings were
13 obtained in 15 patients who fulfilled the clinical criteria for brain death... All of the patients died
14 within 24 hours or upon discontinuation of the mechanical ventilation."^{91, p. 115}

15 70. Dr. Fred Plum, mentioned above, wrote a book chapter on brain death, including a
16 table entitled "Prolonged Visceral Survival after Brain Death," the fifth column of which had the
17 heading "Mode of Death."^{92, p. 38} Included in this column were entries of either "spontaneous
18 cardiac arrest" or "respirator discontinued," implying that these patients were dead *not* by virtue
19 of brain death, which had taken place from 26 to 201 days before, but rather by virtue of
20 circulatory-respiratory arrest. Later in the same chapter, regarding a series of 73 brain-dead
21 patients, Plum wrote: "half experienced asystole by the third day but the bodies of 2 *lived on*
22 until the 10th and 16th day." (emphasis added)^{92, p. 53}

23 71. Attachment 1 contains a bibliography of critiques of the biological "integrative
24 unity" rationale for brain death, to demonstrate that a great many experts share my "minority
25 opinion" regarding the traditional basis for equating brain death with death. (Of course the listing
26 does not imply that I agree with all of the authors in every other way, especially with those who
27 advocate "higher brain" (consciousness-based) formulations of death or the thesis that biological
28 death does not ethically matter for harvesting of vital organs).

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27
28

I declare under penalty of perjury under the laws of the State of California that the foregoing is true and correct. Executed this 12th day of December, 2017 at Encino, California.


D. Alan Shewmon, M.D.

References

1. Shewmon DA. The probability of inevitability: the inherent impossibility of validating criteria for brain death or "irreversibility" through clinical studies. *Stat Med* 1987;6:535-553.
2. Shewmon DA. Chronic "brain death": meta-analysis and conceptual consequences. *Neurology* 1998;51:1538-1545.
3. Cranford R. Even the dead are not terminally ill anymore [editorial]. *Neurology* 1998;51:1530-1531.
4. Shewmon DA. The brain and somatic integration: insights into the standard biological rationale for equating "brain death" with death. *J Med Philos* 2001;26:457-478.
5. Task Force for the Determination of Brain Death in Children. Guidelines for the determination of brain death in children. *Pediatrics* 1987;80:298-300.
6. White RJ, Angstwurm H, Carrasco de Paula IC, eds. Working Group on the Determination of Brain Death and its Relationship to Human Death. 10-14 December, 1989. (Scripta Varia 83). Vatican City: Pontifical Academy of Sciences, 1992.
7. Sanchez Sorondo M, ed. The Signs of Death. The Proceedings of the Working Group 11-12 September 2006. Vatican City: Pontificia Academia Scientiarum, 2007.
8. Machado C, Shewmon DA, eds. Brain Death and Disorders of Consciousness. New York: Kluwer Academic/Plenum Publishers, 2004.
9. President's Council on Bioethics. Controversies in the Determination of Death. Washington, D.C.: President's Council on Bioethics, 2008.
10. Nakagawa TA, Ashwal S, Mathur M, Mysore M. Guidelines for the determination of brain death in infants and children: an update of the 1987 Task Force recommendations - executive summary. *Ann Neurol* 2012;71:573-585.
11. Nakagawa TA, Ashwal S, Mathur M, et al. Guidelines for the determination of brain death in infants and children: an update of the 1987 Task Force recommendations. *Pediatrics* 2011;128:e720-740.

- 1 12. Nakagawa TA, Ashwal S, Mathur M, et al. Guidelines for the determination of brain death in
2 infants and children: an update of the 1987 Task Force recommendations. *Crit Care Med*
3 2011;39:2139-2155.
- 4 13. Wijdicks EFM, Varelas PN, Gronseth GS, Greer DM. Evidence-based guideline update:
5 determining brain death in adults: report of the Quality Standards Subcommittee of the
6 American Academy of Neurology. *Neurology* 2010;74:1911-1918.
- 7 14. American Academy of Neurology - Quality Standards Subcommittee. Practice parameters for
8 determining brain death in adults (Summary statement). *Neurology* 1995;45:1012-1014.
- 9 15. Giacino JT, Ashwal S, Childs N, et al. The minimally conscious state: definition and
10 diagnostic criteria. *Neurology* 2002;58:349-353.
- 11 16. Giacino JT. The vegetative and minimally conscious states: Consensus-based criteria for
12 establishing diagnosis and prognosis. *Neurorehabilitation* 2004;19:293-298.
- 13 17. Bussel B, Roby-Brami A, Azouvi P, Biraben A, Yakovleff A, Held JP. Myoclonus in a
14 patient with spinal cord transection. Possible involvement of the spinal stepping generator.
15 *Brain* 1988;111:1235-1245.
- 16 18. Calancie B. Spinal myoclonus after spinal cord injury. *J Spinal Cord Med* 2006;29:413-424.
- 17 19. Calancie B, Needham-Shropshire B, Jacobs P, Willer K, Zych G, Green BA. Involuntary
18 stepping after chronic spinal cord injury. Evidence for a central rhythm generator for
19 locomotion in man. *Brain* 1994;117:1143-1159.
- 20 20. Dietz V, Colombo G, Jensen L, Baumgartner L. Locomotor capacity of spinal cord in
21 paraplegic patients. *Ann Neurol* 1995;37:574-582.
- 22 21. Thomas CK, Dididze M, Martinez A, Morris RW. Identification and classification of
23 involuntary leg muscle contractions in electromyographic records from individuals with
24 spinal cord injury. *J Electromyogr Kinesiol* 2014;24:747-754.
- 25 22. Andary MT, Green DF, Hulce VD, Pysh JJ. Spinal myoclonus complicating spasticity in
26 spinal cord injury: a case study. *Arch Phys Med Rehabil* 1997;78:1007-1009.
- 27 23. Bussel B, Roby-Brami A, Neris OR, Yakovleff A. Evidence for a spinal stepping generator
28 in man. *Paraplegia* 1996;34:91-92.

- 1 24. Chiodo AE, Saval A. Intrathecal baclofen for the treatment of spinal myoclonus: a case
2 series. *J Spinal Cord Med* 2012;35:64-67.
- 3 25. Dimitrijevic MR, Nathan PW, Sherwood AM. Clonus: the role of central mechanisms. *J*
4 *Neurol Neurosurg Psychiatry* 1980;43:321-332.
- 5 26. Fouillet N, Wiart L, Arne P, Alaoui P, Petit H, Barat M. Propriospinal myoclonus in
6 tetraplegic patients: clinical, electrophysiological and therapeutic aspects. *Paraplegia*
7 1995;33:678-681.
- 8 27. Frenken CW, Notermans SL, Korten JJ, Horstink MW. Myoclonic disorders of spinal origin.
9 *Clin Neurol Neurosurg* 1976;79:107-118.
- 10 28. Mello MT, Silva AC, Rueda AD, Poyares D, Tufik S. Correlation between K complex,
11 periodic leg movements (PLM), and myoclonus during sleep in paraplegic adults before and
12 after an acute physical activity. *Spinal cord* 1997;35:248-252.
- 13 29. Mummidisetty CK, Bohorquez J, Thomas CK. Automatic analysis of EMG during clonus. *J*
14 *Neurosci Methods* 2012;204:35-43.
- 15 30. Telles SC, Alves RC, Chadi G. Periodic limb movements during sleep and restless legs
16 syndrome in patients with ASIA A spinal cord injury. *J Neurol Sci* 2011;303:119-123.
- 17 31. Repertinger S, Fitzgibbons WP, Omojola MF, Brumback RA. Long survival following
18 bacterial meningitis-associated brain destruction. *J Child Neurol* 2006;21:591-595.
- 19 32. Coimbra CG. Implications of ischemic penumbra for the diagnosis of brain death. *Braz J*
20 *Med Biol Res* 1999;32:1479-1487.
- 21 33. Flowers WM, Jr., Patel BR. Radionuclide angiography as a confirmatory test for brain death:
22 a review of 229 studies in 219 patients. *South Med J* 1997;90:1091-1096.
- 23 34. Gramm H-J, Meinhold H, Bickel U, et al. Acute endocrine failure after brain death?
24 *Transplantation* 1992;54:851-857.
- 25 35. Schrader H, Krogness K, Aakvaag A, Sortland O, Purvis K. Changes of pituitary hormones
26 in brain death. *Acta Neurochir (Wien)* 1980;52:239-248.
- 27 36. Arita K, Uozumi T, Oki S, Ohtani M, Mikami T. The function of the hypothalamo-pituitary
28 axis in brain dead patients. *Acta Neurochir (Wien)* 1993;123:64-75.

- 1 37. Sugimoto T, Sakano T, Kinoshita Y, Masui M, Yoshioka T. Morphological and functional
2 alterations of the hypothalamic-pituitary system in brain death with long-term bodily living.
3 *Acta Neurochir (Wien)* 1992;115:31-36.
- 4 38. Drake B, Ashwal S, Schneider S. Determination of cerebral death in the pediatric intensive
5 care unit. *Pediatrics* 1986;78:107-112.
- 6 39. Grigg MM, Kelly MA, Celesia GG, Ghobrial MW, Ross ER. Electroencephalographic
7 activity after brain death. *Arch Neurol* 1987;44:948-954.
- 8 40. Brierley JB, Graham DI, Adams JH, Simpson JA. Neocortical death after cardiac arrest. A
9 clinical, neurophysiological, and neuropathological report of two cases. *Lancet* 1971;2:560-
10 565.
- 11 41. Mizrahi EM, Pollack MA, Kellaway P. Neocortical death in infants: behavioral, neurologic,
12 and electroencephalographic characteristics. *Pediatr Neurol* 1985;1:302-305.
- 13 42. Shewmon DA, Holmes GL, Byrne PA. Consciousness in congenitally decorticate children:
14 "developmental vegetative state" as self-fulfilling prophecy. *Dev Med Child Neurol*
15 1999;41:364-374.
- 16 43. Aleman B, Merker B. Consciousness without cortex: a hydranencephaly family survey. *Acta*
17 *Paediatr* 2014;103:1057-1065.
- 18 44. Merker B. Consciousness without a cerebral cortex: a challenge for neuroscience and
19 medicine. *Behav Brain Sci* 2007;30:63-81; discussion 81-134.
- 20 45. Starr A, Picton TW, Sisinger Y, Hood LJ, Berlin CI. Auditory neuropathy. *Brain* 1996;119 (
21 Pt 3):741-753.
- 22 46. Zeng FG, Kong YY, Michalewski HJ, Starr A. Perceptual consequences of disrupted
23 auditory nerve activity. *J Neurophysiol* 2005;93:3050-3063.
- 24 47. Roberson JB, Jr., Jackson LE, McAuley JR. Acoustic neuroma surgery: absent auditory
25 brainstem response does not contraindicate attempted hearing preservation. *The*
26 *Laryngoscope* 1999;109:904-910.
- 27 48. Andrews K, Murphy L, Munday R, Littlewood C. Misdiagnosis of the vegetative state:
28 retrospective study in a rehabilitation unit. *BMJ (Clinical research ed)* 1996;313:13-16.

- 1 49. Majerus S, Gill-Thwaites H, Andrews K, Laureys S. Behavioral evaluation of consciousness
2 in severe brain damage. *Progr Brain Res* 2005;150:397-413.
- 3 50. Laureys S, Celesia GG, Cohadon F, et al. Unresponsive wakefulness syndrome: a new name
4 for the vegetative state or apallic syndrome. *BMC Med* 2010;8:68.
- 5 51. Schoenle PW, Witzke W. How vegetative is the vegetative state? Preserved semantic
6 processing in VS patients - Evidence from N 400 event-related potentials.
7 *Neurorehabilitation* 2004;19:329-334.
- 8 52. Menon DK, Owen AM, Williams EJ, et al. Cortical processing in persistent vegetative state.
9 [letter] *Lancet* 1998;352:200.
- 10 53. de Jong BM, Willemsen AT, Paans AM. Regional cerebral blood flow changes related to
11 affective speech presentation in persistent vegetative state. *Clin Neurol Neurosurg*
12 1997;99:213-216.
- 13 54. Owen AM, Coleman MR, Boly M, Davis MH, Laureys S, Pickard JD. Detecting awareness
14 in the vegetative state. *Science* 2006;313:1402.
- 15 55. Monti MM, Vanhaudenhuyse A, Coleman MR, et al. Willful modulation of brain activity in
16 disorders of consciousness. *N Engl J Med* 2010;362:579-589.
- 17 56. Cruse D, Chennu S, Chatelle C, et al. Bedside detection of awareness in the vegetative state:
18 a cohort study. *Lancet* 2011;378:2088-2094.
- 19 57. Avidan MS, Jacobsohn E, Glick D, et al. Prevention of intraoperative awareness in a high-
20 risk surgical population. *N Engl J Med* 2011;365:591-600.
- 21 58. Mashour GA, Avidan MS. Intraoperative awareness: controversies and non-controversies. *Br*
22 *J Anaesth* 2015;115 Suppl 1:i20-i26.
- 23 59. Bernat JL. *Ethical Issues in Neurology*, 3rd ed. Philadelphia: Lippincott Williams & Wilkins,
24 2008.
- 25 60. Wijdicks EF. The case against confirmatory tests for determining brain death in adults.
26 *Neurology* 2010;75:77-83.
- 27 61. Halevy A, Brody B. Brain death: reconciling definitions, criteria, and tests. *Ann Intern Med*
28 1993;119:519-525.

- 1 62. Miller FG, Truog RD. Death, Dying, and Organ Transplantation. Reconstructing Medical
2 Ethics at the End of Life. Oxford University Press, 2012.
- 3 63. Truog RD. Brain death - too flawed to endure, too ingrained to abandon. J Law Med Ethics
4 2007;35:273-281.
- 5 64. Truog RD, Miller FG. The dead donor rule and organ transplantation. N Engl J Med
6 2008;359:674-675.
- 7 65. Joffe AR. The neurological determination of death: what does it really mean? Iss Law Med
8 2007;23:119-140.
- 9 66. Nair-Collins M. Death, brain death, and the limits of science: why the whole-brain concept of
10 death is a flawed public policy. J Law Med Ethics 2010;38:667-683.
- 11 67. Nair-Collins M. Taking science seriously in the debate on death and organ transplantation.
12 Hastings Cent Rep 2015;45:38-48.
- 13 68. Henderson DS. Death and Donation. Rethinking Brain Death as a Means for Procuring
14 transplantable Organs. Eugene, OR: Pickwick Publications, 2011.
- 15 69. Posner JB, Saper CB, Schiff ND, Plum F. Plum and Posner's Diagnosis of Stupor and Coma,
16 4th ed. Oxford: Oxford University Press, 2007.
- 17 70. Dosemeci L, Yilmaz M, Cengiz M, Dora B, Ramazanoglu A. Brain death and donor
18 management in the intensive care unit: experiences over the last 3 years. Transplant Proc
19 2004;36:20-21.
- 20 71. Keogh AM, Howlett TA, Perry L, Rees LH. Pituitary function in brain-stem dead organ
21 donors: a prospective survey. Transplant Proc 1988;20:729-730.
- 22 72. Howlett TA, Keogh AM, Perry L, Touzel R, Rees LH. Anterior and posterior pituitary
23 function in brain-stem-dead donors. A possible role for hormonal replacement therapy.
24 Transplantation 1989;47:828-834.
- 25 73. Varelas PN, Rehman M, Abdelhak T, et al. Single brain death examination is equivalent to
26 dual brain death examinations. Neurocrit Care 2011;15:547-553.
- 27 74. Nygaard CE, Townsend RN, Diamond DL. Organ donor management and organ outcome: a
28 6-year review from a Level I trauma center. J Trauma 1990;30:728-732.

- 1 75. Salim A, Martin M, Brown C, Belzberg H, Rhee P, Demetriades D. Complications of brain
2 death: frequency and impact on organ retrieval. *Am Surg* 2006;72:377-381.
- 3 76. Fiser DH, Jimenez JF, Wrape V, Woody R. Diabetes insipidus in children with brain death.
4 *Crit Care Med* 1987;15:551-553.
- 5 77. Wijdicks EFM. Determining brain death in adults. *Neurology* 1995;45:1003-1011.
- 6 78. Hall GM, Mashiter K, Lumley J, Robson JG. Hypothalamic-pituitary function in the "brain-
7 dead" patient. [letter] *Lancet* 1980;2:1259.
- 8 79. Powner DJ, Hendrich A, Lagler RG, Ng RH, Madden RL. Hormonal changes in brain dead
9 patients. *Crit Care Med* 1990;18:702-708.
- 10 80. Amado JA, Lopez-Espadas F, Vazquez-Barquero A, et al. Blood levels of cytokines in brain-
11 dead patients: relationship with circulating hormones and acute-phase reactants. *Metabolism*
12 1995;44:812-816.
- 13 81. Collins T, Leff L. Experts: Clock ticking for CA 'brain dead' teen. Associated Press 2014
14 1/6/2014.
- 15 82. President's Commission for the Study of Ethical Problems in Medicine and Biomedical and
16 Behavioral Research. *Defining Death: Medical, Legal, and Ethical Issues in the*
17 *Determination of Death*. Washington, DC: U.S. Government Printing Office, 1981.
- 18 83. Joffe AR, Anton N. Brain death: understanding of the conceptual basis by pediatric
19 intensivists in Canada. *Arch Pediatr Adolesc Med* 2006;160:747-752.
- 20 84. Joffe AR, Anton N, Mehta V. A survey to determine the understanding of the conceptual
21 basis and diagnostic tests used for brain death by neurosurgeons in Canada. *Neurosurgery*
22 2007;61:1039-1045; discussion 1046-1037.
- 23 85. Joffe AR, Anton NR, Duff JP, Decaen A. A survey of American neurologists about brain
24 death: understanding the conceptual basis and diagnostic tests for brain death. *Ann Intensive*
25 *Care* 2012;2:4.
- 26 86. Cranford RE, Smith DR. Consciousness: the most critical moral (constitutional) standard for
27 human personhood. *Am J Law Med* 1987;13:233-248.
- 28

- 1 87. Pellegrino ED. Personal Statement of Edmund D. Pellegrino, M.D. In: President's Council
2 on Bioethics, ed. Controversies in the Determination of Death. Washington, D.C.: President's
3 Council on Bioethics, 2008: 107-121.
- 4 88. Gómez-Lobo A. Personal Statement of Alfonso Gómez-Lobo, Dr. Phil. In: President's
5 Council on Bioethics, ed. Controversies in the Determination of Death. Washington, D.C.:
6 President's Council on Bioethics, 2008: 95-101.
- 7 89. Ropper AH, Brown RH. Adams and Victor's Principles of Neurology, 8th ed. New York:
8 McGraw-Hill, 2005.
- 9 90. Bernstein IM, Watson M, Simmons GM, Catalano PM, Davis G, Collins R. Maternal brain
10 death and prolonged fetal survival. *Obstet Gynecol* 1989;74:434-437.
- 11 91. Harders A. Neurosurgical Applications of Transcranial Doppler Sonography. New York,
12 NY: Springer-Verlag, 1986.
- 13 92. Plum F. Clinical standards and technological confirmatory tests in diagnosing brain death. In:
14 Youngner SJ, Arnold RM, Schapiro R, eds. *The Definition of Death: Contemporary*
15 *Controversies*. Baltimore, MD: Johns Hopkins University Press, 1999: 34-65.

16
17
18
19
20
21
22
23
24
25
26
27
28

PROOF OF SERVICE

I am a resident of the State of California, over the age of eighteen years, and not a party to the within action. My business address is AGNEW BRUSAVICH, 20355 Hawthorne Blvd., 2nd Floor, Torrance, California. On December 18, 2017, I served the within document **DECLARATION OF D. ALAN SHEWMON, M.D.**

- by transmitting via facsimile the document(s) listed above to the fax number(s) set forth below on this date before 5:00 p.m.
- by placing the document(s) listed above in a sealed envelope with postage thereon fully prepaid, in the United States mail at Torrance, California, addressed as set forth below:
- by placing a true copy thereof enclosed in a sealed envelope(s), and caused such envelope(s) to be delivered by hand delivery addressed pursuant to the document(s) listed above to the person(s) at the address(es) set forth below to be hand delivered at Court.
- by electronic service. Based on a court order or an agreement of the parties to accept service by electronic transmission. I caused the documents to be sent to the persons at the electronic notification addresses as set forth below:

AGNEW BRUSAVICH
SERIOUS INJURY LAWYERS
20355 HAWTHORNE BLVD · TORRANCE, CA 90503
T: (310) 793-1400 F: (310) 793-1499

14 15 16 17 18	Andrew N. Chang ESNER, CHANG & BOYER Southern California Office 234 East Colorado Boulevard, Suite 975 Pasadena, CA 91101 achang@ecbappeal.com	ASSOCIATE ATTORNEY FOR PLAINTIFFS LATASHA NAILAH SPEARS WINKFIELD; MARVIN WINIKFIELD; SANDREA CHATMANH; and JAH McMATH, a minor, by and through her Guardian ad Litem, LATASHA NAILAH SPEARS WINKFIELD (626) 535-9860 FAX (626) 535-9859
19 20 21 22	Thomas E. Still HINSHAW, MARSH, STILL & HINSHAW 12901 Saratoga Avenue Saratoga, CA 95070-9998 tstill@hinshaw-law.com	ATTORNEYS FOR FREDERICK S. ROSEN, M.D. (408) 861-6500 FAX (408) 257-6645
23 24 25 26	Richard Carroll CARROLL KELLY TROTTER FRANZEN McKENNA & PEABODY 111 West Ocean Boulevard 14 th Floor Long Beach, CA 90802	ATTORNEYS FOR DEFENDANT UCSF BENOIFF CHILDREN'S HOSPITAL (562) 432-5855 FAX (562) 432-8785
27 28	/// /// ///	

AGNEW BRUSAVICH
SERIOUS INJURY LAWYERS
20355 HAWTHORNE BLVD · TORRANCE, CA 90503
T: (310) 793-1400 F: (310) 793-1499


1 2 3 4 5 6	Scott E. Murray Vanessa L. Efremsky DONNELLY NELSON DEPOLO MURRAY & EFREMSKY A Professional Corporation 201 North Civic Drive, Suite 239 Walnut Creek, CA 94596-3879 <u>Smurray@dndmlawyers.com</u> <u>vefremsky@dndmlawyers.com</u>	ATTORNEYS FOR DEFENDANT JAMES PATRICK HOWARD, M.D., Ph.D. (925) 287-8181 FAX (925) 287-8188
7 8 9 10 11	Robert Hodges McNAMARA NEY BEATTY SLATTERY BORGES & AMBACKER, LLP 3480 Buskirk Avenue Suite 250 Pleasant Hill, CA 94523 <u>robert.hodges@mcnamaralaw.com</u> <u>karen.merick@mcnamaralaw.com</u>	ATTORNEY FOR ROBERT M. WESMAN, M.D. (925) 939-5330 FAX (925) 939-0203
12 13 14 15 16	Thomas J. Doyle Chad Couchet SCHUERING ZIMMERMAN & DOYLE, LLP 400 University Avenue Sacramento, CA 95825-6502 <u>tjd@szs.com</u> <u>ccc@szs.com</u>	ATTORNEY FOR DEFENDANT ALICIA HERRERA, M.D. (916) 567-0400 FAX (916) 568-0400
17 18 19 20	Kenneth R. Pedroza Dana L. Stenvick COLE PEDROZA LLP 2670 Mission Street, Suite 200 San Marino, CA 91108 <u>kpedroza@colepedroza.com</u> <u>dstenvick@colepedroza.com</u>	ASSOCIATE COUNSEL FOR FREDERICK S. ROSEN, M.D. and UCSF BENIOFF CHILDREN'S HOSPITAL OAKLAND (626) 431-2787 FAX (626) 431-2788

21 I am readily familiar with the firm's practices of collection and processing
22 correspondence for mailing. Under that practice, it would be deposited with the
23 U.S. Postal Service on that same day with postage thereon fully prepaid in the
24 ordinary course of business. I am aware that on motion of the party served,
service is presumed invalid if post cancellation date or postage meter date is
more than one day after date of deposit for mailing in affidavit.

25 (State) I declare under penalty of perjury under the laws of the State of
26 California that the above is true and correct.

27 (Federal) I declare that I am employed in the office of a member of the
28 bar of this court at which direction the service was made.

Executed this 18th day of December, 2017, at Torrance, California.


DEBBIE NAWA