In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 99-0625V

Filed: 23 November 2010

THOMAS KOLAKOWSKI, deceased, by His Parents, Jeffrey Kolakowski and Cathy Kolakowski, Petitioners, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	**PUBLISHED**[1] Thimerosal; Ethyl and Methyl Mercury; Hepatitis B Vaccine; Sudden Death; Toxicology; Cardiology; Pathology

Ronald Craig Homer, Esq., Conway, Homer & Chin-Caplan, Boston, Massachusetts, for Petitioner; *Ryan Daniel Pyles, Esq.*, United States Department of Justice, Washington, District of Columbia, for Respondent.[2]

PUBLISHED DECISION

ABELL, Special Master:

On 4 August 1999, Petitioners filed this Petition for compensation under the National Childhood Vaccine Injury Act of 1986 (Vaccine Act or Act)[3] alleging that, as a result of the

[1] Petitioners are reminded that, pursuant to 42 U.S.C. § 300aa-12(d)(4) and Vaccine Rule 18(b), a petitioner has 14 days from the date of this ruling within which to request redaction "of any information furnished by that party (1) that is trade secret or commercial or financial information and is privileged or confidential, or (2) that are medical files and similar files the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, "the entire decision" may be made available to the public per the E-Government Act of 2002, Pub. L. No. 107-347, 116 Stat. 2899, 2913 (Dec. 17, 2002).

[2] At trial, Petitioners were represented by Sylvia Chin-Caplan, with attorneys Thao Ho and Amy Fashano assisting. For Respondent, Linda Renzi, Althea Davis, and Ryan Pyles each represented Respondent on one of the three subject areas discussed.

[3] The statutory provisions governing the Vaccine Act are found in 42 U.S.C. §§300aa-10 et seq. (West 1991 & Supp. 1997). Hereinafter, reference will be to the relevant subsection of 42 U.S.C. §300aa.

Thimerosal contained within two Hepatitis B vaccinations administered to their son Thomas on 17 December 1998 and 20 January 1999, he died suddenly on 25 January 1999.

After having been filed, this case was grouped together with 24 other cases, all ultimately alleging that thimerosal in pediatric vaccines caused, contributed to, or triggered the death of the vaccinees. *See* Unpublished Scheduling Order, filed 28 July 2006. This case was selected as the "test case," to consider the theoretical mechanism of injury, by which the injury alleged (death) could be caused, as a general causation hearing in the context of trying one particular case. *See* Unpublished Scheduling Order, filed 12 May 2005.

Eventually, seven days of hearings on the ultimate issue of vaccine causation were convened by the Court, the first six held *in vivo* in Washington, the federal District of Columbia on 9, 10, and 11 June 2008, to hear Petitioners' case in chief, and on 24, 25, and 26 June 2008, to hear Respondent's witnesses (Petitioners also offered, on 26 June, rebuttal testimony). Hearing Transcripts 1-6. Due to the new evidence introduced during Petitioners' rebuttal testimony, a further hearing was held in Tampa, Florida on 15 January 2009. Transcript 7. Wherein, the Court heard from medical expert witnesses for both parties: toxicologists, cardiologists, and pathologists. Following those hearings, the parties filed closing briefs with the Court, and this matter is now ripe for a ruling.

As a preliminary matter, the Court notes that Petitioners in this case have satisfied the pleading requisites found in § 300aa-11(b) and (c) of the statute, by showing that: (1) they are the real party at interest as legal representatives of their son Thomas, the injured party; (2) the vaccine at issue is set forth in the Vaccine Injury Table (42 C.F.R. § 100.3); (3) the vaccine was administered in the United States or one of its territories; (4) no one has previously collected an award or settlement of a civil action for damages arising from the alleged vaccine-related injury; and, (5) no previous civil action has been filed in this matter. Additionally, the § 16 requirement that the Petition be timely filed has been met. On these matters, Respondent tenders no dispute.

The Vaccine Act burdens the Office of Special Masters with the duty[4] to make rulings and decisions on petitions for compensation from the Vaccine Program, to include findings of fact and conclusions of law. §12(d)(3)(A)(I). In order to prevail on a petition for compensation under the Vaccine Act, a petitioner must show by preponderant evidence that a vaccination listed on the Vaccine Injury Table either caused an injury specified on that Table within the period designated therein, or else that such a vaccine *actually caused* an injury not so specified. § 11(c)(1)(c).

I. FACTUAL RECORD

Despite their accord on certain factual predicates contained in the filed medical records, there is, unsurprisingly, a pronounced conflict between the parties on certain factual issues of viewing

[4] "Duty, then, is the sublimest word in our language. Do your duty in all things... You cannot do more, you should never wish to do less." From a letter attributed to General Robert Edward Lee, to his son, G. W. Custis Lee, dated 5 April 1852.

understood scientific mechanisms within the context of the expert witness testimony and the medical records. Considering these disputes and the Court's commission to resolve them, it behooves the Court to explain the legal standard by which factual findings are made.

It is axiomatic to say that a petitioner bears the burden of proving, by a preponderance of the evidence–which this Court has likened to fifty percent and a feather–that a particular fact occurred or circumstance obtains. Put another way, it is required that a special master, "believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the [special master] of the fact's existence." *In re Winship*, 397 U.S. 358, 371-72 (1970) (Harlan, J., concurring). Moreover, mere conjecture or speculation does not meet the preponderance standard. *Snowbank Enterprises v. United States*, 6 Cl. Ct. 476, 486 (1984).

This Court may not rule in favor of a petitioner based on his asseverations alone. This Court is authorized by statute to render findings of fact and conclusions of law, and to grant compensation upon petitions that are substantiated by medical records and/or by medical opinion. §§ 12(d)(3)(A)(i) and 13(a)(1).

Contemporaneous medical records are afforded substantial weight, as has been elucidated by this Court and by the Federal Circuit:

> Medical records, in general, warrant consideration as trustworthy evidence. The records contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium. These records are also generally contemporaneous to the medical events.

Cucuras v. Sec'y of HHS, 993 F. 2d 1525, 1528 (Fed. Cir.1993).

Medical records are more useful to the Court's analysis when considered in reference to what they include, rather than what they omit:

> [I]t must be recognized that the absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance. Since medical records typically record only a fraction of all that occurs, the fact that reference to an event is omitted from the medical records may not be very significant.

Murphy v. Sec'y of HHS, 23 Cl. Ct. 726, 733 (1991), *aff'd*, 968 F. 2d 1226 (Fed. Cir. 1992), *cert. denied sub nom. Murphy v. Sullivan*, 113 S. Ct. 263 (1992) (citations omitted), citing *Clark v. Sec'y of HHS*, No. 90-45V, slip op. at 3 (Cl. Ct. Spec. Mstr. March 28, 1991).

Special masters frequently accord more weight to contemporaneously recorded medical symptoms than those recounted in later medical histories, affidavits, or trial testimony. "It has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight." *Murphy v. Sec'y of HHS*, 23 Cl. Ct. 726, 733 (1991). *See also*

Cucuras v. Sec'y of HHS, 993 F.2d 1525, 1528 (Fed. Cir.1993). Memories are generally better the closer in time to the occurrence reported and when the motivation for accurate explication of symptoms is more immediate. *Reusser v. Sec'y of HHS*, 28 Fed. Cl. 516, 523 (1993). However, inconsistencies between testimony and contemporaneous records may be overcome by "clear, cogent, and consistent testimony" explaining the discrepancies. *Stevens v. Sec'y of HHS*, No. 90-221V, 1990 WL 608693, at *3 (Fed. Cl. Spec. Mstr., Dec. 21, 1990).

A. MEDICAL RECORDS *ET AL.*

The medical records in this specific case are rather limited and straightforward, and the Court summarizes them here, followed by the affidavit testimony of Mrs. Kolakowski, a Petitioner in this case:

Thomas Kolakowski was born 17 December 1998. His New Born Infant Record notes his general appearance as "vigorous and alert," his lungs as bilaterally clear, his heart as normal, and his reflexes (including the Moro (startle), grasping, sucking, swallowing, crying, and tonic neck reflexes) as normal, both at birth and at discharge. Pet. Ex. 3 at 2. His only clinical complaint was "mild jaundice."[5] *Id*. Before leaving the hospital nursery, Thomas was noted to have a "loud and lusty" cry. Pet. Ex. 3 at 11. In the cardiorespiratory criteria, Thomas was noted to have "normal breathing sounds," "normal heart sounds," "normal rhythm," and not to have "irregular rhythm." *Id*. Under the reflexes category, Thomas was observed to have intact sucking, grasping, and Moro (startle) reflexes, and to be free of tremors.[6] *Id*.

The day after he was born, before he went home from the hospital, Thomas received his first (of two) Hepatitis B vaccines on 18 December 1998. Pet. Ex. 3 at 13. Also on 18 December 1998, Thomas' "RN Assessment" displayed that Thomas responded to stimuli, had normal tone, showed reflex responses for rooting, sucking, grasping, and Moro (startle) reflexes, had no tremors, had normal breath sounds, and a regular apical[7] pulse, and had a loud and lusty cry. Pet. Ex. 3 at 20.

Thomas Kolakowski's pediatrician records indicate, in a 26 December 1998 record, that communication regarding his circumcision wound was recorded, as was the physician recommendation. Pet. Ex. 4 at 2. In the pediatrician records' notations for 24 December 1998, 26 December 1998, 2 January 1999, and 20 January 1999, there was no reference to tremors. *Id*. At a 20 January 1999 visit to the pediatrician, Thomas received his second of two Hepatitis B vaccinations. *Id*, Pet. Ex. 4 at 4. The next pediatrician records note that on 25 January 1999, the pediatrician received a call from the hospital emergency room that Thomas had arrived there

[5] Jaundice is "a syndrome characterized by hyperbilirubinemia and deposition of bile pigment in the skin, mucous membranes, and sclera, with resulting yellow appearance of the patient." DORLAND'S ILLUSTRATED MEDICAL DICTIONARY (30th ed. 2003) (SAUNDERS) at 963.

[6] A tremor is "an involuntary trembling or quivering." DORLAND'S, *supra*, at 1940.

[7] Apical means "pertaining to or located at the apex." DORLAND'S, *supra*, at 115.

"essentially dead via ambulance," and that on 1 February 1999, a notation of SIDS[8] was made. Pet. Ex. 4 at 2-3.

Emergency responder records note that on the morning of 25 January 1999, they responded to a call at the Kolakowski home to find Thomas in cardiac arrest, his parents frantic, and his father, Mr. Kolakowski, attempting cardio-pulmonary resuscitation (CPR). Pet. Ex. 5 at 2. They were told that Thomas was found by his parents in bed pulseless, and apneic,[9] despite having been previously healthy, and that his parents had last seen Thomas at 3:30 AM that morning. *Id*. The emergency responders' examination manifested that Thomas was pulseless, apneic, flaccid, and non-reactive, even though his skin was warm and dry, and there were no obvious signs of trauma. *Id*. They described mottling[10] and lividity,[11] and blood exuding from the mouth and nose. *Id*. After this assessment, they began treatment of CPR, gave him a dose of epinephrine,[12] ran electrocardiogram (EKG) diagnostics, and intubated him. *Id*. Thomas was asystolic[13] throughout their ride to the hospital, and he was transferred to the hospital staff still pulseless, apneic, and asystolic. *Id*.

Emergency room records corroborate that the above-referenced measures were in process when Thomas was brought in, and recorded that he was still asystolic, with apparent lividity. Pet. Ex. 6 at 1. He was unresponsive and pulseless, his skin was cool, dry, and cyanotic,[14] and showed lividity. *Id*. The physician's report from the emergency room indicates "Anterior[15] dependent lividity." Pet. Ex. 6 at 2. The narrative write-up of his case from the emergency department recorded a "normal birth history aside from being slightly large for dates" and the jaundice, "but was otherwise unremarkable." Pet. Ex. 6 at 4. Thomas was said to be "doing well, feeding well, and showing no signs of significant illness or failure to thrive." *Id*. In discussions had with Mrs. Kolakowski, she had "describe[d] him as having a small head cold or slight head cold, but nothing else was going on," the night before his death. *Id*. The same narrative reports that, "On arrival this child was lifeless, pulseless, asystolic in three leads with no spontaneous respiratory efforts and had

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[8] Sudden Infant Death Syndrome (SIDS) is "the sudden and unexpected death of an apparently healthy infant, typically occurring between the ages of three weeks and five months, and not explained by careful postmortem studies." DORLAND'S, *supra*, at 1833.

[9] Apnea is the "cessation of breathing." DORLAND'S, *supra*, at 115.

[10] Mottling is "a condition of spotting with patches of color." DORLAND'S, *supra*, at 1176.

[11] Lividity is "discoloration, as of dependent parts, by the gravitation of blood." DORLAND'S, *supra*, at 1060.

[12] Epinephrine, in this context, is "a synthetic preparation of the levorotatory form of epinephrine, used ... intravenously as a cardiac stimulant and vasopressor." DORLAND'S, *supra*, at 629.

[13] Asystole is "the absence of a heartbeat." DORLAND'S, *supra*, at 170.

[14] Cyanosis is "a bluish discoloration, especially of the skin and mucous membranes due to excessive concentration of deoxyhemoglobin in the blood." DORLAND'S, *supra*, at 455.

[15] Anterior means "1. situated in front of or in the forward part of an organ. 2. in humans and other bipeds, toward the belly surface of the body; called also *ventral*." DORLAND'S, *supra*, at 97.

signs of dependent lividity on arrival," adding that his temperature upon arrival was 35.8°C. *Id*. Even after the epinephrine administered by the EMTs, at the hospital Thomas' EKGs "continued to show only an agonal QRS complex[16] at best." *Id*. At the same hospital, by the request of the coroner, Thomas underwent a postmortem whole-body radiograph, which demonstrated "[a]lmost complete opacification of the lungs." Pet. Ex. 6 at 9.

Thomas death certificate lists "Immediate Cause (Final disease or condition resulting in death)" to be "Sudden unexplained death," without any listed underlying causes on the appropriate fields that follow. Pet. Ex. 7 at 1. In the field for "Other significant conditions contributing to death but not resulting in the underlying cause given in [Immediate and Underlying Causes section]," there is a notation of "Series II Hepovac." *Id*.

The records from the Forensic Pathology Department of Dauphin County Coroner's Office, completed in September of 1999, form an important facet of this case, and the Court quotes from them liberally here. They relate that, "Recently, he had a heptavax[17] on Wednesday, 1/20/99," but that, "Other than that, he was on no medications." Pet. Ex. 8 at 2. They recall the events of his last 24 hours as follows:

> Per the investigators, he had a rash on his face, which was felt to be infant acne, related to Dial Soap. He had been breast feeding twice per day. He was also taking two bottles of formula per day. On the day prior to his demise, he had not been eating well. Apparently, he had a lot of gas and an upper respiratory tract infection. The mother was able to nurse him in the early morning hours. When she awoke, she identified him essentially unresponsive....
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> The medical records were reviewed from childbirth. In addition, his neogene screening laboratory report accompanied the body. It showed no evidence of genetic abnormality...

Pet. Ex. 8 at 2.

On gross examination, Thomas was described as "moderately-built, moderately-nourished." Pet. Ex. 8 at 3. When weighed, his organs were the following weights: brain, 540g; heart, 30g; right lung, 100g; left lung, 80g. Pet. Ex. 8 at 3-4. His stomach contents were 2cc of brown/green particulate matter and fluid, and there was 30cc of bloody fluid in each chest cavity. Pet. Ex. 8 at 4. Among the findings from the examination of the head and central nervous system, there was "[m]ild to moderate edema[18] [] noted." Pet. Ex. 8 at 4. "Gross examination of the brain stem and

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[16] *See infra* at 40 *et seq*.

[17] Heptavax was a Hepatitis B virus vaccine manufactured by Merck, now replaced with Recombivax HB. *See* http://www.merck.com/product/usa/pi_circulars/r/recombivax_hb/recombivax_pi.pdf.

[18] Cerebral edema is "excessive accumulation of fluid in the brain substance; causes include trauma, tumor, and increased permeability of capillaries as a result of anoxia or exposure to toxic substances." DORLAND'S, *supra*, at 589.

cerebellum was unremarkable." *Id*. "The hippocampi[19] and pineal gland[20] showed no abnormalities." *Id*. Aside from the certain findings, Thomas' organs were generally "of intrinsically normal size, morphology, and anatomic position for this age and sex individual." *Id*. Among these findings were "[f]ine petechial hemorrhages[21] [] noted in the left ventricle,"[22] although "there was no evidence of asymmetry, atrophy, hypertrophy, or dilation" of the heart, and although the myocardium[23] was examined, no adverse or abnormal findings concerning it were recorded. *Id*. In the lungs, "The lung parenchyma[24] showed diffuse consolidation throughout." Pet. Ex. 8 at 5. Apparently in addition to the brown/green material noted above, the notes on the gastrointestinal system report that, "There was a bit of dark-colored fluid, perhaps blood, noted in the stomach, but on further examination, there was no evidence of ulcerations, lacerations, or obstruction noted in the area. *Id*.

The opinion given by the medical examiner was that, "After autopsy and review of the history, it is my opinion that the cause of death was **Sudden Death in Infancy**. The manner of death was **Natural**." Pet. Ex. 8 at 7 (emphasis in original). He elaborated:

> This case has been reviewed by Neuropathologists, a pediatric pathologist and three forensic pathologists (1 pending pathologist at this dictation). There are no findings that indicate an obvious anatomic cause of death. At present we can not [*sic*] draw a conclusion as to whether or not Heptavax is causally connected to this child's death. We do feel, however, that this issue should be examined further.

Pet. Ex. 8 at 7.

Finally, the Court turns to the two affidavits of Thomas' mother, Mrs. Kolakowski. The first was executed on 3 January 2000. Pet. Ex. 1 at 4. *Inter alia*, she recollects that she and Mr. Kolakowski had queried the pediatrician, at a visit on 21 December 1998, regarding "some movements Tommy was making that we were unsure of," which she described as "tremors," and which she "had noticed ... since the day [she] brought [Thomas] home from the hospital." Pet. Ex.

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[19] The hippocampus is "a curved elevation of gray matter extending the entire length of the floor of the temporal horn of the lateral ventricle." DORLAND'S, *supra*, at 853.

[20] The pineal gland is "a small flattened cone-shaped body in the epithalamus, lying above the superior colliculi and below the splenium of the corpus callosum." DORLAND'S, *supra*, at 774.

[21] A petechial hemorrhage is a "hemorrhage from capillary leakage at minute points beneath the skin, mucous membrane, or serosal surface." DORLAND'S, *supra*, at 834.

[22] The left ventricle of the heart (*ventriculus sinister cordis*) is "the cavity of the heart that propels the blood out through the aorta into the systemic arteries." DORLAND'S, *supra*, at 2031.

[23] The myocardium is "the middle and thickest layer of the heart wall, composed of cardiac muscle." DORLAND'S, *supra*, at 1212.

[24] Parenchyma (from the Greek for "anything poured in beside") refers to "the essential elements of an organ; in anatomical nomenclature this refers to its functional elements as distinguished from its framework, the stroma." DORLAND'S, *supra*, at 1371.

1 at 2. She recalled that the pediatrician "assured [her] that they were from the normal development of the nervous system." *Id.* She continued:

> Little by little, I noticed other changes in Tommy that I was uneasy about. He seemed to lack the normal startle reflex to loud noises and continued having the body tremors. He also had very loose bowel movements that the doctors attributed to the fact that he was breast fed....
>
> My mother-in-law noticed these tremors too and asked me if I had mentioned them to the doctor. So, I again questioned the pediatrician with a phone call made on December 26, 1998, concerning the body tremors. I was again given the same explanation. This phone call was not recorded in Tommy's medical records.

Pet. Ex. 1 at 2. At the visit when the second Hepatitis B vaccine was administered, Mrs. Kolakowski says she reported that Thomas had diarrhea, was not feeding well, did not wish "to sleep or nurse for any length of time and seemed very passive." Pet. Ex. 1 at 3. She says the doctor's response was to allay her concerns because Thomas did not have a fever ("although," she says, "they did not check for this"), and that her concerns were not recorded in the medical records. *Id.* She recalled that, following that visit, "Tommy's diarrhea seemed worse but because he was still eating and urinating there was nothing I could do." *Id.*

Mrs. Kolakowski described her recollection of Thomas' last 24 hours:

> By Sunday, January 24, 1999, Tommy was still not acting right. I knew he was not feeling well and he still had the tremors, which he would have almost daily. We all went to bed that night and Tommy awoke between [3:00 and 3:30 AM] for a feeding. I let him lay [*sic*] with me after that feeding. When I awoke at approximately [7:00 to 7:15 AM], I discovered my son in my arm not breathing. There was blood coming from his nose and mouth and blood on the sheets....

Pet. Ex. 1 at 3. She added that later, after Thomas' funeral, she recalls Mr. Kolakowski relating that before he could attempt CPR with Thomas the morning of 25 January 1999, "he had to flatten out [Thomas'] tongue because it was all balled up." *Id.*

In preparation for the hearings, Mrs. Kolakowski was ordered to compose and file another affidavit, describing clearly, by physical description and not medical terminology, the phenomenon she had described as tremors. Wherefore, Petitioners filed Pet. Ex. 35, the supplemental affidavit of Cathy Kolakowski, on 22 October 2007. Mrs. Kolakowski admitted therein that, it having been nine years since Thomas had died, it was "difficult to clearly remember the few weeks that Tommy was with us." Pet. Ex. 35 at 1. She proceeded to be as specific as she could:

> I noticed Tommy to have intermittent involuntary movements of his extremities and torso from the time he was a few days old. These involuntary movements are what I called tremors in my first affidavit. He had episodes where his body would stiffen and then the involuntary movements would begin[, at which point,] I would be unable to reposition him to sooth[e] him. These movements lasted briefly, and would be over in minutes. When his extremities would tremble, I would try to hold his hand or comfort him. Despite what I did, the trembling would continue and stop when it

> had run its course.... He also had times when he would stare at a fixed area. Despite trying to distract him, he would continue to stare. During these staring episodes, I noticed that he would not blink. Eventually he would stop staring, but nothing I did would interrupt the staring.

Pet. Ex. 35 at 1-2.

Based on this testimony, Petitioners retained a well-received pediatric neurologist, with much experience testifying before this Court, Dr. Kinsbourne, to opine regarding a differential diagnosis of Thomas' "tremors" thus described. Dr. Kinsbourne stated in his report:

> The "tremors" could represent jitteriness or seizures. Jitteriness is frequently and exclusively observed in the newborn. The movements are generalized, symmetrical, rhythmical and equally alternating. They are amplified by sudden stimuli and can readily be abolished by gentle physical restraint of the child. They occur in children who have suffered a variety of generalized brain insults, but also in children who are free from any known impairment. Neonatal seizures can generate "tremors" which are "clonic", jerking that has alternating fast and slow components. They usually have associated deviation of gaze. Sensory stimulation and restraint are ineffective in modifying the movements in a clonic seizure. Staring fixedly can occur as a seizural phenomenon, and cannot be arrested by stimulating the child. However, "nonepileptic seizures" also occur in the neonatal period. These are seizure-like behaviors which however are unaccompanied by correlated paroxysmal features...
>
> Thomas' movements [as described] have some of the properties of neonatal seizures, notably their resistance to stimulation and to restraint, and they would have justified electrophysiological study. The same applies to the "staring" spells. However, seizures can be difficult to differentiate from the wealth of other movements that newborn children often make. In the absence of a more precise characterization of the movements, and of EEG[25] confirmation, I cannot arrive at an opinion to a reasonable medical probability as to their exact nature.

Pet. Ex. 36.

B. Testimony Bearing on Entitlement

The Court heard oral testimony from seven different experts, some of them more than once, over the course of weeks and months. The Court here summarizes what it views as the relevant portion of that testimony, but itself reviewed all of the testimony provided.

[25] An electroencephalogram is "a recording of the potentials on the skull generated by currents emanating spontaneously from nerve cells in the brain.... Fluctuations in potential are seen in the form of waves, which correlate well with different neurologic conditions and so are used as diagnostic criteria." Dorland's, *supra*, at 596.

1. George W. Lucier, Ph.D.

The first day of hearings presented the testimony of Petitioner's expert in toxicology, Dr. Lucier.[26] Transcript of Hearing on 9 June 2008 (Tr. 1) at 33. Dr. Lucier has enjoyed a long career of public service as a toxicologist, during which time he has served as researcher, research team leader, manager, inter-agency coordinator, colloquium moderator, and liaison.

Dr. Lucier's graduate school work was in insecticide toxicology at the University of Maryland's School of Agriculture, coming on the heels of the publication of Rachel Carson's book on DDT, *Silent Spring*. Tr. 1 at 8. His Ph.D. also specialized in insecticide toxicology, again at the University of Maryland, and included a minor in Chemistry as well. *Id*. His dissertation was on "the new breed of organophosphate pesticides." Tr. 1 at 8-9. However, he does not possess "a degree that is formally recognized by the American Board of Medical Toxicologists," which would require licensure as a medical doctor. Tr. 1 at 136. After completing his Ph.D., he began his thirty-year career at the National Institutes of Health (NIH), beginning as a staff fellow in the Laboratory of Cell Biology, and studying how the body metabolizes and ultimately excretes a variety of chemicals. Tr. 1 at 9-10. Dr. Lucier's research experience in that capacity taught him that clearance is not directly linked to age, that there are some chemicals that a newborn could metabolize faster than an adult, and vice-versa. Tr. 1 at 11. His area of research moved to receptor systems in the body. Tr. 1 at 11-12. Due to the areas of his research, he became increasingly involved in the study of chemical exposure risk management planning in the federal government, culminating in becoming associate director of the National Toxicology Program and a director of the Toxicology Program at the National Institute of Environmental Health Sciences. Tr. 1 at 12.

In the former of those capacities, his responsibility was "to coordinate toxicology research and testing across the federal agencies for compounds of interest in which toxicological information was needed." Tr. 1 at 16. In this pursuit, Dr. Lucier felt it his responsibility to encourage laboratory *in vitro* studies, the results of which could be used by federal regulatory authorities to estimate toxicology standards. Tr. 1 at 19. He favored *in vitro* studies because "[m]echanism data provides the biological plausibility or response that might occur.... epi[demiologic] studies are notoriously insensitive, and ... animal [studies are] often criticized for high doses." Tr. 1 at 20.[27] Dr. Lucier continued by explaining his involvement with the study of mercury's relative toxicity. He had served as chair of a group formed to synchronize "risk assessments across federal agencies for methyl mercury,"[28] with the goal of "a common risk assessment across these federal agencies." Tr. 1 at 23.

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[26] Respondent stipulated to Dr. Lucier's expertise in the area of toxicology, and the Court accepted his expert testimony bearing on toxicology. Tr. 1 at 33-34.

[27] He explained that the World Health Organization and the Environmental Protection Agency both accept this form of research on toxicology, as did the National Toxicology Program under his helm. Tr. 1 at 20-21.

[28] He also explained the concept of biological magnification through the food chain that makes eating fish a risk for mercury consumption. Tr. 1 at 24-25.

Methyl mercury has been known to be a neurotoxin since the 1950s;[29] the problem became settling on a "safe" level. Tr. 1 at 25. Dr. Lucier has authored approximately 250 articles, but only a couple of those were on mercury, and those were early in his career.[30] Tr. 1 at 27.

Dr. Lucier next discussed the result of his efforts to reach a consensus on safe mercury levels among the various federal authorities. Unsurprisingly, the agencies differed on which studies and which data were most persuasive, and what the data implied. Tr. 1 at 31-33. There were divergent results from a few studies in the Faroe and Seychelles Island populations, which all agreed were properly performed, notwithstanding the difference in their findings. Tr. 1 at 33. Dr. Lucier's role was as an organizer, not as a researcher or as an analyst of the different data sets. *Id*.

Limitations in logistics, funding, and time required federal researchers to perform safety level testing on only representative samples of larger chemical groups, testified Dr. Lucier, and it was his purview "to make an initial decision about what should be tested." Tr. 1 at 44-45. When it came to the grouping of organomercurials, there already existed sufficient data indicating that methyl mercury was a neurotoxin, whereas little was known about the potential toxicity of ethyl mercury. Tr. 1 at 45. Therefore, to assess ethyl mercury's toxicity, Dr. Lucier opted to use the data for methyl mercury, inasmuch as "they were structurally similar, differing only in the methyl group[, b]oth form the same major metabolite, inorganic mercury[, and b]oth were neurotoxicants, so it seemed like a very good surrogate." Tr. 1 at 45. He reported the consensus to employ this process of extrapolation was widespread, as it was a common practice. Tr. 1 at 46.

This extrapolation was based on the presumed similarity between ethyl mercury, the organomercurial that forms a component of thimerosal, and methyl mercury. Dr. Lucier communicated his understanding of the medical literature extant, stating that the symptoms associated with methyl mercury toxicity are identical to those experienced with ethyl mercury: "tremors, convulsions, ataxia, all these kinds of things that have been seen with high levels of methyl mercury exposure." Tr. 1 at 37-38. Among the few differences he noted were that ethyl mercury–more so than methyl mercury–has been associated with electrocardiographic alterations by the poisoning studies from Minamata Japan, and cardiovascular effects as discussed in the Cinca paper. Tr. 1 at 38. Both methyl and ethyl mercury are "organomercurials," also referred to as "alkyl mercurials." Tr. 1 at 40. The significance of this fact to Dr. Lucier is that although "organomercurials have considerable stability in the environment," they are slowly metabolized in the human body, producing inorganic mercury (specifically mercuric chloride, $HgCl_2$) as the metabolite (i.e., the end product of that process). Tr. 1 at 41. This is true of both methyl and ethyl mercury. *Id*. The difference between the two, said Dr. Lucier, is that "in the human body that de-

[29] He later added that ethyl mercury had also been found to be a neurotoxin as well, based on data from Iraq, Ghana, and Romania; however, he did not reference where in the filed exhibits those data could be found, and Petitioner did not introduce them. Tr. 1 at 35-36.

[30] As he put it, "Most of my work with mercury was involved in chairing the committees that I talked about.... So most of my work with mercury has been involved with a risk assessment of it, not performing bench-related research, although I did have a couple articles [that] were pretty much mechanistic articles looking at how mercury affected different kinds of cytochromes." Tr. 1 at 27.

ethylation reaction occurs faster for ethyl mercury than it does for the corresponding de-methylation for methyl mercury ... [i]t's a greater rate of formation of inorganic mercury from ethyl mercury than there is from methyl mercury, although it's the same metabolite once formed." Tr. 1 at 42. Dr. Lucier stated that, inasmuch as thimerosal is composed of nearly 50% ethyl mercury, the body's metabolism of thimerosal would result, in part, in that same metabolite, in a chemical process quite the same as would occur with methyl mercury. Tr. 1 at 42-43. Indeed, he added, both forms of organomercurials are toxic. Tr. 1 at 43. Later, he elaborated more specifically on this point:

> [T]he generally accepted mechanism of action is that they affect sulfhydryl[31] groups. They affect a lot of enzymatic pathways, a lot of cellular macromolecules. They affect glutathione[32] levels that regulate a lot of clearance mechanisms for other toxic chemicals, so they are broad spectrum toxic and they affect many, many different organs.

Tr. 1 at 115.

When it came to evaluating ethyl mercury's toxicity on the basis of methyl mercury studies,[33] the question arose as to what data sets to use from the extant methyl mercury toxicity research. One researcher in the field was a Dr. Grandjean, who oversaw research on the Faroe Island population:

> The big question that quickly arose when the Grandjean studies were published is are they credible? Are they useful for risk assessment purposes? Are they solid enough studies to do that? You just don't want to take any study when you do risk assessment.
>
> ...[W]e did something far, far more rigorous than a scientific review. We actually asked the authors for their raw data so it could be analyzed by independent statisticians, independent epidemiologists, independent pharmacologists, independent neurobehavioral toxicologists.... I organized that and chaired that activity to determine whether or not the Seychelles studies and the Faroe Island studies were credible studies. The conclusion was that they were. There were some possible reasons why they were different, but the conclusion of that quite distinguished panel of independent scientists was they were both good studies.

Tr. 1 at 48-49.

Dr. Lucier summarized the Faroe Islander and Seychelles Islander series of studies, stating that they analyzed neurological effects sustained by islanders who ate fish contaminated with methyl mercury, using analytical methods employed in the Iraqi studies. Tr. 1 at 38-39. The Seychelles

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[31] Sulfydryl is "the univalent radical, —SH." DORLAND'S, *supra*, at 1791.

[32] Glutathione is "a tripeptide" that "functions in various redox reactions, such as the destruction of peroxides and free radicals, the detoxification of harmful compounds, and activity as a cofactor for enzymes." DORLAND'S, *supra*, at 784.

[33] Weighing the differential attributes between ethyl and methyl mercury, Dr. Lucier concluded that they are "equally toxic." Tr. 1 at 127.

studies did not demonstrate measurable negative neurological effects. Tr. 1 at 39. The mercury ingested in the fish remained at a relatively low dose level. *Id*. Dr. Lucier contrasted the Seychelles studies with the Faroe Islands studies conducted by Phillippe Grandjean *et al.* The subjects of the Faroe studies had consumed whale meat contaminated with methyl mercury. *Id*. Neurobehavioral assessments indicated negative effects related to sensation and motor ability, and cognitive deficits were noted in children whose mothers had eaten the mercury-tainted whale meat while pregnant. Tr. 1 at 39-40. Dr. Lucier also stated that similar types of problems were also seen in an unnamed New Zealand study, where mercury intake was at lower doses. Tr. 1 at 40.

Dr. Lucier discussed the disparity in results between the Faroe Island and Seychelles Islands studies, noting that, even though his team examined the studies' methods and data sets, they could not resolve or explain the disparity, except to identify some variances in the subjects of the studies:

> [One,] the ages were different. The children were younger when they were evaluated in the Seychelles study than the Faroe study, and the younger the children are the more variation there are in the results because of normal developmental differences, so that was one possible explanation, the age at which the measurements were made.
>
> Two, there were different neurobehavioral measurements made in both studies. The Grandjean studies [in the Faroes] were domain specific, and the Clarkson [studies in] the Seychelles were global measures of neurobehavioral function, so there were a different set of measurements made.
>
> A third possible difference was that in the Faroe Island it was more of an episodic kind of exposure. You know, you don't eat necessarily whale meat every day, [but an occasional] big meal of it, and then might not eat it again for another few weeks and so the exposure was more episodic whereas in the Seychelles they were eating some fish every day. The whale meat was more contaminated, but the people didn't eat it as often, so a difference in the patterns of exposure I guess that could have accounted for some of the differences.
>
> ...A job of that workshop was to determine whether or not those studies are credible and whether the Faroe Island study could be used in risk assessment. The conclusion was it could be because it was a solid study, but we couldn't explain why it was different results from the Seychelles studies.

Tr. 1 at 61-62. To circumvent the negative data set in favor of the positive data set, to show that "the Seychelles population might be a bit different in how they handle mercurial compounds," Dr. Lucier referenced a table from Pet. Ex. 18, Tab HHH, at 320, which list variables such as "toxicodynamic variability," "nutritional deficits," and "co-exposure to other neurotoxicants" (including other forms of mercury) as "sources of uncertainty." Tr. 1 at 64-66. Of course, the same could just as easily be said about the Faroe Island population.

The conclusion of the National Academy of Sciences, in choosing between the two to settle on a reference dose for risk assessment purposes, was to use "a positive study that was well conducted. In other words, you have real data in which you can determine benchmark doses." Tr. 1 at 67. This choice is based on the rationale that, "You certainly wouldn't want to take a negative

study because you have to assume that a well done positive study, the effects are in fact real." Tr. 1 at 67. Therefore, the NAS used the Grandjean–Faroe Islands data as the basis for their risk assessment.

Dr. Lucier discussed in some detail some of the multifarious factors that account for the variability in mercury toxicity within the population, including age, sex, and genetic predisposition:

> Age is one.... [D]eveloping systems, whether it be the nervous system or any system, are more sensitive to risk because that organ is developing and differentiating at a critical stage where, if you disrupt it, it will never become right, and this is a well-known tenet of developmental and reproductive toxicology. So there would be critical windows for each organ system where they would be most sensitive to toxic effects. ...[T]he nervous system development is spread out. It starts *in utero*, certainly, but continues on into development. So you have processes like synaptogenesis, cell migration, myelinization that are going on for years after the baby is born. So for neurotoxicants, exposure as an infant is a critical window.
>
> ...Gender[34] is another. There are common gender differences in risk that males or females might be more sensitive to risk than we know.
>
> ...[Examining the different results of the Faroe and Seychelles studies] indicate[s] all of the factors that are involved in diet, causing differences in response.
>
> ...Genetic differences ... you have genetic predisposition to the toxic effects [of] many chemicals, and this is the reason why drug companies now are getting genetic screenings, genetic tests, to try to determine which of their patients might be adversely affected by drugs.... Mercury is no different. There are genetic predispositions.
>
> ...Differences in clearance rate. I've seen a number of numbers from [methyl] mercury. There aren't as many numbers for ethyl mercury, but the difference seems to be -- that one paper I looked at had a difference between a 36- and 189-day half-life for methyl mercury. This is just looking at blood; never mind the other compartments. So that's a fivefold difference just by itself.
>
> ...Existing body burdens of mercury are already high in some people [i.e., previous exposure level and individualized response thereto].

Tr. 1 at 120-123.

A significant factor that bears more emphasis than Dr. Lucier placed thereupon was what was meant by a "safe" level, and what that would mean for a potential "dangerous" level. When asked what standard was used to determine a safe level, Dr. Lucier stated as follows:

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[34] Gender is a linguistic and metaphysical term, not a biological or physical term, a regrettably common misunderstanding and error. David Lee Mundy, *Hitting Below the Belt: Sex-ploitive Ideology & the Disaggregation of Sex and Gender*,14 REGENT U. L. REV. 215 (2002); Richard A. Epstein, *Gender is for Nouns*, 41 DEPAUL L. REV. 981, 982-83 (1992).

> [A]t this time the risk assessment practices at EPA were changing. The traditional risk assessment approach was to identify a LOEL, a lowest observed effect level, or a NOEL, no observed effect level, so NOEL/LOEL, no observed effect level -- this was from animal studies -- or a LOEL. Then you use safety factors[35] to lower that dose. For example, if you only had LOEL data and you didn't have a NOEL endpoint you'd have to put a safety factor in to account for that. That might be 10. You'd have to account then for the fact that humans might be more sensitive than the species being tested. You might put another factor of 10. You would then have to account for interindividual variation, variation among the population.... They have to put that in there too, so you could have quite an accumulation of safety factors. It was that approach that EPA had used in their report to Congress using the Iraqi data. I can't remember all the exact numbers, but it was that approach that they used and ultimately got down to recommended RFD, so-called virtually safe exposure level, of .01 micrograms methyl mercury per kilogram per day.

Tr. 1 at 51-52. This approach led the EPA to reach a much lower safe dosage–0.1 micrograms per kilogram per day–than did other governmental entities, such as the Toxic Substances Disease Registry of the Centers for Disease Control (0.6 micrograms per kilogram per day), which had relied on the data from the Seychelles study, or the Food and Drug Administration (0.4 micrograms per kilogram per day), which, Dr. Lucier said, was based on adult data. Tr. 1 at 53. The National Academy of Sciences tried to integrate those safe level assessments from those agencies to arrive at a risk assessment "benchmark dose," which is a dose at which some statistical interval, measured from a percentage of the population with a physical response to the dose (*e.g.*, a dose at which 5% of the population would evidence a response). Tr. 1 at 54-55. Using the data from cognitive testing performed on the subjects of the aforementioned studies, they performed a series of estimations and calculations to translate to a blood level of 58 micrograms per liter of blood as the benchmark dose; from that they then applied a safety factor of 10 to further insulate a safe level, so as to account for differences between individuals, resulting in a "safe" dose of 5.8 micrograms per liter of blood. Tr. 1 at 55-56.

Dr. Lucier made a curious and unexplained conversion of this data. He stated that the National Academy of Sciences' safe dose, measured as a blood level of 5.8 micrograms per liter of blood, corresponds to the EPA's safe intake dose of 0.1 micrograms per kilogram per day, as an equivalent measure, which would then confirm the accuracy of one another through corroboration. Tr. 1 at 56. However, Dr. Lucier did not elaborate how an intake dose could ever be equivalent to a blood level dose in evaluating toxicology of a compound, especially after he had made so much of the differences between individuals in neurological sensitivity and disparities in absorption and clearance rates into and out of the bloodstream. That is to say, if how the compound is delivered to

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[35] Dr. Lucier explained the use of safety factors (customarily a single power of ten) in "safe dose" research. In moving from experimental data to guidelines for humans, be it from a mechanistic (*e.g.*, cell) study, or an animal study, the safe level from the experimental data is factored by a power of ten to account for the possibility that humans are more sensitive than the experimental subject. Tr. 1 at 57. Likewise, for each unknown variable that differentiates the public from the known, quantified data, a safety factor is applied (*e.g.*, age/developmental differences, general variability within the population). *Id*.

the body, the efficiency by which the body moves that compound from its point of delivery into the bloodstream, the relative speed that the compound is metabolized, and the relative speed that the compound is excreted from the blood stream are all variables affecting the blood level of a compound, how can an intake dose ever correspond linearly with a blood level amount across a population? If there is an answer to this question, Dr. Lucier did not explain it.

Dr. Lucier testified that the reference dose chosen by the EPA was "meant to be a reference dose for women of child bearing age." Tr. 1 at 58. It is difficult to extrapolate to a safe level for infants due to a lack of data, because infants do not typically eat foods with methyl mercury in them, as older children and adults might, and they are not affected by the adult diet so directly, as unborn children *in utero* are. Tr. 1 at 58.

Dr. Lucier brought attention to a puissant difficulty in studying or demonstrating a link between organomercurials and a given injury:

> Mercury poisoning is not manifested immediately after exposure.... That means when you administer mercury into an animal or whether it's an accidental exposure the effects aren't seen right away. The effects are delayed. You see this in all animal studies. They can be delayed for a considerable period of time. In the case of developmental studies, the exposure *in utero* may not be manifested until later in life. This is well known for a lot of chemicals where you get *in utero* exposures. Imprinting effects. There's other kinds of effects that are not expressed sometimes until adulthood for some chemicals.

Tr. 1 at 66, 68.

A seminal article of medical literature relied upon by Dr. Lucier was Pet. Ex. 18, Tab M, Thomas M. Burbacher *et al.*, *Comparison of Blood and Brain Mercury Levels in Infant Monkeys Exposed to Methylmercury or Vaccines Containing Thimerosal*, 113 (8) ENVIRONMENTAL HEALTH PERSPECTIVES 1015-21 (August 2005). Dr. Lucier summarized the study, stating that the team of Dr. Burbacher, who has been researching organomercurial toxicity for over twenty years and is well known in his field, compared mercury levels in the blood and brain 2-28 days after either oral gavage[36] of methyl mercury or intramuscular injection of four vaccines containing thimerosal. Tr. 1 at 70. Dr. Lucier summarized their findings, among which was that both forms and delivery methods led to mercury infiltrating both the blood and the brain. Tr. 1 at 71. Another finding was that "ethyl mercury appeared to be cleared faster than methyl mercury from the blood because that's what their clearance data is, predominantly in the blood," but that, comparing ethyl to methyl mercury, "[t]here was an apparent greater rate of conversion of organic mercury into inorganic mercury." *Id*. The four thimerosal-including vaccines were administered to the subjects weekly, "so it wasn't exactly like a vaccine schedule." Tr. 1 at 73.

Although the Burbacher study only recorded mercury levels for 28 days, Dr. Lucier reported that "inorganic mercury had an infinite half-life in the brain." Tr. 1 at 72. Therefore, Dr. Lucier

[36] Gavage (from the French for "cramming") is "1. forced feeding, especially through a tube passed into the stomach. 2. the therapeutic use of a very full diet; superalimentation." DORLAND'S, *supra*, at 760.

explained, a linchpin in the Burbacher study is the relationship between organic mercury intake and persistent inorganic mercury levels:

> The organic mercury does apparently have a clearance rate from the brain. The inorganic mercury does not. So ... right after exposure the organic mercury is slightly higher in the brain than inorganic mercury. If you look 28 days afterwards, the inorganic mercury is clearly higher, and that may be because -- again, this is speculation by Burbacher and others as well as myself, but this is presumably because the ethyl mercury in the brain is being dealkylated there to form inorganic mercury, and once inorganic mercury is in the brain it doesn't seem to be able to escape.

Tr. 1 at 72.

Dr. Lucier wanted the Court to focus on how the initial mercury measurements (taken on day two of the regimen) were similar as between the subjects who orally received methyl mercury and those who received ethyl mercury by injection (each having a level around 10 ng/ml). Tr. 1 at 73. However, a glance at those tables reveals that the several vaccine doses (administered over four weeks) did not raise this initial level over 16 ng/ml, whereas the oral intake of methyl mercury raised the subjects' mercury levels steadily higher, to between 30 and 40 ng/ml. Pet. Ex. 18, Tab M at 1018. The fact that the initial reading on day two was equivalent would suggest to the Court a similar starting point between the test groups, not that the ethyl mercury delivered by vaccine was as potent as the ingested methyl mercury in forming inorganic mercury in the brain.

Dr. Lucier also focused on the fact that the Burbacher study's measurement of blood levels and recordings on clearance from the blood did not track where the mercury went when it left the bloodstream: "It may be anywhere in the body. It may be excreted. It may be in the body. It may be in the brain. It may be in the heart. All they know is it's not in the blood." Tr. 1 at 74. The Court found it remarkable then that Dr. Lucier's next statement was that, "In fact, obviously it stays in the brain because that has an infinite half-life. If you look at the rate of loss of wash out of total mercury that's disappearing pretty fast. It doesn't disappear fast from the brain. It doesn't disappear at all." *Id*. Although the connection is unclear and remained unexplained, it would seem that Dr. Lucier was saying that the mercury in the bloodstream went to the brain, and this immediately after stating that it is unknown where the blood-borne mercury goes when it leaves the bloodstream.

Also, Dr. Lucier stipulates that the ethyl mercury had a quicker clearance rate than did the methyl mercury, which lingered in the bloodstream longer. Tr. 1 at 75. However, he then describes the clearance data charts to conclude, "the degree of accumulation of inorganic mercury in the brain is 10 times greater from the thimerosal treatment from an equivalent dose or starting point than it is for the methyl mercury treatment. Ten times on order of magnitude more inorganic mercury in the brain following thimerosal than it would be for the methyl mercury." Tr. 1 at 77. Dr. Lucier's statements do not jibe with the Burbacher article, however, which summed up the data on this point as follows. The regression estimate for methyl mercury half life is "58.4 $\pm$ 25.0 days (r = 0.57);" however, "[t]he half life of inorganic Hg is too long (> 120 days) to be accurately estimated from the present data (i.e., r is not significantly different from 0)." Pet. Ex. 18, Tab M at 1018. In comparison, the regression estimate for ethyl mercury half life is "14.2 $\pm$ 5.2 days (r = 0.76);"

likewise, similar to the other data set, "[t]he half life of inorganic Hg is too long (> 120 days) to be accurately estimated from the present data (i.e., *r* is not significantly different from 0)." Pet. Ex. 18, Tab M at 1019. Moreover, if anything, the semilogarithmic plot of the inorganic mercury washout falls more significantly within the ethyl mercury data group than in the methyl mercury data group, where it stayed more closely constant. Compare *Id*. at page 1018, Figure 4 with *Id*. at page 1019, Figure 7. The Court does not understand how Dr. Lucier can seriously argue that there is a ten-fold greater retention rate of inorganic mercury with ethyl mercury as compared to methyl mercury, based on this data.[37]

All the while, Dr. Lucier's statements seem to assume that the inorganic mercury levels in the Burbacher test subjects were the result of the organic mercury treatments, and rose on a scale commensurate with each mercury exposure:

> Q Okay. And would this data that you've just cited support the fact that the inorganic mercury from the administration of thimerosal in the immunized monkey persisted?
>
> A Yes.
>
> Q Even after 30 days there was virtually no change?
>
> A Yes. So in other words, if you look at this data you have a flat line on the inorganic mercury, so if you had two injections of thimerosal four weeks apart the second injection in terms of inorganic mercury in the brain would be additive to what was already there. So if you have a second injection 28 days later you would double the amount of inorganic mercury in the brain. That's pretty much what that data is saying.

Tr. 1 at 78. However, without testing a set of control subjects who had not received the mercury treatments, there is no support for the contention that levels of inorganic mercury (which remained

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[37] Dr. Lucier's argument seems to have been based on figures 2, 4, 5, and 7 on pages 1018 and 1019 of the Burbacher article, depicting the blood mercury levels and washout rates of methyl and ethyl mercury respectively. On the last day that mercury was administered (day 28 of the study), the blood level for methyl mercury value was 38 ng/ml, and 28 days after that (day 56 of the study), the brain level of inorganic mercury was at an average level of 4 ng/g (based on two data points, one at 8 and the other at 1). However, based on earlier values, the best fit line at that point has a value of 8 or 9, not 4. Correspondingly, on the last day that mercury was administered (day 28 of the study), the blood level for ethyl mercury value was 11 ng/ml, and 28 days after that (day 56 of the study), the brain level of inorganic mercury was at an average level of 11 ng/g (based on 4 data points, two each just above and below that value). Dr. Lucier's argument (which he did not explain well at all) seems to be that the much higher blood level for methyl mercury corresponds to a lower brain level of inorganic mercury, whereas a comparatively lower blood level of ethyl mercury corresponds to a comparatively higher brain level of inorganic mercury. See Tr. 1 at 76-77. What this argument ignores is that inorganic mercury in the brain was statistically pretty constant in both the methyl and the ethyl mercury groups, and did not seem responsive to the intake of organic mercury to a statistically relevant degree. The argument strains to the breaking point because it attempts to relate two different values that are not necessarily related, assuming a relationship which the argument sets out to prove, rendering the argument circular. Also, as other studies have pointed out, whatever metabolizing of the organic mercury would have occurred would have taken more than 28 days to do so, further attenuating any relationship between the organic mercury blood levels and the inorganic mercury brain levels.

at a fairly static level in the subjects that did receive the mercury treatments) increased significantly due to the mercury treatments. It is just as likely that the inorganic mercury levels preexisted, independent of the mercury treatments. But this assumption of Dr. Lucier seems to be central to Petitioners' theory of causation, and the lack of underlying, supporting proof does not bode well for the higher edifice.

Dr. Lucier discussed the negative effects of inorganic mercury, basing his comments upon *in vitro* nerve cell studies that were performed on snail neurons. Tr. 1 at 79. Turning to *in vivo* experiments to discuss the danger of inorganic mercury, Dr. Lucier quoted the Burbacher study just discussed to say, "Conclusions regarding the safety of thimerosal drawn from blood clearance data may not be valid," and "the Pichichero study is not valid for assessing safety of thimerosal." Tr. 1 at 80. On the same point, Dr. Lucier noted the data of a paper by Charleston *et al*., "an animal toxicity study that he was doing with monkeys," which "suggests that the inorganic mercury present in the brain is accumulating after a long-term subclinical methyl mercury exposure may be a toxic form of mercury responsible for changes within the astrocyte[38] and microglial[39] population." Tr. 1 at 80-81. Dr. Lucier then quoted at length from the National Academy of Sciences report on methyl mercury *vis-à-vis* inorganic mercury:

> "Methyl mercury is very slowly but ultimately metabolized *in situ* in the brain to inorganic mercury. Elemental mercury is also oxidized to inorganic mercury in the brain. It is unclear whether methyl mercury toxicity at the cellular level is caused by the parent compound itself due to its inorganic mercury, that is the metabolite, or caused indirectly by free radicals generated by the metabolism of methyl mercury to inorganic mercury."
>
> So they raise three possibilities as plausible possibilities. One, the methyl mercury itself; two, the inorganic mercury itself; and, thirdly, the process by which inorganic mercury is formed, which would release reactive oxygen which is known to be a cellular toxicant, so just a conversion of methyl mercury to inorganic mercury would produce reactive oxygen species which could be toxic. If the ultimate toxic form of methyl mercury is indeed its inorganic mercury metabolite, that suggests the dose of inorganic mercury to the brain from elemental mercury exposure -- and they didn't talk about thimerosal here; this was methyl mercury they were talking about -- might be cumulative. I'm going to go back and start. I missed a line.
>
> "That suggests that the dose of inorganic mercury to the brain from elemental mercury exposure, particularly from dental amalgams, and methyl mercury might be cumulative. That is the case even if oxidation of elemental mercury in the blood before absorption to the brain is considered. Risk assessment models for methyl

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[38] An astrocyte is "a neuroglial cell of ectodermal origin, characterized by fibrous, protoplasmic, or plasmatofibrous processes. Collectively, such cells are called astroglia." DORLAND'S, *supra*, at 169.

[39] Microglia are "the small, non-neural, interstitial cells of mesodermal origin that form part of the supporting structure of the central nervous system. They are of various forms and may have slender branched processes. They are migratory and act as phagocytes to waste products of nerve tissue." DORLAND'S, *supra*, at 1152.

> mercury therefore should consider additional chronic exposures to mercury such as dental amalgams."
>
> The only reason they're saying that is because they think that the dental amalgams obviously release mercury vapor that is converted to inorganic mercury, so they're saying that inorganic mercury should be considered in the risk assessments of mercurial compounds.

Tr. 1 at 82-83.

Dr. Lucier summarized the findings of the Winship article (Pet. Ex. 18, Tab JJJJ, K. A. Winship, *Organic mercury compounds and their toxicity*, 3 ADV. DRUG REACT. AC. POLS. REV. 141-180 (Oxford 1986)) as follows:

> On page 151 they're talking about severe reactions, including death, would be acute studies. They conclude here that the LD-50,[40] which is the measure of acute toxicity, of the different types of mercury compounds and given in acute massive doses are similar because mercury in any chemical form will denature proteins, inactivate enzymes and cause severe disruption of any tissue which it comes into contact with in sufficient concentrations. The signs of acute toxicity usually consist of shock, cardiovascular collapse, acute renal failure and severe gastrointestinal damage.

Tr. 1 at 85, citing Winship at 151.

The next article of medical literature Dr. Lucier discussed[41] was Pet. Ex. 18, Tab KKK, Rudolph Pfab *et al.*, *Clinical Course of Severe Poisoning with Thiomersal* [*sic*], 34 (4) Clinical Toxicology 453-60 (1996). The data from that paper show a logarithmic half life for mercury in the blood, the first half life occurring at 2.2 days, and the second at 40.5 days. Tr. 1 at 87-88. Nevertheless, he also said in that discussion:

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[40] He added that "LD-50" refers to a dose that would be lethal for 50% of those who received it. Tr. 1 at 86.

[41] He summarized the course of the study thusly:

> This is a case where a 44-year-old man tried to kill himself apparently with thimerosal. Probably not a good choice. He was rushed to the hospital presumably by his family, and they started taking mercury measurements in his blood shortly -- I think four to five hours -- after he was admitted to the hospital, so I presume not too long after.
>
> If you look on Figure 1, you can see the time course of mercury disappearance from the blood. Here they're measuring total mercury. The clinical folks who did this actually did a fairly decent study in following the time course. As you can see, they have multiple, multiple points, really more than the Burbacher study, more than any other study I've looked at, even though it was a single individual. There were more measurements made on that individual.

Tr. 1 at 87. The subject of the case study ingested a concoction containing 5g (5 million micrograms) of thimerosal, was in health so poor as to "require[] mechanical ventilation from day 16 to day 18," but evidenced zero deficiencies in neuropsychological testing on day 46 following ingestion, and "neuropathy had resolved completely except for sensory defects in two toes" by day 148. Pet. Ex. 18, Tab KKK at 454.

> They're making a statement of half-life in the blood. After 2.2 days, half the mercury is gone from the blood.... So if you look 4.4 days after, three-quarters. The amount of mercury in the blood would be down to one-quarter of what it was at peak.

Tr. 1 at 88.

On this point, Dr. Lucier is in patent error. At the point of the first half life, half of the original mercury has dissipated. At the end of the second half life, half of that amount, or one quarter of the original amount, has dissipated. That means that three quarters of the original amount has dissipated at the second half life. Since it is a logarithmic scale, not a linear scale, one cannot just double the time of the first half life to arrive at the second half life. Therefore, three quarters of the original amount had dissipated at 40.5 days, not at 4.4 days. As a matter of science, this is extremely basic, and it does not reflect well on Dr. Lucier's general persuasiveness to make such a blatant mistake on so simple a matter.

Surely this was a unintentional error on Dr. Lucier's part, but it does cut into his persuasiveness nevertheless, even to make such an error. Dr. Lucier compared the Pfab data with the Burbacher clearance data as similar, in the sense that the ethyl mercury follows a logarithmic clearance schedule. Tr. 1 at 88-89. Unlike the Burbacher research, where the brain tissue of sacrificed animal subjects could be observed, the Pfab research, relating to a human patient, did not examine brain tissue; however, cerebrospinal fluid was removed for observation of mercury concentration. Tr. 1 at 89; Pet. Ex. 18, Tab KKK at 456. The mercury level rose from 15 micrograms per liter of CSF on day 10 to 25 micrograms per liter on day 15, before falling back to 15 on day 23 and 8 on day 30, such that the last measurement on day 45 indicated a mercury CSF level of 5 micrograms per liter. Pet. Ex. 18, Tab KKK. Dr. Lucier compared this data to the mercury measured in the blood thusly:

> Well, if you compare the blood to the cerebrospinal fluid, if you look at the blood, they measured that over let's take 10 to 45 days. It looks like it's about 800 in the blood going down to probably 50, all right, so there's quite a loss in the blood during that period of time. Whereas in the cerebral spinal fluid it starts out at 15, peaks up to 25 and then starts coming down some, but it only goes down in total threefold during that 40-day period so it's obviously much more persistent in the cerebral spinal fluid, again consistent with what you might expect.
>
> Whether that's inorganic mercury, methyl mercury or ethyl mercury who knows because they didn't measure that, but what it does say is that it's a biphasic curve with a half-life, and this is important I believe, of 2.2 days in the blood and then a second half-life of 40.5 days which reflects its clearance probably from other tissues, including the brain. That's relatively consistent to what Burbacher had seen if you look at the shape of those curves.

Tr. 1 at 89-90.

Dr. Lucier compared the Pfab and Burbacher articles with an earlier article from the 1960s, which also demonstrated a logarithmic curve for the mercury washout rate, which he found "confirmatory of one another." Tr. 1 at 90-91. From this he moved to say (without citation) that the data "show an initial rapid loss in the blood and a second phase which likely, even though I can't say

for sure, but likely relates to removal from the organs, which is much slower," such that "there's a more prolonged exposure in the brain despite the earlier clearance in the blood" applicable to " all forms of mercury." Tr. 1 at 91. That conclusion may indeed be true, but Dr. Lucier did not explain how a conclusion of "early clearance from the blood and more persistent exposure in the brain" followed simply from the data of a recurrent washout rate curve. Tr. 1 at 92.

Dr. Lucier moved on to discuss the effects that mercury has on the body beyond the neurological system. These include "some effects on reproduction and development in terms of the classical parameters," although "[r]enal, immune, [and] cardiovascular probably receive the most attention." Tr. 1 at 94. Dr. Lucier then turned his focus more specifically to cardiovascular effects, and the medical literature that has explored that area. First in this regard was the Grandjean study,[42] which "found that mercury had an effect on blood pressure; that it was actually a more sensitive indicator than the neurological deficits, and I think they occurred at blood concentrations between one and 10 micrograms per liter." Tr. 1 at 95. Dr. Lucier saw this as significant, because to him, "The .01 micrograms per kilogram per day, the so-called EPA safe dose, would be equivalent to 5.8 micrograms per liter." *Id*. The next study on the potential cardiovascular effects of mercury was a Finnish study comparing risk rates of myocardial infarction to mercury levels in the hair, presumably from eating fish. Tr. 1 at 96. Dr. Lucier stated that the study "found a doubling of myocardial infarctions in the exposure level that was above two parts per million [(ppm)] in the hair." *Id*. Dr. Lucier testified that 1.2ppm in the hair is the equivalent of 5.8 micrograms per liter in the blood or 0.1 micrograms in the diet per kilogram of body weight. Tr. 1 at 96. He did not explain how those conversions were made, or from what source he was gleaning those conversions. As noted *supra*, the Court is dubious about equating intake levels with blood levels, given the variables involved, and the same can be said, *a fortiori*, about hair levels.

From there, Dr. Lucier moved on to discuss cardiovascular effects of mercury, referencing the National Academy of Sciences publication, *Toxicological Effects of Methylmercury*.[43] to state

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[42] Pet. Ex. 18, Tab EE, Grandjean et al., *Cognitive deficit in 7-year old children with prenatal exposure to methylmercury*, 19 (6) NEUROTOXICOLOGY AND TERATOLOGY 417-28 (1997). For some reason, this was referred to by the parties as the "Sørenson article."

[43] The Court here reproduces the significant portion of the text to which he referred:

> The cardiovascular effects of Hg exposure in humans ...[include] tachycardia and elevated blood pressure, ... [i]ncreases in blood pressure and heart rate, ... [and m]arked hypertension.... Exposure to organic Hg has also been associated with cardiovascular changes. Three clinical case reports and two epidemiological investigations have reported similar effects.... Abnormalities seen in severely poisoned patients included irregular pulse and electrocardiograms showing ventricular ectopic beats, prolongation of the Q-T interval, depression of the S-T segment and T inversion. Electrocardiograms of four family members who consumed ethylmercury-contaminated pork revealed similar findings, including abnormal heart rhythms with S-T segment depression and T-wave inversion. Deaths of two children in this family were attributed to cardiac arrest, and their autopsies revealed myocarditis. A child who was diagnosed with acrodynia following exposure to vapors from a paint that contained phenylmercuric acetate exhibited a rapid heart beat and hypertension.
>
> Two recent epidemiological investigations have found associations between exposure to low levels MeHg and adverse cardiovascular effects. [The Sørenson article] showed an association between

that " although the database is not as extensive for cardiovascular effects as it is for other endpoints, ... the cardiovascular system appears to be a target for methyl mercury toxicity in both humans and animals," and "adverse health effects can occur at very low mercury exposures." Tr. 1 at 97-98. Dr. Lucier described the NAS summary of the Sørenson paper as stating "alter[ed] blood pressure regulation and heart rate variability" occurred at lower mercury concentrations than other symptoms: "In other words, it was the most sensitive endpoint less than 10 micrograms per liter." Tr. 1 at 98. Dr. Lucier read that article summary in tandem with another, which found "Men who consumed at least 30 grams of fish per day or had hair mercury concentrations of two parts per million or more had a higher risk of suffering a fatal or nonfatal acute myocardial infarction," and that "Mercury exposure was also correlated with increased risk of dying from coronary heart disease or cardiovascular heart disease." Tr. 1 at 99. Together, Dr. Lucier found the data from both studies supported the conclusion that "the cardiovascular system is the most sensitive endpoint." *Id*. Dr. Lucier used this concept of cardiovascular sensitivity to mercury toxicity below the threshold of other health effects to state that the EPA reference dose for mercury intake should be even lower than the 0.1µg Hg/1kg body weight/day standard, due to the potential for adverse cardiovascular effects–perhaps even 50% lower, or half the current reference dose. Tr. 1 at 100-101. He alluded to analytical approaches that have viewed studies' data selectively to arrive at a higher risk estimation than did the EPA in arriving at its reference dose. Tr. 1 at 101. Dr. Lucier opined that "there are arguments that the EPA RFD could be reduced even further," and reported that an EPA alumna, Debra Rice, co-wrote an article[44] indicating "the EPA reference dose should be 0.0596,

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> prenatal exposure to MeHg and cardiovascular function at age 7. The study of 1,000 children from the Faroe Islands found that diastolic and systolic blood pressures increased by 13.9 and 14.6mm Hg, respectively, as cord-blood Hg concentrations rose from 1 to 10µg/L. In boys, heart-rate variability, a marker of cardiac autonomous control, decreased by 47% as cord-blood Hg concentrations increased from 1 to 10µg/L.
>
> ...Effects of MeHg on the heart and circulatory system have been observed in several animal models. [One report] described cerebrovascular lesions in four nonhuman primates following long-term exposure to near-toxic to toxic doses of MeHg hydroxide (90 to 120µg/kg per day). Lesions were similar to those observed in humans with hypertension; intimal thickening, smooth-muscle cell proliferation, and adventitial fibrosis were reported.

Pet. Ex. 18, Tab HHH, Committee on the Toxicological Effects of Methylmercury, *Toxicological Effects of Methylmercury* 169-172 (2000).

[44] After summarizing the process by which the EPA reached their reference dose, the authors discuss "other issues not addressed in the RFD," including the following:

> Another area that EPA identified for further analysis was the association between methylmercury exposure and adverse cardiovascular effects. In a study of 1,000 seven-year-old Faroese children, diastolic and systolic blood pressures increased by 13.9 and 14.6mm Hg, respectively, as the cord-blood mercury increased from 1 to 10µg/L. A 47% decrease in heart rate variability (an indication of decreased cardiac autonomic control) was also observed. In a seven-year observation period of 1,833 Finnish men, individuals with hair mercury in the highest tertile (2ppm or higher) had a 2.0 times greater risk of acute myocardial infarction compared with the rest of the study population. The body burden at which cardiovascular effects are observed in adults should be identified, preferably with quantitative procedures such as BMD analysis.

Pet. Ex. 18, Tab NNN, Deborah C. Rice *et al.*, *Methods and Rationale for Derivation of a Reference Dose for*

which she for EPA purposes conveniently rounded off to .1, when in fact it would be more appropriately rounded off to .06 micrograms per kilogram per day." Tr. 1 at 101-02. In Dr. Lucier's mind, this means that "with respect to the cardiovascular system the [current consensus] risk assessment[s] may not take into account that cardiovascular effects occur at a lower dose of mercury.... [Conducting] a risk assessment on those studies you'd come out with a lower number or a greater risk." Tr. 1 at 116. Additionally, the evidence discussed *supra* convinced him that, "Ethyl mercury is more toxic ... to the cardiovascular system," and there is "a greater rate of inorganic mercury formation from ethyl mercury than methyl mercury." Tr 1 at 117.

Next, Dr. Lucier described the Pichichero study,[45] wherein blood samples from vaccinated infants were taken at two and six months of age, the conclusion of which, said Dr. Lucier, was that "Overall, the results of this study show that amounts of mercury in the blood of infants receiving vaccines formulated with thimerosal are well below concentrations potentially associated with toxic effects." Tr. 1 at 103. Dr. Lucier questioned the validity of that conclusion, based on the unit of measure employed for quantifying mercury levels, inasmuch as the researchers measured mercury dosage by nanomoles per liter of blood instead of micrograms. Tr. 1 at 104. He explained that the dosage of 29 nanomoles per liter is the equivalent of 5.8 micrograms per liter. *Id*. Under those circumstances, Dr. Lucier found it unsupportable that "blood results from [the infants tested] were lower than the 5.8 micrograms." *Id*. The reason for his position is that the Pichichero researchers "took blood samples between three and 28 days," which to Dr. Lucier means that, "his measurements at best are starting when already two half-lives have elapsed, and remember half-life is the time it takes for half of it to leave the blood, so it would be very likely that that number is estimated fourfold. It's not big-time math. Two times two is four." Tr. 1 at 105. Assuming, as he did, that two half lives had passed at five days, he then multiplies the day five level by a factor of four to reach a value of 16 micrograms per liter, which he estimates as the "likely real value that those children had experienced" at day one, such that "it's likely that some of those children had blood levels that had exceeded the safe level."[46] Tr. 1 at 106. All in all, Dr. Lucier opined that "the Pichichero data drew a conclusion that ... [was] indefensible because of the timeframe in which the bloodwork was drawn." Tr. 1 at 109.

Methylmercury by the U.S. EPA, 23 (1) Risk Analysis 107, 113-114 (2003).

[45] Pet. Ex. 18, Tab JJJ, Michael E. Pichichero *et al.*, *Mercury concentrations and metabolism in infants receiving vaccines containing thimerosal: a descriptive study*, 360 THE LANCET 1737-41 (2002).

[46] Beyond that, Dr. Lucier estimated that the Pichichero data was low by an additional factor of 1.7, based on figures three and six from the Burbacher paper, which compared the mercury concentration of the blood and the brain, as between methyl and ethyl mercury respectively. Tr. 1 at 110-111. He based this on the presumption that the Pichichero research used methyl mercury properties to estimate ethyl mercury values. *Id*. However, Dr. Lucier did not cite to the appurtenant portion of the Pichichero article. Also, to the extent Dr. Lucier explained his mathematical extrapolations at all, they were unpersuasive because they moved far beyond the data themselves, and assumed relationships unsupported by the graphical data or the Burbacher paper itself. Moreover, he combined this extrapolation with his estimated day 1 level of 16 μg/L, which the Court found unsustainable and unpersuasive. Thus, the 1.7 factor was ultimately irrelevant, as it was predicated on another faulty calculation.

Dr. Lucier (and Petitioners, accordingly) appear desperately to misunderstand the concept of logarithmic scale half lives. Dr. Lucier himself had stated earlier that the first and second half lives for mercury in the blood were 2.2 and 40.5 days. Tr. 1 at 87-88. As noted *supra*, one cannot estimate the second half life by doubling the time of the first, because half life is not linear, but rather logarithmic in scale. In this case, two times two does not equal four. The Court feels chagrin on Dr. Lucier's behalf for making such a simple error not once, but twice, and not from an extemporaneous answer to a sidelong query, but in the course of testimony explaining his previously-composed expert opinion. If that is the heart of his contention with the Pichichero study, his argument is ascian[47] indeed.

Dr. Lucier also briefly mentioned, without directing the Court to an exhibit, that a study by NHANES evaluated mercury levels in the population and found an average level in children of 0.3 to 0.4 micrograms of mercury per liter of blood, but as high as 1.1 micrograms in adult women, and indicated that "eight percent of the women in this country have mercury levels that exceed the safe level." Tr. 1 at 107. On the basis of Dr. Lucier's earlier testimony, the Court notes that 0.3-0.4 micrograms of mercury per liter of blood meets the safety criteria of all the other governmental agencies (excluding than the EPA), and the information about adult women is relevant only for adult and perhaps *in utero* human populations, neither of which are at issue in this proceeding. But Dr. Lucier stands on these results as proof that "there seems to be a subgroup of people who are at greater risk of harm from exposure to mercury ... a lot of people that are already right at the edge." Tr. 1 at 108. Elaborating, Dr. Lucier stated:

> If you've got eight percent of the women at child bearing age already have levels that are considered to put their developing offspring at risk, any additional mercury exposure of those offspring could very well kick them over the edge. You know, it's a very simple concept. You're not starting from ground zero when you inject thimerosal. Like I said earlier, everyone in this room has a certain body burden of mercury, so we're not starting from zero. We're starting from some number.

Tr. 1 at 108. This testimony is relevant to the extent that the mercury added to the preexistent body burden is a *substantial* factor in any injury caused by mercury toxicity. It might have been helpful if Dr. Lucier had been able to opine on the relative additive toxicity of the mercury added by vaccines containing thimerosal in comparison to the other mercury inputs from the environment; however, the Court understands that this would be quite difficult to quantify. In any event, it is Petitioners' burden of proof to demonstrate that the ethyl mercury contained in thimerosal-bearing vaccines could potentially be, and was actually, a substantial factor in causing the injury complained of, up to and including the death of Thomas Kolakowski (*inter alios*).

Another fashion in which Dr. Lucier believes ethyl mercury to be a greater threat than is generally conceived, is the disparity between blood levels and brain levels: blood levels are what researchers use to quantify mercury input, because of the ease of testing; however, it is the brain that is seen as at the most risk from mercury toxicity. Tr. 1 at 110. In contrast, brain samples of living humans are hard to come by, and inconvenient to remove. Dr. Lucier augments this problem with

[47] "Σκιᾶς ὄναρ ἄνθρωπος." ("A dream of a shadow is Man.") Pindar, *Pythian* 8.92-97.

an argument that ethyl mercury is comparatively more persistent in the brain as compared to the blood, where it washes out much more rapidly than does methyl mercury:

> [Blood and brain mercury] levels are going down in every case, but they're going down at differing rates. Again, you're looking at total mercury. The inorganic mercury is yet a different story and actually is even more pronounced than what I just talked about. But looking at total mercury, the brain to blood ratio is much higher for ethyl mercury than it is for methyl mercury, and we're not concerned about blood toxicity. We're concerned about brain toxicity here, so the blood levels for ethyl mercury, put in the context of the methyl mercury risk assessment, would underestimate risk.

Tr. 1 at 113-14.

The Court was not persuaded by Dr. Lucier's argument on this point. It would seem that Dr. Lucier is comparing the wrong two curves. True, as shown by the Burbacher data (figures three and six), there is a greater disparity between the brain and blood washout rates with ethyl mercury than there is with methyl mercury. However, this is only because the blood clearance rate with ethyl mercury is so much faster than with methyl mercury. In fact, the brain clearance rate of ethyl mercury appears as fast or faster than the brain clearance rate of methyl mercury.[48] In one sense, Dr. Lucier is comparing inapposite values in a fractional proportion: instead of comparing the blood and brain clearance rates of ethyl mercury as a ratio to the corresponding ratio for methyl mercury, it would seem more sensible and relevant to compare blood clearance rates as between ethyl and methyl mercury, and the brain clearance rates as between ethyl and methyl mercury, so as to isolate for examination the variables that are most important.

As a second, more practical point, the relative rate of blood washout would seem necessarily to affect the brain accumulation and washout rates for organic mercury. Whereas blood washout graphically resembles a simple negative logarithmic function, beginning at the level of input and decaying through half lives to approach zero, the brain must receive substances such as mercury indirectly from the blood.[49] Therefore, mercury level in the brain follows a sort of unsymmetrical "bell" curve, where the brain increasingly receives mercury from the blood up to a peak level before decreasing to a level approaching zero through washout. That means the brain's bell-shaped curve is dependent on the slope of the blood washout curve (assuming a sporadic exposure to the organic mercury) to determine its slope and peak level, because brain mercury level is dependent on the input concentration of mercury from the blood. Since ethyl mercury washes out so much more quickly than does methyl mercury, this actually militates for a shallower curve of mercury input into the brain. Also, the relatively faster brain washout rate of ethyl mercury (as compared to methyl mercury brain washout rate) will flatten that curve out as well.

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[48] It is difficult to compare these rates absolutely in the Burbacher figures because figure 3 is a logarithmic scale graph with *y*-axis values from 10 to 1,000, whereas figure 6 is a logarithmic scale graph with *y*-axis values from 1 to 100.

[49] Dr. Lucier himself explained this. Tr. 1 at 151.

Finally, to conclude his direct examination, Dr. Lucier discussed thimerosal in vaccines directly. He stated that, to the best of his knowledge, the Hepatitis B vaccines given to infants contain an average of 12.5 micrograms of ethyl mercury. Tr. 1 at 129. Given that Thomas Kolakowski was "about 4 kilo[grams]" (8.8 lbs.) when he received his Hepatitis B vaccine, Dr. Lucier opined, "That would be a dose of three micrograms per kilogram. The EPA RFD is 0.1 micrograms per kilogram, so that would exceed the EPA RFD by thirtyfold ... just based on that one day of administration." *Id*. Moreover, Dr. Lucier opined, based on figures 2-7 (pages 1018-19) of the Burbacher article, that "for the inorganic mercury[,] there is no degradation rate. It has an infinite half-life. So two days after, there's roughly 11 nanograms per gram; at 28 days, there's one nanogram per gram.[50] So I think it's reasonable to assume that a second dose of mercury would double that inorganic mercury concentration in the brain because that has stayed constant." Tr. 1 at 130. However, Dr. Lucier's assumption presumes that the recorded inorganic mercury in the Burbacher subjects' brains following the last dose of organic (ethyl) mercury was there in the brain entirely because of the organic mercury doses administered in the study. Without a control, sacrificed and measured before any mercury doses were administered, this is impossible to prove, and too speculative for the Court to rely thereupon.

On cross-examination, Dr. Lucier stated that he considers himself "an expert in developmental pharmacology and perinatal pharmacokinetics," and even if not an expert in genetics, then "one who is very knowledgeable in genetics," since he oversaw "a lot of the genetic toxicology work that was done at the National Institutes of Health under the auspices of the National Toxicology program." Tr. 1 at 133-34. Dr. Lucier has never performed "bench research" on ethyl mercury or thimerosal, but has on methyl mercury. Tr. 1 at 138. He has not authored original research literature published in a peer-reviewed journal on either ethyl mercury or thimerosal. Tr. 1 at 138-39. Dr. Lucier agreed that he is neither specially trained in, nor otherwise an expert in the field of neurology, but that he has "had to learn quite a bit of neurotoxicology as part of [his] role as NTP associate director because [he has] had to oversee some of the research activities and make decisions about what should be tested and how it should be tested." Tr. 1 at 177-78. Nonetheless, he admitted that he is "not a neurotoxicology specialist." Tr. 1 at 179.

Dr. Lucier clarified his position from his expert opinions in other lawsuits as to whether he thought autism could be caused by vaccines:

> [M]y conclusion has been that thimerosal and infant vaccine cause neuro-developmental disorders, and some of the symptoms associated with autism may be among those disorders. I have never said that thimerosal causes autism.
>
> ...[T]himerosal, at the dose given, will cause some neuro-developmental disorders in some children, for the reasons that I've stated this morning, and some of those effects may well be considered part of an autistic syndrome, but I have never said that it has caused autism.

[50] The Court could not tell what data Dr. Lucier was referring to in this statement. Those figures do not appear to correspond with data in Burbacher's figure 7, or with any of the others therein.

> ...[T]he level of thimerosal ethyl mercury exposure can cause effects because it's exceeding a safe level. Whether or not it causes death is another issue. I don't know whether it can cause death or not for any individual, but I do know that the safe level was exceeded, and whenever you exceed the safe level, you're at risk for any number of different health effects.

Tr. 1 at 142-44.

Turning to the proceedings at hand, Dr. Lucier opined that, even though he does not "have an opinion on this specific case, ... the safe level of mercury was exceeded," and thus he believes that "death is one of the adverse effects that could be caused by exceeding the safe exposure level." Tr. 1 at 144. However, he conceded that his opinion is limited to the "can it" aspect of this case (and the others to which this Causation Ruling pertains), and he did not venture to offer an opinion on the "did it" aspect;[51] in fact, he did not even review the medical records specific to Thomas Kolakowski. Tr. 1 at 146. Later, Respondent queried Dr. Lucier as to the relevance of his expert report–which detailed the neurodevelopmental effects of mercury–to this case and those grouped with it, where Petitioners claim that ethyl mercury contained in vaccines with thimerosal caused cardiac problems and death, to which he responded:

> I'm just here to provide information on the toxicity of mercury, and that's my contention, in terms of the evidence at hand, that it has caused developmental neurotoxicity in some children and went through the reasons why because everyone is already exposed to a certain level. Any additional exposure is likely to have some effect in some children, and the most common effect of mercurials is developmental disorders. So I think there is a logical and defensible sequence of information there that leads to that statement, that it will cause neurodevelopmental disorders in some children. How you relate that to this particular case is really up to you.

Tr. 1 at 180. Furthermore, Dr. Lucier conceded that he does not proffer a theoretical framework for "how ethyl mercury and thimerosal-containing vaccines results in sudden death;" his point is merely "that what has been considered safe exposure levels for ethyl mercury or organic mercurials have been exceeded by the use of thimerosal-containing vaccines in infants." Tr. 1 at 181. He added, "I can only say that safe levels have been exceeded and exceeded by not just a trivial amount," and that he was unable to offer an expert opinion "whether it is thimerosal's effect on the heart tissue or the brain that would cause sudden cardiac arrest." Tr. 1 at 183.

Respondent challenged Dr. Lucier with the concept expressed by the maxim that "the dose makes the poison." Tr. 1 at 148. When Respondent begged the question whether any substance could be harmful or fatal with the right dosage intake, Dr. Lucier seemed to disagree with that proposition on the basis of "latent effects." *Id*. His answer did not address whether high dosage

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[51] Petitioners did not offer *any* medical opinion from an expert in toxicology stating specifically that the amount of thimerosal received by Thomas Kolakowski in particular was an unsafe or lethal dose that *did cause* mercury toxicity sufficient to result in injury or death. However, this is part of Petitioners' burden of proving "but for" causation, *via* a "logical chain of cause and effect." *Pafford v. Sec'y of HHS*, 451 F. 3d 1352, 1355 (Fed. Cir. 2006), *rehearing and rehearing en banc denied*, (Oct. 24, 2006), *cert. den.*, 168 L. Ed. 2d 242, 75 U.S.L.W. 3644 (2007).

could cause harm, but focused instead on whether such harm could be identified, isolated, or quantified, as a practical matter. Tr. 1 at 149. He explained that nonspecific or general symptoms are hard to pinpoint to a particular cause, it is true, and mercury toxicity takes some time interval to work its harm, undoubtedly; however, these are research challenges, and have little to do with whether, as a theoretical matter, with high enough doses, any substance could be harmful or lethal. Dr. Lucier's argument appears to be that just because no clinically-evident symptoms are observed or measured, does not mean that harm is not being done, and that harm is being done by small amounts of a toxin up to and including the moment when symptoms are noticeable:

> [Y]ou can detect these early changes at much lower doses than you can detect statistically significant increases in adverse health outcome, and that's the whole basis of mechanism-based toxicology, why that's becoming used so much now in the EPA regulations and FDA regulations and biomedical research, so you can detect early changes prior to adverse outcomes. When you can do that, there is a reasonable chance that some of those individuals will develop an adverse effect.

Tr. 1 at 150. Dr. Lucier found it "scientifically valid to compare the doses of ethyl mercury and thimerosal-containing vaccines to the exposures of methyl mercury that were consumed by the population of Minamata." Tr. 1 at 159. That is, he thought the difference in adverse effects between very small doses and very large doses was one of degree, not kind, that a little dose would have a proportionally small effect, and that a big dose would have a larger effect. Tr. 1 at 159-62.

When Respondent pressed further on the relationship between dosage and the nature of corresponding symptoms (as well as their severity), Dr. Lucier elaborated:

> [Y]ou might have one set of responses that would occur at high doses, yet a different set of responses that would occur at low doses.... The amount that you receive is important, but I'm saying that you don't have necessarily a straight, linear dose response curve for adverse outcomes, especially for something like ethyl or methyl mercury where you have different organs that it affects, you have different latencies, and you're going to have different shapes of the dose response curve, and you're going to have different sensitivities.... Maybe the cardiovascular system is, in fact, the most sensitive organ.... So the dose response in the cardiovascular system is resulting from a different molecular target. It's going to have a different dose response, probably, likely, than you would for neurological deficits, and even for neurological deficits, you're going to have some things that happen at high doses, some things happen at low doses. So what you're getting at, I think, is an oversimplification of biological reality.... I mean, you may have subtle loss of IQ that we wouldn't detect in the population. Obviously, if you look at children now, hyperactive disorders are on the rise.... I'm not attributing that to mercury.... There are a lot of behavioral problems that are here. Whether or not they are related to thimerosal or mercury is uncertain.

Tr. 1 at 153-55. Respondent then drew Dr. Lucier back to the theory of the case at hand. He conceded that he was not aware of the existence of "any articles that these children actually suffer from neurodevelopmental delays or cardiac problems as a result of mercury exposure," and that any

articles of that sort would "have been, like the Grandjean studies, the articles from New Zealand, which have related mercury exposure to adverse neural behavioral outcomes." Tr. 1 at 156.

Respondent challenged Dr. Lucier's opinion on dosage using Pet. Ex. 18, Tab F, F. Bakir *et al.*, *Methylmercury Poisoning in Iraq: An interuniversity report*, 181 SCIENCE 230-241, 238 (July 1973):

> Q While you have that in front of you, do you recall the threshold dose of methyl mercury that was measured in Iraq, in this article, before effects of toxicity were observed?
>
> A You mean statistical effects.
>
> Q Clinical effects.
>
> A No, I don't.
>
> Q I'll refer you to page 238, second column: "Nevertheless, the threshold value of 25-to-45 milligrams of mercury [that] is computed for paresthesia agrees remarkably well with the threshold figure of 30 milligrams of mercury computed by Swedish experts," and it goes on. Now, 25 milligrams; that's the equivalent of 25,000 micrograms. Is that correct?

Tr. 1 at 163-64. Dr. Lucier did not accept Respondent's characterization:

> I would not agree that that's necessarily a threshold ... because effects have been observed at lower doses since, lower levels of mercury exposure. So, obviously, it was not a threshold; they just did gross measurements of neurological function and didn't look at the kind of neural behavioral outcomes that were present in the Faroe Islands. So, obviously, it was not a threshold. Later science showed that statement to be wrong.

Tr. 1 at 165.

Respondent next challenged Dr. Lucier on his offered opinion regarding cardiac effects of mercury, in reference to Pet. Ex. 18, Tab U, Shawkat S. Dahhan and Hussain Orfaly, *Electrocardiographic Changes in Mercury Poisoning*, 14 THE AMERICAN JOURNAL OF CARDIOLOGY 178-183 (August 1964):

> Q In the Dahhan article where you just state it discusses the cardiac effects of ethyl mercury, is that correct? Is that why you cited that article?
>
> A I cited that as their adverse electrocardiograms and things like that as an example of effect, and I think one of the authors, and I've have to go back and look, had said that those were effects that were seen with ethyl mercury but apparently not methyl mercury, was one of the conclusions of the authors, not my conclusion because I haven't looked at all of the papers in that respect.
>
> Q Okay. But the Iraqi ethyl mercury poisoning case described in the Dahhan paper; do you know what kind of mercury that was? ... the initials are "EMPTS," ethyl mercury paratoulene sulfonamide.... Does EMPTS have the same

pharmacokinetics as the ethyl mercury and methyl mercury in thimerosal-containing vaccines?

A That, I don't know. I don't know if it does or not.

Q Has the cardiotoxicity that was described as a feature of the ethyl mercury at the EMPTS, has that ever been described with thimerosal-containing vaccines?

A I'm not aware of that.

Q Do you know the dose of EMPTS that caused the effects observed in the Dahhan paper?

A I'm sure that all of those doses were high doses because the people consume large amounts, and then no one found out about it until many of the people got sick. So I'm sure it was a fairly significant dose.

Q Those doses were dramatically different, you would agree.

A I have no doubt about that. It's actually difficult, from this article and my recollection of it, to sort out the exact dose.

Q And for the patients who had this cardiac toxicity, did any of them die suddenly, to your knowledge, have a sudden cardiac arrest?

A I don't know.

Q Did the patients show other signs of clinical neurotoxicity, mercury toxicity?

A I didn't review that paper; I just looked at it for the basis of the electrocardiographic changes that were seen as a consequence of that exposure.

Tr. 1 at 167-69.

Respondent asked Dr. Lucier to explain the difference in attributes between ethyl and methyl mercury, to which he replied:

There were two differences that I alluded to. One was the dealkylination was faster for ethyl mercury than methyl mercury, and this led to the longer retention in the brain of the inorganic mercury that was formed from the ethyl. It's presumed that it was formed there since it doesn't pass the blood brain very, very well.

...Again, as I said this morning, when we looked at 2,800 high-production-volume chemicals for EPA, for which there was little or no toxicological information in the publicly available literature, that industry didn't want to test all of those 2,800. The agreement was they would test the ones that they needed to test but not the ones they didn't. So they would send requests for categories of chemicals, and if I got a request for ethyl and methyl mercury, I would say that's a category; you only have to test one, not the other, and that's basically the procedure that EPA went through with that because the similarities are so much. ... They are both neurotoxicants, they both have a common metabolite, and they have a very similar structure. It was very usual, in the high-production-volume chemicals, to have different aliphatic chain lengths and

> call them part of a structural class, and that's what you have here, a difference in aliphatic chain length, and what you would do with that is generally test the one that you would predict would be most toxic and then apply that to the others, and that was the procedure they went through.
>
> ...They are still basically structurally similar, and if you don't have data on one of them, you can assume data for the other. You can assume it from the other, and that's a common practice.

Tr. 1 at 173-76.

Dr. Lucier pointed to the Sørenson article as support for the potential for a cardiac event as an adverse effect of thimerosal-containing vaccines, because such study noted the symptoms of "heart rate variability and effects on blood pressure" observed at levels that Dr. Lucier says "would have been exceeded in the case of thimerosal-containing vaccines," because those symptoms are "risk factors, certainly, for adverse cardiac outcomes." Tr. 1 at 181. He qualified this opinion, however, saying, "Whether or not they would be sufficient to cause death, that's for a cardiologist to examine." Tr. 1 at 182.

Dr. Lucier apparently fell into speculation when he opined on the substantiality of thimerosal as the proverbial "straw that broke the camel's back":

> Q You said that they are already at risk that they have exceeded the safe levels before they are even born. Why do you believe it's the thimerosal-containing vaccines that tip these children over the edge or cause cardiac arrest as opposed to the breast milk, which also contains mercury?
>
> A Any additional exposure, whether it's from thimerosal, whether it's from breast milk, could tip them over the edge. I'm just saying that it's clear that thimerosal has tipped some of the kids over the edge.
>
> ...What I'm saying is that we do know, with great certainty, what the exact dose was that the infants received, and we know what the current levels are in the population, and, therefore, we know that the addition of thimerosal-containing vaccines has pushed some of those individuals over the top in the case of mercury toxicity. Whether other things have pushed them over the top or not is another issue, but not relevant today.

Tr. 1 at 184. The Court is not aware where in his testimony Dr. Lucier provided the basis for this opinion. When questioned on this point, Dr. Lucier vaguely alluded to "the National Academy of Sciences," but stated that ultimately, the basis for this claim was "all just logic ... irrefutable logic":

> If you already have 60,000 people, babies, born each year from mothers who have too much mercury in their bodies, an additional burden to those kids is going to cause an additional proof of children to be put at risk for mercury-related diseases. The National Academy of Sciences stated that there are 60,000 babies born at risk. The Centers for Disease Control have said that eight percent of the women in this country currently have mercury levels that are above the safe level.

> So I don't think it takes any stretch -- this is pure, irrefutable logic -- that any additional exposure will put you at an additional risk. It's sort of like if you drink five beers at home, you have five beers at home, you have a blood alcohol content of .07, and then you go to a bar and have another beer, it puts you at .09. You say, "Well, I only had one beer at the bar, so it wasn't the beer that caused the effect." You already have a starting point that is many of the babies are already at the edge. Any additional mercury exposure will put them at risk.

Tr. 1 at 185-86. Dr. Lucier soon after conceded that those 60,000 children discussed by the National Academy of Sciences were stated to be "at risk for neural behavioral outcomes," and not "cardiac arrest or sudden infant death syndrome or sudden death." Tr. 1 at 187.

Respondent challenged the contention in Dr. Lucier's expert report that "the developing brain is a approximately five to 10 times more sensitive to developmental neurotoxic effects of organic mercury than is the adult brain." Tr. 1 at 187. Dr. Lucier clarified that by that, by "developing," he meant the prenatal brain, because organ systems are more vulnerable when developing than they are before or after that development, and that he drew that figure from the studies of Japanese Minamata Bay where children "*in utero* seemed to have the most damage." Tr. 1 at 187-89. He added that "it's almost as much with infant damage but considerably less with adults." Tr. 1 at 188. He quoted from the abstract of Pet. Ex. 18, Tab QQQ, Patricia M Rodier, *Developing Brain as a Target of Toxicity*, 103 ENVIRONMENTAL HEALTH PERSPECTIVES 73-76 (1995 Suppl. 6), reading:

> The human brain forms over an unusually long period compared to other organs. While most of the basic structure is laid down before birth, neuron proliferation and migration continue in the postnatal period. The blood-brain barrier is not fully developed until the middle of the first year of life. The number of synaptic connections between neurons reaches a peak around age two and is then trimmed back [by about half]. [Similarly, there] is great postnatal activity in the development of receptors and transmitter systems, as well as the production of myelin. Many of the toxic agents known to damage a developing brain interfere with one or more of those developmental processes. Those with [antimitotic] action, such as x-ray ... and methyl mercury, have distinctly different defects on structure[] depending upon which neurons are forming at the time of exposure. Vulnerability to agents that interfere with cell production decrease[es] rapidly over the early postnatal period.

Tr. 1 at 191-92. All of the studies proffered in support of this contention mentioned methyl mercury by name, not ethyl mercury. Tr. 1 at 192. Dr. Lucier could not say whether most of the populace has as frequent environmental exposure to ethyl mercury as it does with methyl mercury. Tr. 1 at 193-95.

Respondent's questioning of Dr. Lucier on the Charleston study led to the following interchange:

> Q Okay. Is there any evidence that you've pointed to today that inorganic mercury in the brain resulting from thimerosal-containing vaccines causes neurological damage? Any peer-reviewed articles that conclude that the inorganic mercury from thimerosal-containing vaccines causes neurological damage?

> A No. I would refer you, though, to the Burbacher article, who said the long retention in the brain meant that the safety of thimerosal could not be judged by blood levels of ethyl mercury because the half-life of inorganic mercury within the brain was so much longer.

Tr. 1 at 198. Respondent pursued Dr. Lucier to differentiate methyl mercury, which had been studied in detail for adverse effects, from ethyl mercury, which has not been so studied, but Dr. Lucier returned to his point that the two are more or less identical in that, when metabolized, they both end up as inorganic mercury as their metabolite, which is unquestionably harmful (in one dose or another) to cells. Tr. 1 at 198-200.

Respondent's questioning next turned to acute lethal outcomes of mercury toxicity. Dr. Lucier could not point to a reference discussing sudden death following inorganic mercury exposure in the brain. Tr. 1 at 201. When queried for "any reports of increased rates of either sudden infant cardiac arrest" in the Minamata and Iraq data, Dr. Lucier responded that, "They only referred to numbers of deaths," and that the authors did not report "the cause of death." *Id*. He conceded that all who died in those mass exposures received "fairly high" doses, and admitted that he was "not aware of any" peer-reviewed medical literature establishing a higher proportion of "cardiac arrest or sudden infant death [among] children who received thimerosal-containing vaccines." *Id*.

Dr. Lucier conceded, regarding the Faroe Islands data (used by the EPA to calculate the "safe dose"[52]), that the neurological findings measured by the researchers *via* neuropsychologic testing were not manifestly observable neurological deficits, but were only discovered by the nuanced testing. Tr. 1 at 203-04. From there, Respondent asked Dr. Lucier to compare the amount of mercury exposure typical among the Faroe Island test subjects with the amount of ethyl mercury in a Hepatitis B vaccine. He answered:

> [T]here was a gradation of exposure in the Faroes, and I think the cord blood level effects were seen at roughly 20 micrograms per liter.... In the case of thimerosal, I think I went through the estimates from the Pichichero data that there was probably 20 micrograms -- 16 micrograms per liter would have been a reasonable extrapolation of the fact that he didn't take any samples until two half-lives had exceeded, so it would have missed the peak exposure by roughly two half-lives for the initial 2.2 days that represents the blood half-life. So the real exposure would be 16 micrograms per liter in that case.
>
> Then there was the additional relationship to the brain. If you're concerned with the brain being the target organ, then the ratio between the blood and the brain has to be corrected for in the thimerosal case because that number is higher. It's preferentially

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[52] The EPA reference dose is a ten-fold reduction from a dose which, when pregnant mothers consumed it, was producing adverse effects in their children, measured once they were born. Tr. 1 at 215-217. This is not the same thing as a threshold dose:

> A threshold dose says it's a dose below which no effects will occur. The reference dose is a virtually safe dose, which means that there will be very few individuals who will be affected below that dose.

Tr. 1 at 218.

> retained in the brain, and that adds a factor of, again, almost two, so you get up close to a functional level of about 30 micrograms per liter, which, again, exceeds the EPA safe level.

Tr. 1 at 205-06. As noted *supra*, the Court does not accept Dr. Lucier's critique of the Pichichero article, nor his extrapolation therefrom. Additionally, the Court can find no support in the record for a two-fold difference in rate of brain retention for ethyl versus methyl mercury, and Dr. Lucier did not provide a persuasive supporting reference for that claim.

When asked to list the most likely neurological deficits a person would be at risk for if their mercury intake exceeds the EPA safe dose every day, Dr. Lucier stated:

> You might get a little hyperactive, you might have a slight change in blood pressure, based on the other Grandjean study, in terms of blood pressure. You might feel a little heart rate variability because, remember, that happened at blood levels between one and 10 micrograms per liter, and we don't know if children are more sensitive than adults for that effect.

Tr. 1 at 224-25.

Respondent called into question whether ethyl mercury could be considered equally toxic as methyl mercury, inasmuch as Dr. Lucier had previously stated his opinion that the two had different toxic attributes that roughly balanced out to an equally potent level of toxicity.[53] Dr. Lucier conceded that no peer-reviewed study had pronounced ethyl mercury to be more toxic than methyl mercury, but alluded to a study from the FDA concluding "that ethyl and methyl mercury need to be considered equitoxic." Tr. 1 at 225-26. Respondent pressed Dr. Lucier to comment upon the Magos article (Pet. Ex. 18, Tab YY[54] at 260), which states in its abstract that "there was little difference in the neurotoxicities of methyl and ethyl mercury," but finding that methyl mercury is approximately 20% more toxic than ethyl mercury. Tr. 1 at 227-28.

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[53] Dr. Lucier himself stated it thusly:

> One argument is that ethyl mercury might be less toxic than methyl mercury because it is, in fact, dealkylated and cleared from the blood more readily, although [the Magos] article doesn't address where that mercury is going. It may be going into other tissues, it may be going to the heart, it may be going to the liver, or it may be going to the spleen. It may be going a lot of places that we would be concerned about. All he knows is it is disappearing from the blood. So that's an issue. But, on the other hand, his study shows that there is greater retention in the brain of inorganic mercury that's formed from the ethyl mercury in comparison to what happens with methyl mercury. So that would cause an increased concern, and I think those two concerns are going in opposite directions. I believe they balance themselves out, and I think, in the absence of any data to the contrary, they have to be considered equitoxic for the use and risk assessment, and since they both are neurotoxicants, they both have the same common metabolite, they have similar structures dealing only in the alkyl chain length. The methyl mercury data are appropriate for use in risk assessment.

Tr. 1 at 231-232.

[54] Pet. Ex. 18, Tab YY, L. Magos *et al.*, *The comparative toxicology of ethyl- and methylmercury*, 57 ARCHIVES OF TOXICOLOGY 260-267 (1985).

The last aspect of Dr. Lucier's testimony relevant to the Court's analysis is his discussion on the use of *in vitro* mechanistic studies in studying toxicity, a question raised by Respondent on cross-examination. Dr. Lucier defended reliance upon such studies, stating the following regarding their application:

> One way is as a prescreen. Pharmaceutical companies would do this with their new drugs. They have a list of things. If they gave a new drug to any of these systems, and they found effects at this level, do you know what would happen with that drug? It would be tossed out the window because of fear of an adverse effect. So they use it as a screening tool. The other way it can be used is to help substantiate what might be an inconclusive result in animal experiments. What this provides is biologic plausibility, which is one of the criteria for causality.
>
> One thing you would not use it for is if you only had *in vitro* data, you would not conclude anything about health risks with that data alone. What you're using it for is in conjunction with your entire amount of data to make an overall toxicological evaluation, and that's what I've done here, and that's what's customary.
>
> These *in vitro* studies have not looked specifically at the cardiac system. The only one that's probably relevant to that, in terms of cardiac arrest, would be the number four, the Sebashe study, in which it compared thimerosal and methyl mercury to disrupt normal cellular calcium levels, and cellular calcium levels are involved in cardiac function.

Tr. 1 at 244-45, 247-48.

2. Thomas M. Connor, M.D.

Dr. Thomas M. Connor is a physician by profession, pediatric cardiology in specialty. Transcript of Proceedings convened on 10 June 2008 (Tr. 2) at 356. He summarized his professional background thusly:

> I have a Bachelor of Science, biology major, chemistry minor; a medical degree in 1966, followed by an internship at Grasslands Hospital, which is now New York Medical College, where I did a rotating internship which included obstetrics, surgery, internal medicine, pediatrics, emergency room; two and a half year as a pediatric resident.
>
> In 1970, I was accepted for a fellowship by Norm Towner at Yale New Haven Medical Center where I spent two years in pediatric cardiology. After I finished pediatric cardiology, I was then brought to the Naval Hospital at Bethesda, Maryland, where I became chief [of] pediatric cardiology from 1972 to 1986. During my time here, I was also part of Children's Hospital, National Medical Center. I was also an assistant professor at George Washington University. Later, in 1976, when they opened up USIS, which is Uniform Services Medical, I was then brought on as the coordinator of pediatric cardiology for the Army, Navy, Air Force public health. I then achieved a rank of assistant professor at USIS. When I left in 1986, went to New Jersey where I joined the staff of Children's Hospital of New Jersey as an

> associate in pediatric cardiology. We were affiliated with UMDMJ, which is the medical school [where] I was given rank of associate professor.
>
> In ... 1988, I went to St. Joseph's Medical Center in New Jersey, and was the chief of pediatric cardiology there. We had rotations with people from Seton Hall. I was given the rank of associate professor of graduate medical education at Seton Hall University. Later, when we were trying to affiliate with Columbia Presbyterian, I was on their staff as an assistant professor of pediatrics, clinical pediatrics. Later, forgot the exact date, Mary Ellen Engle at Cornell granted me a clinical associate professorship in pediatrics.

Tr. 2 at 357-58. Dr. Connor is "board certified in pediatrics, and ... board certified in pediatric cardiology," the practice area he has worked in from 1970 to the present. Tr. 2 at 358, 360.

Dr. Connor began his testimony by discussing relevant facts in the clinical course of Thomas Kolakowski, from his birth to his untimely death. Thomas was born at 39 weeks (full term) at a weight of eight pounds, twelve ounces, with an Apgar score of nine at both one and five minutes. Tr. 2 at 360-61. The day after his birth (the 18th), he was vaccinated for Hepatitis B. Tr. 2 at 361. By the 19th, his mother allegedly began to notice odd behavior or symptoms in Thomas, which she referred to as "tremors," to which Dr. Connor added, "jitteriness is difficult to separate out from tremors." Tr. 2 at 361-62. Dr. Connor stated that Mrs. Kolakowski later raised a concern about an irregular, asymmetrical startle response in Thomas,[55] although Dr. Connor admitted that he did not "see it addressed in the physician's note" from that time. Tr. 2 at 362.

Dr. Connor recounted that, at a doctor's visit on 24 December 1998, the pediatrician noted healthy weight gain, and that "Once again a startle reflex was brought -- was not addressed per se, but he did also have loose bowel movements." Tr. 2 at 363. The Court remained unclear as to whether Dr. Connor was opining that a concern about Thomas' startle reflex was or was not raised. This ambiguity persisted:

> On December 24-25, the grandmother or mother-in-law brings up some remarks about the tremors. Once again, we have Christmas day. Nothing can be done about it. On the 26th, mom calls the baby's doctor again because she felt something wasn't correct.
>
> I have nothing really in the way of any notes that this was conveyed to the chart. On New Year's Day, which was December 31st, basically passes, and the child is seen for the first time after that phone call on the 2nd of January. Once again there is a very short note. There were no problems noted on that note. The doctor was silent as to whether or not the mother raised any concerns.

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[55] Dr. Connor explained the concern where a startle response is asymmetrical:

> When you have a startle response that is in question, then you begin to look for peripheral injury, whether it's been an arm or a leg, whether it's a muscle, a tear, a neurological injury such as an injury to the brachial plexus, for instance, or that you have a cerebral injury.

Tr. 2 at 362.

Tr. 2 at 363-64.

Continuing on, Dr. Connor recounted that Thomas was administered his second Hepatitis B vaccine on 20 January 1999, at 34 days old, at which time any newborn jitteriness should have passed, and jerky, involuntary movements would have become suspect. Tr. 2 at 364.

> [A]ccording to the mother's note on that visit, she brought up that he was not feeling well, that he had diarrhea, he was passive, and that's when he got the second shot. Now, they also brought up the incidents of tremors and basically at that point we have a transition timeframe. Going to the jitteriness of newborns, particularly full-term newborns, jitteriness is generally there for a few weeks.... Jitteriness now becomes a tremor that becomes in question the possibility of cerebral injury.

Tr. 2 at 364-65.

Following that visit, said Dr. Connor reporting from the affidavit of Mrs. Kolakowski, Thomas showed an increase in these so-called tremors and Thomas was not eating healthfully, which Dr. Connor described as "subtle signs of a baby that is possibly sick, and has just received a vaccination." Tr. 2 at 365. Similarly, Dr. Connor stated that, following that second Hepatitis B vaccination, Mrs. Kolakowski had to tend to Thomas more than usual, in order for him to go to sleep. *Id*. During one such time, Mrs. Kolakowski was holding Thomas to soothe him to sleep at around 3:30 in the morning, and herself fell asleep, only to awake to find that Thomas was not breathing properly, which prompted her to call for help from Mr. Kolakowski and emergency medical services. Tr. 2 at 365-66.

When EMTs arrived, they noted Thomas to be without a pulse, apneic, his skin still warm, but with flaccid extremities, dilated pupils, and blood coming from his mouth; there were no signs of trauma. Tr. 2 at 366. Their attempts to clear the airway of blood, or to resuscitate Thomas, were unavailing. *Id*. Dr. Connor restated the EMT impression of "cardiac arrest, wh[ich] was stated by a trained professional on the scene." *Id*. Resuscitation attempts continued on the way to the hospital and upon arrival, but were ultimately unavailing, and Thomas was pronounced dead, whereupon EKGs were recorded:

> [O]n the EKGs that the patient had after they stopped the CPR, one would see some agonal rhythm, showing that the heart was in the mode of dying at that time. Agonal rhythms are usually described as slow. They are described with the complexes, QRS complexes Y, AT can have prolonged QT intervals, but these cannot be used in the diagnosis of a cardiac injury. This is really what you would see at the end of life.
>
> ...Agonal rhythm became obvious after the CPR was determined to be nonproductive and that the child was clinically dead. The agonal rhythm can be seen after death for a period of time. But if the child had been dead for a longer period of time before EMT arrived, there would not have been an agonal rhythm.

Tr. 2 at 366-67. Dr. Connor summarized his opinion of the moment of Thomas' death, that "the child may have died just shortly before mother awoke, or may have had a cardiac arrest shortly before mother awoke." Tr. 2 at 367.

Dr. Connor summarized the autopsy:

> [T]he autopsy showed, at least on several organ systems, the brain, the heart, the liver, that there was enlargement; that these were significantly enlarged, and as they looked through the system, they looked through the child's body. They looked with x-rays. They found no injury to the skeletal system. They examined the brain. They examined the lungs. They examined the heart. They examined the GI tract, and they found no apparent abnormalities to explain the child's death.
>
> ...The forensics pathologist found no -- as far as abuse goes, they found nothing wrong specifically with the child, but there was a concern that this child had received in the interim two Hep. B vaccinations, and felt that these needed to be investigated further.

Tr. 2 at 368.

Following his restatement of the medical record, Dr. Connor stated his general opinion upon causation: "That mercury contained in these vaccinations can have cardiovascular effects." Tr. 2 at 369.[56] Dr. Connor then went into some detail, discussing the controlled timing which the central nervous system exercises over the heart's activity:

> The general pacemaker that usually all of us have and rely on is the sinoatrial node.[57] It is the portion of tissue that goes through its electrical discharge quickly. It returns quickly to baseline. It is ready to receive the next beat, and transmitted directly into the heart, and keep a rhythm of the heart regular and keep the heart, and this will respond to demands for the heart to increase its [rate] and decrease its rate.
>
> ...[T]he conduction system itself shows in this particular picture that the sinoatrial node goes through the internodal pathways to carry the electrical impulse in neurological type tissue to the AV-node,[58] which is the stopping station before the electrical current is distributed into the ventricles. From that particular point, the

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[56] *See also* Pet. Ex. 20, Dr. Connor's Expert Report, at 4:

> In my opinion, Thomas Kolakowski's death, more probably than not, was caused by the mercury contained within the vaccines that were administered to him on Days 2 and 33 of life. Early in his life, he demonstrated subtle signs of mercury toxicity which his care providers did not recognize since it was not known at the time that vaccines contained a mercury preservative.

[57] The sinoatrial node is "a microscopic collection of atypical cardiac muscle fibers (Purkinje fibers) at the superior end of the sulcus terminalis, at the junction of the superior vena cava and the right atrium. The cardiac rhythm normally takes its origin in this node, which thus is known also as the *cardiac pacemaker*." DORLAND'S, *supra*, at 1276 (emphasis in original).

[58] The atrioventricular node is "a small area of specialized cardiac muscle cells and fibers that receives the cardiac impulses from the sinoatrial node and passes them on toward the ventricles, introducing a delay in impulse conduction. It is located in the right atrium between the tricuspid valve and the orifice of the coronary sinus, is composed of a meshwork of (Purkinje) fibers continuous with the atrial muscle fibers and the bundle of His, and is supplied by a branch of the right coronary artery." DORLAND'S, *supra*, at 1272.

> electrical current is then put into the bundle of hysts [*sic*] which is in the septum between the right and left ventricle.
>
> ..[The s]eptum is the wall that begins at the AV valves. The right-sided one is the tricuspid. The left-sided one is the mitral. The septum then continues down to the tip of the heart or the apex of the heart and completes the wall dividing these two chambers from each other. The electrical impulse coming down through the right bundle along the septal wall, goes to the tip of the heart and then carries back the outside wall of the ventricle towards the tricuspid valve. The left bundle goes to the apex, enters into the left ventricular wall, and carries impulse from the apex back to the mitral valve.

Tr. 2 at 370-372. As for a normal heart rate, Dr. Connor qualified:

> Basically the range in adults can be between 60 and 100. In infants, it will be different. It will be higher because they normally have -- their hearts will run at a much higher rate because of the smaller volume of the ventricles.

Tr. 2 at 370.

Dr. Connor moved from there to discuss how an Electrocardiogram (EKG) can physically locate heart abnormalities, because different portions of the heartbeat's wave function correspond to different sections (chambers) of the heart:

> The first portion of the EKG, which shows a small hump, is that of the atrial contraction and forms what they call the P-wave. The P-wave is where the electrical impulse begins in the SA-node. As it travels through the interatrial conduction system you see a flat line, which is the baseline. That's called the PR segment. This is the time that it requires to go from the SA-node into the AV-node across the atrium. Generally, they can be less than 200 microseconds. Anything over 200 microseconds in the adult world can be considered a block, a first degree block, *et cetera*.
>
> The next portion that you see is the dip. The dip in there is called the Q-wave. The Q-wave itself is where you begin the electrical pathway or the electrical impulse from the AV-node down the septal wall. The Q-wave then turns, goes into a large, tall R-wave. This is the electrical impulse through the ventricle. It depends on where the electrodes are located on the precardial leads as to what this R-wave intends. If the electrical waves are being read from V-5 and V-6, this R-wave represents the left ventricle. If you are dealing with V-1, V-2, V-3, this would represent the right ventricle.
>
> As the electrical activity goes through and the R-wave returns to baseline, you have a dip below the line. The dip below the line is important because basically that is reflecting information of the contralateral[59] ventricle. If it's left for the R, the S-wave

[59] Contralateral means "situated on, pertaining to, or affecting the opposite side." DORLAND'S, *supra*, at 414.

> is for the right ventricle. If the R-wave is reflecting the right ventricle, then the S-wave is reflecting the left ventricle.
>
> Then at the point where the S-wave comes back to the isoelectric line, the straight line you see at the bottom there, is where you have what they call the J-point. The J-point is where all electrical activity has begun to cease, and now you're beginning the repolarization process. The repolarization process goes from the beginning -- from the J-point through the isoelectric point through the next large hump, which is the T-wave, and that then is the completion of the electrical cycle.

Tr. 2 at 373-75. In particular, studying the P-wave is useful for detecting a defect or irregularity in the atria:

> [T]he P-wave basically should be under 2.5 millimeters. When it is 2.5, more than 2.5 millimeters, you begin to look at the possibility that you're dealing with an enlargement of the small pumping chamber. If it is particularly tall P-waves on the right side, you are concerned about the possibility that there may be high pressures in the right ventricle, which you can see in cases of pulmonary hypertension. You can see that also when the valves leak and enlargement of the atrium once again, the tall P-waves will discern enlargement of the right atrium. If the P-wave looks like the letter "M" where it's wider and delayed, it may be what they call a P-mitral, which means the left atrium is enlarged.

Tr. 2 at 376-77.

> Dr. Connor's direct examination continued in this vein:
>
> Now, when you get to the AV-node, basically the distance between the beginning of the P-wave and the beginning of the Q-wave is what they call a PR segment. Normally speaking, the PR segment should show up with a faster heart rate. In the case where you have this elongated, then you begin to look for the possibility of a disease process in the muscle and electrical system, giving you a part of atrial block.
>
> Q Doctor, when you look at the PR interval, is that an indication that there is some sort of defect within the conduction system between the SA-node and the AV-node?
>
> A Yes.
>
> Q And could you tell the Court what the QRS complex represents?
>
> A Once again, this represent[s left] ventricular contraction....
>
> Q And then, Doctor, on this EKG, after the QRS complex, there is a line that leads into another little bump. What is that line called?
>
> A That's called ST segment. It is part of the repolarization process that begins after the contraction is complete.
>
> Q ...[W]here within the [EKG] would be the repolarization?

A Basically between where the S-wave comes into the isoelectric line, carried to the T-wave, and through the T-wave to where it comes back to the isoelectric line. That is called the ST segment, and the T-wave, the "repo" is the phase[] of repolarization of the ventricle, and basically it tells us how the muscle is behaving. ST segments are particularly important when you're looking at ischemic[60] changes. They do not act correctly. They can either be elevated or they can be dropped below the isoelectric line, and they tell you that there is something wrong with the way the heart is recharging itself.

Q And Doctor, you spoke about the T-wave earlier. What does the T-wave represent?

A The T-wave is a part that is divided initially into what they call the absolute refractory period. This is a time where you cannot stimulate the heart to contract, and then at the apex of the T-wave you start entering into what they call the relative refractory period. A relative refractory period is important because basically stimulus at this point either from outside sources or from abnormal beat or beats that run into the relative refractory period, and initiate abnormal heart rates. Examples are ventricular tachycardia.[61]

Q Doctor, would it be fair to state that once the T-wave begins its downward slope that's the period of relative refractory?

A Yes.

Tr. 2 at 377-379.

Continuing onward, Dr. Connor described the norm against which cardiac abnormalities are compared:

> The PR-segment and ST segment are on the same isoelectric line. The T-wave, when it goes back, should be on the same isoelectric line. As long as they are in those areas, your PR-segment basically -- I'm sorry -- your isoelectric line is telling you that these segments are in their normal position. It's when they vary in the PR-segment before the QRS or the ST segment after the QRS are raised above or below the isoelectric line outside the P-wave and outside the T-wave begins to tell you that there is a distortion going on.

Tr. 2 at 380.

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[60] Ischemia is the "deficiency of blood in a part, usually due to functional constriction or actual obstruction of a blood vessel." DORLAND'S, *supra*, at 954.

[61] Ventricular tachycardia is "an abnormally rapid ventricular rhythm with aberrant ventricular excitation (wide QRS complexes), usually in excess of 150 per minute, which is generated within the ventricle and is most commonly associated with atrioventricular dissociation. Minor irregularities of rate may also occur. Evidence implicates a reentrant pathway as the usual cause." DORLAND'S, *supra*, at 1850.

Next, Dr. Connor elaborated further on the significance of the "QT interval" as a diagnostic metric:

> QT interval is what you look at to determine whether or not that it falls within a certain normal pattern, and the general rate, the QT intervals that by themselves can vary with heart rates. The QT interval, if it is given out, may have a distance in them that can vary according to the size of the child, the rate of the heart rate at that point, and when you look at this you're looking to see is there a way that there is something going on that might make this QT interval longer.
>
> Now, the older method was just measure the QT interval by itself, and then compare it to known standards of what would be considered a norm. However, people felt that this was clumsy, so they corrected the heart rate to 60. There is a mathematical formula where the QT interval would give you a number that will vary anywhere from 380 to 440. QT intervals corrected to a heart rate of 60 becomes called QTC, corrected QT intervals. This is a simpler tool to use because the standard is the same for everybody from child to adult whereas the QT by itself varies according to age and weight of the child. So the QT corrected is what we use today to look for a problem.
>
> Normally speaking, QTs that are above 440 milliseconds are considered a borderline. You know, keep an eye on it. QTs above 480 are beginning a prolongation. QTs over 500 makes the refractory period much wider and more easily attacked so that if there should happen to be an abnormal heartbeat going on in the area of the QT correct -- the widened relative refractory period, that particular individual can have an abnormal heart rate begin. An example would be an extra beat, a premature ventricular contraction. If it lands in the relative refractory period, it can initiate and take over the control of the heart.

Tr. 2 at 382-84. Dr. Connor then spoke to the dangers threatened by an abnormally prolonged QT interval:

> You can have premature contractions and basically those premature contractions landing outside of the absolute refractory period become less of an issue than those who land inside.... At that point it changes the electrical activity in the heart, and instead of going back to the pacemaker in the SA-node, that portion of the heart can take over the control of the heart, initiate an abnormal heartbeat, but because it is not using the atrial/ventricular combination, you would begin to drop the pressures, the effective contraction of the heart muscle is impaired, the blood pressure can drop, and other scenarios can take place, including syncope, which is a passing out episode, or it can go on past that into a atrial flutter, and then into a fibrillation,[62] and at that point the blood pressures are no longer working, the blood pressure drops out, the

[62] Atrial fibrillation is "an arrhythmia in which minute areas of the atrial myocardium are in various uncoordinated stages of depolarization and repolarization due to multiple reentry circuits within the atrial myocardium; instead of intermittently contracting, the atria quiver continuously in a chaotic pattern, causing a totally irregular, often rapid ventricular rate." DORLAND'S, *supra*, at 695.

> flow of blood from the left ventricle drops, the flow into the coronary circulation drops, ischemia takes place, myocardial injury occurs, and unless resuscitation is begun the continued loss of blood pressure and the continued loss of oxygenated blood to the nervous system will then result in cerebral death.

Tr. 2 at 384-85. Dr. Connor agreed that "the reason it is dangerous is because it allows the development of abnormal rhythms." Tr. 2 at 385.

Dr. Connor then classified the different kinds of irregularities that arise from a prolonged QT interval:

> [Y]ou can have a wide-based QT interval which could involve the T-wave. You can have a double humped one which involves, once again, the division of the increase between the repolarization of the one ventricle being separated out from the repolarization of the other. There is indistinct ones where the termination of the T-wave is difficult to interpret because there can be a U-wave complex which could reflect problems with potassium and calcium.

Tr. 2 at 386. The electrolytes potassium and sodium are important because "potassium basically is important in the cell membrane active depolarization where you exchange sodium for potassium in order to allow for the contraction to take place.... the alternation of the sodium pump can be as much of a cause of a problem for prolonged QT as potassium, but potassium by in large rules." Tr. 2 at 387. Much later, he stated on the same topic of the risk of a prolonged QT interval:

> [With a prolonged QT interval,] there is an alteration of the repolarization process of the ventricle, and that when the relative refractory period is prolong[ed], it leaves it exposed to abnormal conduction, and at that particular point it sets the heart up for serious cardiac arrhythmia, such as a ventricular tachycardia. In the cases where it self-terminates, it's an annoyance. In the case where it doesn't, there is a death.

Tr. 2 at 395-96.

Following his general discussion of the heart and its rhythms, and QT interval irregularity in particular, Dr. Connor began his discussion of his opinion regarding the effects of mercury on the heart's proper functioning. The first article discussed by Dr. Connor was Dahhan,[63] which studied "people who were in Iran used seed grain that was contaminated with mercury, and as a result had mercury poisoning[, of which t]here were 42 patients involved, 28 were under 20 years of age, all EKGs were abnormal, and they were broken out into four grades." Tr. 2 at 388-89. He explained the study's results as follows:

> Well, basically, there were five grades, Grade 1 through Grade 4. Grade 1 was the mildest involvement. The Grade 4 was the most severe involvement.
>
> Basically, in Grade 1, there were five cases where there were only slight changes in the T-wave, which was the last of those complexes in the QT interval, were actually

[63] Pet. Ex. 18, Tab U, Shawkat S. Dahhan and Hussain Orfaly, *Electrocardiographic Changes in Mercury Poisoning*, 14 THE AMERICAN JOURNAL OF CARDIOLOGY 178-183 (August 1964).

> half the size. This means that there was some impairment of the repolarization process of the electrocardiogram. However, basically that would not get you into a lot of problems.
>
> Grade 2 was considered moderate. There was 10 cases, and in this particular case the ST segment, which it was the area between where the S-wave ended and the T-wave began. It was supposed to be on the isoelectric line, which is seen -- on the reference -- seen before the P-wave and after the T-wave. That segment was either up or down. In these particular cases ... that tells you that there [are] changes going on in the repolarization process that can be due to inflammation, which can alter the conductivity of the repolarization process through the injured area. If the injured area is compromised with decreased oxygenation, that zone of injury will increase. Whether it goes up or down depends on where these leads are seen on the chest. Basically they will all lead to areas in the heart, whether it is focal or it is global. In the case where the T-waves are inverted, this, once again, tells you that the repolarization, the axle [*sic*] refractory period, and the relative refractory period are changed and their repolarization tells you that the electrical process has been altered by the way these T-waves behave.
>
> Grade 3, in which there were 21 cases, is considered severe. In those cases, all T-waves were inverted and in those particular T-waves you also had other changes which were seen over the precordium[64] which would suggest that there is a severe myocardial injury, and when they talk about "all", they are talking about a global myocardial injury.

Tr. 2 at 389-91.

Dr. Connor discussed the types of arrhythmias observed in the Dahhan study, including "sinus arrhythmia," a change in atrial heartbeat that is somewhat voluntary, in that it is linked to inhalation (accelerates the heartbeat) and exhalation (decelerates the heartbeat) in breathing by the vagus nerve,[65] which is a cranial nerve. Tr. 2 at 391-92. Another form of arrhythmia noted was "ventricular beeps," which are "single abnormal electrical conductions through a zone in the ventricle where there was a delay, and as a result there is a distortion of the QRS complex," which are either isolated or multi-focal on the EKG, meaning the aberrant signals originate from different locations within the heart, which would then indicate "a more global involvement of the myocardium." Tr. 2 at 392. The relative danger presented by such irregularities depends on where in the rhythm cycle they occur:

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[64] The precordim is "the region of the anterior surface of the body covering the heart and stomach; it comprises the epigastric region and the inferior part of the thorax." DORLAND'S, *supra*, at 1499.

[65] The vagus nerve is the tenth cranial nerve: "descending through the jugular foramen, it presents as a superior and inferior ganglion, and continues through the neck and thorax into the abdomen. It supplies sensory fibers to the ear, tongue, pharynx, and larynx, motor fibers to the pharynx, larynx, and esophagus, and parasympathetic and visceral afferent fibers to thoracic and abdominal viscera." DORLAND'S, *supra*, at 1243.

> [W]hen you begin to see PVCs, this is an area where you can have injury that could either exacerbate with exercise. You could have these PVCs land in the wrong portion of the electrical complex, and in particular, if it lands in the relative refractory cycle called R-on-T, in other words, when you see the R-wave of the abnormal beat land on top of the T-wave, which is a repolarization one in the area of the refractory electrocardiogram, it generally doesn't give you a problem. But if that same abnormal R-wave lands on the relative refractory portion of the T-wave, that is, the portion where it's going down hill, then you can initiate a serious cardiac arrhythmia.

Tr. 2 at 393. The Dahhan researchers also observed other arrhythmias among those studied:

> They basically had alternation of the electrical conduction from the P-wave to the Q-wave, giving you various forms of heart block. There was also a short run of ventricular tachycardia encountered in two cases. A ventricular tachycardia obviously was a short run, so even though it was there it was self-terminated, but right there you're no notice [*sic*] that this is a more serious problem.

Tr. 2 at 394. Based on this reading, Dr. Connor agreed that the Dahhan study subjects experienced cardiac symptoms in addition to neurological symptoms. *Id*.

The relevance of those cardiac symptoms to the issue at bar was less clear. While clear that "only a portion" of the people that were exposed to mercury experienced these arrhythmias, Dr. Connor did not believe that this fact was "an indication of a genetic susceptibility toward developing cardiac disorders" in response to mercury exposure or toxicity. Tr. 2 at 394-95. He did agree that "there are genetic cases where you can have an abnormal set of beats going into a ventricular arrhythmia, and resulting in sudden death in what appeared to be an otherwise normal heart," which would support causation independent of mercury exposure, rather than indicate especial genetic susceptibility to mercury poisoning. Tr. 2 at 395.

The next article of medical literature discussed by Dr. Connor was the Cinca article[66] from 1979, wherein "[an ethyl] mercury-containing compound in fungicide resulted in contamination of meat products which were consumed by" the study's subjects, a family in Romania. Tr. 2 at 396. One son had poisoning so severe that he lost the ability to walk, and had difficulty in talking and suffered muscle wasting. Tr. 2 at 396-97. His electrocardiogram was irregular, and he died three days following admission to the hospital from mercury poisoning, *via* cardiac arrest. Tr. 2 at 397. Another son evidenced similar symptoms ("difficulty walking, difficulty talking, difficulty walking, weakness, abnormal reflexes"), and, within 10 days of hospital admission, likewise died *via* cardiac arrest. *Id*. According to Dr. Connor's reading, the third child in the family, a teenaged daughter, evidenced:

> [A]bnormal electrocardiogram which showed the fact that the ST segments were found below the isoelectric line, and were negative. There is not a big description

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[66] Pet. Ex. 18, Tab V, I. Cinca *et al.*, *Accidental ethyl mercury poisoning with nervous system, skeletal muscle, and myocardium injury*, 43 JOURNAL OF NEUROLOGY, NEUROSURGERY, AND PSYCHIATRY 143-49 (1979).

> other than for the fact that this was a global type of ST segment change, indicating that there would be some kind of an inflammatory or ischemic activity going on within the heart itself.... [A]ccording to the reading of the electrocardiogram, the reader ascertained that this was left ventricular hypertrophy,[67] which would be seen in the QRS complex, which is the large spike, generally found over the chest and reads 4, 5 and 6, which is on the outside of the precordial over the left ventricle.

Tr. 2 at 397-98. The fourth subject was the family's mother, who, Dr. Connor related, "had weakness, abnormal gait, muscle pains, basically she had unusual or abnormal high eye movements." Tr. 2 at 398. He added that the result of her electrocardiogram was "flattening of the T-waves in the precordial lead which, if we go back to the Dahhan article..., this would indicate a milder form of cardiac involvement." Tr. 2 at 398-99.

Most interesting for Dr. Connor, the two brothers that died in the Cinca study both suffered from "interstitial myocarditis":

> There was involvement of both in case one, and it was chronic, because this is one where the consumption of meat took place before the symptoms took place, and then from the symptoms to the involvement of the heart to the final death. I think that this is over a 28-day period of time. The findings at the time of autopsy on both case one and case two was an interstitial -- the call it interstitial chronic myocarditis.[68] ...[This i]ndicates that there is an inflammation within the myocardium which makes up the ventricular walls of the heart.

Tr. 2 at 399. He added that the mention of "chronic myocarditis" meant that the myocarditis had persisted for longer than 21 or 30 days. Tr. 2 at 400. Dr. Connor stated that the Cinca study "supports [his] belief that ethyl mercury affects the cardiovascular system." Tr. 2 at 400.

The next study discussed by Dr. Connor in explanation and support of his opinion was the Jalili article,[69] which studied another Iraqi population that had consumed seed grain coated with EMPTS, an ethyl mercury-containing preservative (but not the same population studied by the Dahhan paper). Tr. 2 at 402. He found "indications of cardiovascular effects from the administration of seed grain treated with ethyl mercury." *Id*. He noted the following findings:

> [T]here were several cases of irregular pulses, and very slow heart rates. In other cases, they once again showed ventricular ectopic[70] beats with prolongation of the QT

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[67] Ventricular hypertrophy is "hypertrophy of the myocardium of a ventricle, due to chronic pressure overload; it is manifest electrocardiographically by increased QRS complex voltage, frequently accompanied by repolarization changes." DORLAND'S, *supra*, at 890.

[68] Interstitial myocarditis is "inflammation of the muscular walls of the heart ... affecting chiefly the interstitial fibrous tissue." DORLAND'S, *supra*, at 1212.

[69] Resp. Ex. A, Tab 27, M. A. Jalili and A. H. Abbasi, *Poisoning by Ethyl Mercury Toluene Sulphonanilide*, 18 BRITISH JOURNAL OF INDUSTRIAL MEDICINE 303-308 (1961).

[70] Something is ectopic if it is "located away from normal position." DORLAND'S, *supra*, at 587.

> interval, and depression of the ST segment with T-wave inversion, which is a very serious involvement of the myocardia.
>
> ...[Among the EKG readings,] there were six records where they had prolonged PR intervals. They had irregular heart beat. The ST segment with the depression, the ST segment and the inverted T-waves would indicate severe myocardial damage.... They did 12 EKGs on 15 patients, six were within normal limits, others showed involvement.

Tr. 2 at 403. He agreed that some of those poisoned did not manifest cardiological symptoms, but only neurological symptoms, and added that the neurological symptoms observed in the Jalili study were "very similar to the neurological injuries we just spoke about in Dahhan and in Cinca articles." Tr. 2 at 403-04.[71]

The last article Dr. Connor discussed was the Zhang[72] article, among which he found the following cases cardiologically significant:

> Seven patients showed prolonged QT intervals with depression of the ST segment, inverted T-waves, and large E-waves, which would be consistent with low serum potassium levels, and basically the EKG findings are consistent with a toxic carditis.

Tr. 2 at 404-05. These were significant to him because they demonstrated to him the following EKG abnormalities:

> In the initial part of the EKG, in the PR segment, there was a prolongation above -- it would have to be above 200 milliseconds in order to have first degree AD block. So that involves the SA-node, the electrical activity through the atrial wall to the AV-node, which is where the ventricles begin. So there is clearly involvement of the upper portion of the small collecting chambers of the heart.

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[71] On cross-examination, Respondent queried Dr. Connor on the differences between the Jalili study and others, and between individual subjects in the studies:

> Q Is it your opinion that the patients in the Jalili papers and the Dahhan papers were exposed to the same amount of mercury?
>
> A They were exposed to a similar type of mercury, yes.
>
> Q Isn't it likely that they were exposed to varying amounts of mercury?
>
> A Yes.
>
> Q Can the symptoms described in those papers then be attributable to the varying doses of mercury received by the study populations?
>
> A I don't know how to answer that one.

Tr. 2 at 471-72.

[72] Pet. Ex. 18, Tab NNNN, Jimel Zhang, *Clinical Observations in Ethyl Mercury Chloride Poisoning*, 5 AMERICAN JOURNAL OF INDUSTRIAL MEDICINE 251-58 (1984).

> The second one is that patient had prolonged QT intervals which basically start at the point where the V-wave -- it looks like a small "v" on the electrocardiogram -- to the end of the T-wave. Once again, greater than 480 milliseconds using a corrected QT as a guideline. At this point they are also saying that the ST segments, which come at the end of the contraction of the ventricle, that's were the S-wave comes into this segment, that connects the QRS complex to the T-wave.
>
> That ST segment once again is showing signs of inflammation or ischemia because it's raised or is lowered, depending on where the lead is being read, indicating that there is an abnormality in the repolarization process either from lack of oxygen, inflammation, injury.

Tr. 2 at 405-06.

These articles of medical literature led Dr. Connor to conclude that "ethyl mercury can cause cardiovascular abnormalities," which "can lead to cardiac arrest and death." Tr. 2 at 406. More specifically, Dr. Connor concluded that "the ethyl mercury which was contained in the two doses of thimerosal, which was in the Hepatitis B vaccine that Thomas received ... substantially contributed to [his] death." Tr. 2 at 406-07. He explained the basis for his conclusion as follows:

> Well, first of all, the problem with giving vaccinations on birth days is that you do get a very large dose of thimerosal in relation to the child. This is kind of a one size fits all. The biggest problem of one size fits all, and the timing of this is important because in the case where one size fits all you don't make any adjustments for the weight.
>
> Number two, you're giving it in a very critical timeframe in a child's entry into the world. The reason it's critical is because for the first couple of weeks of life we are still going through the maturing process of the infant. The cell membrane transport system is immature. The liver, which can produce protein that combined mercury, are immature and therefore will not do the job. The kidneys are immature, cannot eliminate it. The GI tract is immature because it's in the process of colonizing so that it can metabolize the bile in order to lock down the mercury. At this point the amount of mercury that is introduced can be extremely high against standards.
>
> The second thing is you can have, as a result, sensitization of the individual to the product, and when you have that occurring you now begin the process, an inflammatory -- antigen antibody process that can begin to show as these articles, that the problem can build over the next several weeks.
>
> The second dose in a person that is already sensitized to the ethyl mercury can be a triggering of an extremely severe reaction which can take place over a matter of hours, and at that particular point from the autopsy there is involvement of the brain, there is involvement of the heart, there is involvement of the liver. These are all suggestions that there has been an ongoing inflammatory process.

Tr. 407-408. He added some caveats that arose from the facts in this specific case:

> Basically, I'm dealing with the articles and I'm basically dealing with certain things that were a little bit problematic. One of the items that were problematic was the continued jitteriness of the child. Based on the development of children, jitteriness should subside in a few weeks. It did not according to the mother. Once you start to see jitteriness continue, then you begin to worry about the possibility that there is an involvement of the nervous system.
>
> In the case of people having jitteriness with an usual startle response, then you begin to worry about the possibility that either the child is not hearing well or there is an injury in the brain stem. In those particular cases the response then is abnormal, and although people don't generally know startle response in the first child, they begin to learn, oh, is this a normal type of response, and in the course of conversation, they can be told, you know, it's normal.
>
> I've looked at the medical record and I don't see referrals to it, so when one puts down nervous system, *et al.*, and haven't responded to it, I don't know what it means, but I have to go with the Petitioner who said that she felt it was abnormal. We have a third party, which is either a mother or a mother-in-law, who felt it was not right, and the fact that this is a second time mother, she should know what to expect.

Tr. 2 at 409-10.

On cross-examination, Dr. Connor admitted that he is not an expert in toxicology or medical toxicology, nor is he board-certified in either specialty. Tr. 2 at 411. Though he did observe one case of mercury poisoning in the form of acrodynia[73] during his pediatrics residency, he has never diagnosed anyone with clinical symptoms of mercury poisoning nor for "toxic exposures to chemicals, pharmaceuticals or environmental toxins." Tr. 2 at 412-13. Among the nine peer-reviewed medical articles he has written, none were in the field of toxicology, none on either methyl or ethyl mercury, none on thimerosal or vaccines, none on treating mercury toxicity, none on the cardiologic sequela of ethyl mercury. Tr. 2 at 413-14. Likewise, Dr. Connor does not claim to be an expert in neurology, has not received special training in neurology, is not board-certified in neurology, has not authored medical literature in the field of neurology, and does not treat or diagnose neurologic conditions in his patients. Tr. 2 at 414. He has not authored articles of medical literature on "neurologic effects of mercury poisoning," "the neurologic effects of ethyl mercury exposure," or "the neurological effects of methyl mercury exposure." Tr. 2 at 414-15. Similarly, Dr. Connor does not claim to be an expert in immunology, has not received special training in immunology, is not board-certified in immunology, and has not authored medical literature in the field of immunology. Tr. 2 at 415.

Moving to Dr. Connor's area of expertise, cardiology, regarding whether he has "studied the effects of either ethyl mercury or methyl mercury on the heart," Dr. Connor stated that he had, as part

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[73] Acrodynia is "a disease of early childhood characterized by pink, swollen, painful fingers and toes; listlessness, irritability, failure to thrive, and photophobia; rashes, profuse perspiration, loss of teeth, and sometimes redness of the cheeks and tip of the nose. Most cases are toxic neuropathies caused by mercury poisoning; individual sensitivity may also be a factor." DORLAND'S, *supra*, at 20.

of his preparation for testimony in this matter, but that it was limited to reviewing relevant articles of medical literature, and did not extend beyond that. Tr. 2 at 415-16. Dr. Connor had not written any articles, published in peer-reviewed journals specializing in cardiology, on the topic of effects on the heart from ethyl mercury or thimerosal-containing vaccines, nor on the cardiac nerve plexus in particular. Tr. 2 at 416.

Dr. Connor agreed that "dose is one of the most fundamental concepts in the field of toxicology" and "is a fundamental consideration in prescribing medications" within "the field of cardiology." Tr. 2 at 417. He agreed "that prescribing medications that are considered beneficial at one dose can be toxic or lethal at another dose," and "that toxicity occurs when the dose any substance is sufficient to cause adverse symptoms or clinical manifestations." Tr. 2 at 417-18.

Dr. Connor clarified his opinion on causation:

> In the case of this type of a situation, we can have a sensitization, antibody antigen type reaction, which can take place at the tissue level, and does not necessarily have to be absolutely the mercury in the heart.

Tr. 2 at 418. He added that this would mean an immunologic reaction to "the products in the vaccination," such as the thimerosal, the antigen, or "the breakdown components." Tr. 2 at 418. This led to the following, rather odd, interchange:

> Q When you say "antigen", are you referring to the Hepatitis B surface antigen?
>
> A Possibly.
>
> Q And when you say "the breakdown components", what are you speaking of specifically?
>
> A Well, basically the components that make up a vaccination, which can be the antigen component, the ethyl mercury. The ethyl mercury can break down into -- I'm sorry, the thimerosal which can break down into ethyl mercury and thiosalicylates, and the aluminum hydroxide.
>
> Q So any one of those components could be the source of the sensitization and the inflammatory reaction that you spoke of?
>
> A They are components.
>
> Q But my question was, any one of those components, in your opinion, could be the source of the inflammatory reaction.
>
> A The inflammatory reaction can most likely be due to the ethyl mercury, certainly the possibility of the antigen itself.
>
> Q What about the aluminum?
>
> A I don't know.
>
> Q So we're going to limit it to most likely the ethyl mercury or the Hepatitis B surface antigen, is that correct?
>
> A Yes.

...

Q And the mechanism, in your opinion, is an inflammatory reaction?

A Yes.

...

Q Was the first vaccine the cause of Thomas Kolakowski's death?

A The first vaccine can be the sensitization. The second vaccine was the cause of the death.

Tr. 2 at 418-19.

Respondent challenged Dr. Connor's expressed opinion, that "Thomas Kolakowski suffered an immunologic reaction to either the ethyl mercury in the thimerosal or the Hepatitis B surface antigen," and that "the first dose sensitized him and the second dose killed him." Tr. 2 at 422. Respondent's challenge was premised upon the perceived absence of such a theory within Dr. Connor's written expert report, submitted prior to the hearing, but Dr. Connor believed that the immunologic theory was "implied in the mercury toxicity, and basically it was implied in the fact [Thomas] received a vaccination on the first day of life and again 33 days later." Tr. 2 at 423. He went on to explain his opinion on the relationship between mercury toxicity and immunologic reaction:

> Mercury toxicity basically is the thimerosal which breaks down into the ethyl mercury and to the thiosalicylates. These elements can cause an immune reaction, and these basically can create an inflammatory basis which can damage cells, and at this particular point if it involves the heart, you will end up with a myocarditis.

Tr. 2 at 423. Furthermore, he agreed that dosage was a factor for both mercury toxicity and immunologic reaction:

> Simply by an increased amount in the system that normally might be able to adjust for smaller amounts in the case of infants where you have basically an intent to stimulate the immune system which is what vaccines are intended to do, to stimulate the immune system so that they can produce a response at a later date when the disease takes place. And at that particular point I would say what can be a therapy for one could be a poison for another, particularly when you are dealing with doses that are not individualized but one size fits all, which is what this infant received.

Tr. 2 at 424.

When asked to explain the interplay of this immunologic response across the neurologic and cardiologic systems, Dr. Connor explained:

> [I]f you have an immunologic reaction in which you're going to have the production of the antibodies ... or the antigen can lock into the system, it can involve your immune system, your heart or the cardiovascular system, and the neurological system.

Tr. 2 at 425. Dr. Connor further explained his belief that this was an ongoing inflammatory process, which began following administration of the first Hepatitis B vaccine, and continued until his untimely death:

> [T]he child had enlarged cerebral edema, enlarged heart, enlarged liver, and these things usually are based on chronicity; that is, you would have to have a period of time that the process was going on. It can be aggravated and accelerated when the next dose is put in place, and then the problems would begin -- the final stage would begin at that point.

Tr. 2 at 426. Dr. Connor stipulated that such a mechanism of inflammatory reaction "is independent of the effects of mercury on the brain." Tr. 2 at 427.

Dr. Connor could not "cite to any articles that show that inflammatory reactions in the heart are more common in children who received thimerosal-containing vaccines than in children who have not." Tr. 2 at 427. Dr. Connor could not cite support for his expert report's assertion that, "The IOM has acknowledged that mercury can cause cardiac and respiratory adverse effects." Tr. 2 at 427-28.

Respondent also challenged the statement within Dr. Connor's expert report that, "Most articles have treated organic mercurials as one, and have not distinguished between ethyl and methyl mercury," regarding what he meant by that statement:

> Basically, mercury compounds are basically similar in nature. They are all toxic. There is no articles that show that they have a benefit. They basically are deleterious to the body.... [However,] dose does matter also.

Tr. 2 at 429.[74]

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[74] This led to the following interchange, which was useful for the Court's assessment of Dr. Connor's persuasive probity:

> Q Do organic mercurials have the same chemical properties?
>
> A I don't understand what the question would be.
>
> Q What organic mercurials are you referring to?
>
> A Basically, ethyl mercury and then you can have methyl mercury, and they have certain properties that are similar in nature.
>
> Q But do they have the same chemical properties?
>
> A I'm not a chemist.
>
> Q I'm sorry. I didn't hear you.
>
> A I'm not --
>
> Q You're not a chemist?
>
> A -- a toxicologist.
>
> Q Okay. Do they have the same adverse effects on the body?
>
> A They can have the same adverse effects on the body.

Dr. Connor did agree that the studies of mass mercury poisonings in Iraq and the Minamata Bay of Japan "involved exposure to high doses of methyl or ethyl mercury compounds due to ingestion of contaminated food," and that "the amount of mercury involved in these poisonings far exceeds the exposure to ethyl mercury in thimerosal-containing vaccines." Tr. 2 at 433. Dr. Connor's comment in his expert report, that, among those mass poisoning cases, "significant numbers ... develop[ed] signs and symptoms which are not attributable to mercury toxicity," those signs and symptoms he was referring to were "involvement of the cardiovascular system as shown on the electrocardiograms." Tr. 2 at 433-34. He stipulated that the deaths reported in those studies were not immediate, but followed two to three or four weeks after mercury exposure. Tr. 2 at 434.

Q Does dose level matter?

A Dose level would matter.

Q Are there any articles you can refer me to that support your opinion that organic mercurials are treated as one?

A I think Lucier is probably the one person who has looked at a lot of these particular products, and I'm relying on his statements that says that they do.

...

Q Are you aware of any articles that distinguish ethyl mercury and methyl mercury?

A Yes.

Q And what articles are they?

A Ethyl mercury --

(Pause.)

THE COURT: Perhaps, ma'am, if you have a specific article in mind, you could indicate that.

MS. DAVIS: I do not. I'm just asking the basis of his opinion, and if he knew of any articles that distinguish between them.

(Pause.)

THE WITNESS: There are two but I just don't remember where they are located.

Tr. 2 at 429-30.

Respondent's questioning unearthed some fundamental errors and unfounded leaps of logic in Dr. Connor's opinion.[75] Whenever Respondent confronted Dr. Connor with one of these, however, Dr. Connor often answered that he was not qualified to opine on the topic.

After noting Dr. Connor's statement about the half-life of methyl mercury in his expert report, Respondent asked him if he knew how that compared to ethyl mercury, which led to the following interchange:

> Ethyl mercury by itself can clear in a shorter timeframe, but it accumulates over a longer timeframe elsewhere. It's not eliminated from the body.[76]
>
> Q If we assume that for the sake of argument the half-life of ethyl mercury is on the order of seven days, does that affect your opinion whether Thomas Kolakowski's death resulted from his exposure to thimerosal in his vaccines?
>
> A I don't know how to answer that because basically you would have to be more of an immunologist in order to make that kind of judgment call. I'm not an immunologist.

Tr. 2 at 435. Petitioners did not offer any expert witness evidence on the subject of immunology.

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[75] Dr. Connor's expert report stated, *inter alia*:

> A review of the literature has revealed some very interesting studies on the variation of the biologic half-life of methyl mercury in man. The range appears to be approximately 40 to 120 days, with an average of roughly 70 days. Exceptions were noted, however, which suggests that from a public health point of view, a certain group of people may face a much higher risk of a longer biological half-life than others with the same mercury burden. This indicates that there are individuals who may genetically be predisposed to a prolonged biological half-life....
>
> ...While the early reports all treated the various forms of organic mercury alike, recent, improved methods of analysis seem to indicate that ethyl mercury in the same dosage as methyl mercury is much more potent. (Rosenstein, 1960; Sonsjn, 1973; Toxicity study for USPHS and EPA, 1989). In addition, it is important to realize that inorganic mercury in high exposures can cause death due to loss of respiratory function as a result of severe pulmonary damage (Campbell, 1948; Matthes, 1958; Tangerberman, 1959; Tenet, 1959). In these exposures, the animals suffered severe pulmonary edema within 24-48 hours following an acute exposure to inhaled forms of mercury.
>
> Recent data has indicated that depending on the age of the subject, ethyl mercury at the same blood level appears more toxic than methyl mercury. The increased toxicity appears to occur for several reasons: (1) ethyl mercury compounds take longer to cross the blood:brain barrier and achieve peak levels; (2) demethylation occurs in the central nervous system leaving higher amounts of inorganic mercury to accumulate in the CNS; (3) apoptosis (accelerated programmed cell death) and necrosis of cells occur simultaneously in ethyl mercury exposures rather than sequentially as in methyl mercury exposures; (4) ethyl mercury appears to be more lipophilic than methyl mercury thereby having a greater affinity for tissues which contain fat such as the nervous system tissue, the neuronal tissue, the deep cardiac nerve plexus. In addition, mercury can also affect the sympathetic and parasympathetic systems thereby affecting the regulation of cardiac speed, function, rhythm and contractility, timing intervals of the PR interval, and the QRS and QTC which affects the rhythmicity of the heart *per se*.

Pet. Ex. 20 at 2-3.

[76] This statement is belied by the data from, *inter alia*, the Burbacher study, *supra*.

Regarding Dr. Connor's reference in his expert report to a population segment genetically predisposed to mercury toxicity, Dr. Conner stated that such a concept "was alluded to in one of the [Iraqi mass poisoning] studies," but qualified that "I can't quote exactly where the paper is located at this point, but they did studies on people, and there was at least one case where it went to 189 days, and that was felt to be a genetic difference in those particular individuals or that particular individual," before finally admitting that genetic predisposition to mercury toxicity was not proven or demonstrated in the paper's findings, but "was speculation." Tr. 2 at 435-36 ("This was alluded to. It was speculation."). When pressed to state his opinion based on his own expertise, regarding whether "genetically predisposed individuals are at the tail-end of the ... dose response curve," Dr. Connor responded, "I have no way of telling because, once again, that's genetics medicine." Tr. 2 at 436-37. Dr. Connor stated that he based his statements on genetic predisposition on geneticists:

> Some of the information from there comes from the articles that were written for the PA. There is a tremendous amount of information on people who have had abnormal responses to it, and it's alluded to that people are different genetically, and would have a different -- and some people have a different response.

Tr. 2 at 437. Respondent also queried Dr. Connor for support of his statement, in his expert report, that "The exceptions were noted which suggest that from a public health point of view a certain group of people may face a much higher risk of a longer biological half-life than others with the same mercury." Tr. 2 at 438. Dr. Connor could point to no specific support for that statement. *Id*.

Regarding the facts of this case, Respondent asked Dr. Connor whether he believed "that Thomas Kolakowski had a genetic predisposition," and he replied that he did not, and that he was not aware of any "mercury testing done on Thomas Kolakowski." Tr. 2 at 441.

Dr. Connor agreed to the propositions that "the route of exposure, meaning whether it was inhaled, ingested or introduced via IV or subcutaneous ... is also important in assessing the effects in the body," that "the toxicokinetic profile of mercury exposure differs depending on the form of mercury, the route of exposure to the mercury, [as well as the] size of the dose, ... [a]nd the age at the time of exposure" and that "dose is an important factor in assessing the adverse effects." Tr. 2 at 441-42.

There was some difficulty arriving at an understanding as to what Dr. Connor meant by his use of the word "potent" within his expert report's statement that "Improved methods of analysis seems to indicate that ethyl mercury in the same dosage as methyl mercury is more potent." Tr. 2 at 442 *et seq*. Dr. Connor began his explanation by citing "a study done in 1985, I believe it was 1984-85, on rats which basically gave a very specific dose to these rats of methyl mercury, of ethyl mercury, and methyl mercury basically shows that at two, four, six, eight, [and twelve] hours it had almost the same response as ethyl mercury." Tr. 2 at 442-43. Dr. Connor continued summarizing that article, as follows:

> However, when they continued on to 24 hours, methyl mercury plateaued off and dropped, ethyl mercury went up another 20 percent before it began to fall, which indicated that ethyl mercury peaks out much later than methyl mercury, and in that particular case you are showing a difference in absorption, and you're showing that

> many of the studies done were done at two, four, six, and eight hours, and never completed their studies until -- at 12 or 24 hours. So that some of the information that was given over basically led to the belief that they both were acting similarly when in fact there was a difference in the way they were absorbed and the peak level of ethyl mercury was far greater and later than that of methyl mercury.

Tr. 2 at 443. No reference to such an article was ever provided.

Respondent then challenged Dr. Connor with Dr. Magos' 1985 article[77] "regarding ethyl and methyl mercury, which, Dr. Connor conceded, had "conclude[d] that at equal doses methyl mercury is more toxic than ethyl mercury." Tr. 2 at 443-44. Dr. Connor explained, "the method of analysis was altered in 1985 by Dr. Magos. At that point they changed the extraction method of ethyl mercury ...[such that] they were able to extract more ethyl mercury than ... previously." Tr. 2 at 444. His summary continued:

> [A]t that point they actually were able to get more ethyl mercury out of the sample, which would imply that there was greater toxicity than previously anticipated.... Or greater amounts which may, as we were talking about dose-related, was now the knowledge that the amount was at a greater dose than previously thought.

Tr. 2 at 444-45. When asked if his opinion was that Dr. Magos' methodology was faulty, Dr. Connor replied:

> I'm saying Dr. Magos altered the extraction method by changing the amount of stannous chloride to a lower amount so that they were extracting more ethyl mercury than previously was done, and that by altering that they had a better method for identifying how much of the ethyl mercury was actually in the system.
>
> Q But ultimately his conclusion was that in equal doses ethyl mercury was less neurotoxic than methyl mercury.
>
> A I don't recall that.[78]

Tr. 2 at 445.

Returning to the meaning Dr. Connor intended by his description of ethyl mercury's "potency," Dr. Connor explained:

> [T]he understanding of the amount of ethyl mercury was at a different level than they previously thought. I don't know that that makes it more toxic because it was already being judged, but it would tell you that there were higher levels of ethyl mercury than previously thought, and based on the previous analysis if one were to look at a drug that you were studying and you were trying to analyze, and you found a specific level

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[77] Pet. Ex. 18, Tab YY, L. Magos *et al.*, *The comparative toxicology of ethyl- and methylmercury*, 57 ARCHIVES OF TOXICOLOGY 260-267 (1985).

[78] However, Dr. Connor had just stipulated to this summary of Dr. Magos' conclusion moments before. Tr. 2 at 444.

that was no different than methyl, then you would say ethyl and methyl were the same.

But if you basically come out and say that the ethyl was actually at a higher dose than previously believed, and the amount used to obtain that or to make that dose gives you a higher dose of ethyl mercury than you previously thought, then the dose is important because it would have to be lower in order to be the same as methyl mercury.... And larger amount of ethyl mercury would imply that there was more ethyl mercury available at that particular dose level than people understood.

Q So the higher amount available is the same as potency to you?

A No.

Q What did you mean by more potent?

A More available.

Tr. 2 at 446-447.

Regarding that same sentence of his report, Respondent queried Dr. Connor as to what "improved methods of analysis" he was referring to, which, to him, had indicated the greater potency of ethyl mercury. Tr. 2 at 448. Dr. Connor's response was that the literature he cited for support was not applicable, but that "the improved method of analysis was basically Magos, 1985." Tr. 2 at 448-49. When Respondent asked about the studies he listed in support, Dr. Connor conceded, "No, these were about methyl mercury, and basically once again both before the recognition that there was a difference ... for ethyl mercury." Tr. 2 at 449. Ethyl mercury was never studied by the papers he referenced, and "[t]hey only deal with methyl mercury." Continuing on, Dr. Connor stated his summary of Magos' findings, conceding that the Magos study does not conclude "that ethyl mercury, in the same dose as methyl mercury, is more potent," but rather, "that they [could] extract ... more ethyl mercury using the revised method ... from the sample." Tr. 2 at 450. As Magos stands for the exact opposite of Dr. Connor's proposition, it is not overstatement to find that Dr. Connor's stated opinion on this point, in his expert report and at the hearing, are incorrect, and do not persuade the Court on the issue(s) it addresses.

Later, Dr. Connor discussed another statement from his expert opinion that was based on his (faulty) understanding of the 1985 Magos article:

Q ...At the bottom of page 2 and onto page 3 you state, "The recent data has indicated, depending on the age of the subject, ethyl mercury at the same blood level appears more toxic than methyl mercury." You don't provide any citations. Could you tell me what you were relying on?

A Basically, I was relying on Magos, '85, the understanding that the amount of ethyl mercury turned out to be higher than the methyl mercury that some of the statements from Lucier, who is a toxicologist, implies that ethyl mercury, in his opinion, is more toxic than methyl mercury. I'm relying on his statements. He's a toxicologist, not me.

Tr. 2 at 454.

When challenged on another portion of his expert report, Dr. Connor could not point to "any studies that show loss of respiratory function or pulmonary damage as a result of exposure to thimerosal-containing vaccines," and apparently was not sure whether such a study existed. Tr. 2 at 452. He conceded that the studies he cited in his expert report, regarding "severe pulmonary damage," "all dealt with mercury vapor inhalation," *i.e.*, inhalation of inorganic, elemental mercury. Tr. 2 at 452. He agreed that those studies were "not relevant" to the issue at bar, and agreed that, to his knowledge, Thomas Kolakowski had not been exposed to elemental mercury vapor. Tr. 2 at 453-54. Dr. Connor's expert report had blurred the distinction between organic and inorganic mercury, and when Respondent asked whether "the [] ethyl mercury compound or the inorganic mercury that led to Thomas Kolakowski's sudden death," Dr. Connor's only response was "I wouldn't know. That's a chemist's decision. That's a toxicologist's call." Tr. 2 at 456. Unfortunately for Petitioners in this particular case, Dr. Lucier offered no opinion on that question. Tr. 1 at 146.

Dr. Connor defended his understanding that because "ethyl mercury is slower to cross the blood brain barrier," this makes "ethyl mercury more toxic than methyl mercury," with the reasoning that "[ethyl mercury] crosses that over a longer period of time and at a higher level." Tr. 2 at 455. He said this reasoning was supported by "a study that was done with the rats which showed that basically at 12 hours methyl mercury peaks out, at 24 hours ethyl mercury peaks out and at 20 percent higher," that he would forward to Petitioners later on, but could not name at the time of the hearing. *Id*. Dr. Connor used the same explanation for his expert report's statement when Respondent asked what he was relying on for support of his statement that "[apoptosis] and necrosis occurs simultaneously in ethyl mercury exposures versus sequentially in methyl mercury exposures"–he could not provide the citation then, but would later. Tr. 2 at 456-57. He never did.

To the Court's mind, this is unacceptable. Dr. Connor had over two years between the writing of his expert report and his hearing testimony to familiarize himself with the medical literature upon which he (ostensibly) based his opinion. He knew, or should have been informed, that he would be held to account for the basis of his expert opinion at that hearing–indeed, that was the primary reason for his appearance that day. His lack of preparation made his testimony largely unhelpful to the Court, and did not allow Respondent to cross-examine the source of his opinions. All of this makes his opinion testimony less persuasive.

When Respondent pressed further on the subject of apoptosis,[79] and asked what relation that process had to do with "thimerosal-containing vaccines causing sudden cardiac arrest," Dr. Connor stated that there was no relation, because it is "a chronic condition." Tr. 2 at 457. He elaborated:

> Basically, apoptosis requires a stimulation of the immune system to produce various leutokines. These basically can cause injury to cells which can result in early death, but this is not something that happens right away. This is something that occurs over a longer period of time.

[79] Apoptosis (from the Greek for "a falling off") is "a morphologic pattern of cell death affecting single cells, marked by shrinkage of the cell, condensation of chromatin, formation of cytoplasmic blebs, and fragmentation of the cell into membrane-bound apoptotic bodies that are eliminated by phagocytosis. it is a mechanism for cell deletion in the regulation of cell populations ... Often used synonymously with *programmed cell death*." DORLAND'S, *supra*, at 117.

Tr. 2 at 457.[80]

Dr. Conner stated that the basis for his conclusion that "ethyl mercury appears more lipophilic than methyl mercury" is that "since there can be higher levels of the ethyl mercury in the system and since both of the mercuries are lipophilic by nature, that you are going to get a higher level," but he admitted, "That's an extrapolation." Tr. 2 at 459. He did not mean that ethyl mercury is more lipophilic by nature, just that "[t]here is more there to be bound, therefore by just definition it's going to -- and it has a higher peak level, the same volume of methyl is not necessarily going to give the same volume of mercury as ethyl because, once again, methyl peaks earlier and plateaus off whereas ethyl continues to plateau at 24 hours and is about 20 percent higher." Tr. 2 at 460. Again, Dr. Connor's presupposition about the slower clearance of ethyl mercury does not jibe with the

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[80] He went on to elaborate further, which led to the following, rather odd conclusion:

> In the case of Thomas, the inflammation process begins with the first injection, continues on, but it doesn't necessarily mean that's the cause of this child's death. It means it's the cause that the immune system then is where you are getting the interstitial myocarditis going on that sets up the child for an abnormal cardiac response, but this information here is much more for a cell biologist and an immunologist to explain to others how it works.
>
> Q I'm not asking to explain how it works, but can you explain the relevance to thimerosal-containing vaccines causing sudden death?
>
> A The relevance is that the thimerosal, like methyl mercury, like other products in there, are capable of stimulating the immune system to produce the antibody -- I'm sorry -- the antigens which will provoke the inflammatory response.
>
> Q And what does apoptosis and necrosis have to do with that?
>
> A Because that's where you have early cell death, and that's part of the process that one will find in an inflammatory reaction.
>
> Q So the apoptosis and the necrosis are occurring as part of the inflammatory process?
>
> A Yes.
>
> Q That, in your opinion, was stimulated by the ethyl mercury?
>
> A Yes.
>
> Q If apoptosis and necrosis occurred sequentially as you say that happens in methyl mercury exposure, how does that affect the inflammatory process?
>
> A It is part of the inflammatory process.
>
> Q So whether it's sequential or simultaneous, it doesn't matter.
>
> A I don't understand what you mean by simultaneous.
>
> Q Well, you say apoptosis and necrosis occurs simultaneously in ethyl mercury versus sequentially in methyl mercury.
>
> A Yeah, and they could be the same. There are similarities.
>
> Q Whether it's sequential or simultaneous?
>
> A I don't believe so.

Tr. 2 at 457-59.

medical literature discussed in this regard, Burbacher in particular. Moreover, the point is moot because Dr. Connor refused to support his opinion, choosing instead to dismiss the issue as beyond his bailiwick:

> Q Assuming that ethyl mercury is more lipophilic than methyl mercury, how does the lipophilicity of ethyl mercury make it more toxic than methyl mercury?
>
> A I think I have to leave that to a toxicologist.

Tr. 2 at 460-61. Dr. Connor's defense of other statements in his expert report was similarly unhelpful:

> Q You say that mercury can also affect the sympathetic and parasympathetic systems. What type of mercury are you talking about?
>
> A Can be methyl, can be ethyl.
>
> Q And at what dose?
>
> A I don't know.
>
> ...
>
> Q Can the amount of ethyl mercury in a thimerosal-containing vaccine affect the sympathetic and parasympathetic systems?
>
> A I would believe so but I am not really at this particular point ready to cite any literature to that.

Tr. 2 at 461-62.

During the next phase of Dr. Connor's cross-examination, Respondent challenged Dr. Connor's interpretation of the mass poisoning studies. Respondent questioned Dr. Connor about his statements regarding the ethyl mercury pesticide which coated seed grain consumed by the subjects in the Dahhan study (EMPTS), and the cardiological aspects of the study:

> Q Is EMPTS the same as thimerosal?
>
> A Ethyl mercury is, according to Dr. Lucier, the same regardless.
>
> Q Regardless of what?
>
> A Because basically it's going to break down. The product is still going to break down to ethyl mercury, and other side products.
>
> Q So for the purpose of this discussion, the ethyl mercury, the EMPTS in that paper is the same as ethyl mercury in vaccines?
>
> A Yes.
>
> Q Does it have the same toxicokinetics?
>
> A And to that I'm not a toxicologist.
>
> Q How were the people selected as part of that study? Do you know?
>
> A Good question. The answer is no, I don't.

Q Weren't all of the people involved in that study already exhibiting signs of mercury toxicity?

A Yes, ma'am, they were.

Q So potentially that was one criteria for participation in this?

A I would assume so.

Q Okay. Do you know what the dose of the EMPTS was that caused the poisoning in that case?

A No.

Q Do you know how much they were exposed to?

A Once again, no.

Q Were there any clinical signs of cardiotoxicity?

A I don't believe that the -- the people themselves had some clinical signs here that were on Table 1, which talk about sore mouth, metallic tastes, blue lines, vomiting, diarrhea, fever, insomnia, tremors, dysarthria, ataxia, hyperreflexion, hyporreflexion, atrophy. They give a ratio of how many of these signs per the 42.

Q Were there clinical signs of cardiotoxicity?

A The cardiotoxicity on a clinical basis was not described. Cardiotoxicity on an EKG basis was described.

...

Q Is the exposure in this paper comparable to the doses of ethyl mercury in thimerosal-containing vaccines?

A I wouldn't know that.

Q Is it scientifically valid to compare the doses of EMPTS in this paper to ethyl mercury in vaccines?

A I wouldn't know that.

...

Q Will you agree that EMPTS is different from ethyl mercury?

A No.

Tr. 2 at 463-66.

The next article discussed was the Cinca article, the details of which eluded Dr. Connor's recollection:

Q Do you know the dose of ethyl mercury chloride that caused the poisoning in that case?

A I wouldn't know that.

Q Do you know how long after exposure to this contaminated meat that they showed first symptoms of mercury toxicity?

A I wouldn't know that.

Q Do you know were there any signs of cardiotoxicity?

A Apparently the cardiotoxicity was based on EKG. At this point I don't know that there were any clinical cardiotoxic signs since the majority of the problem appeared to be neurologic.

Q Are there any reports of sudden death as a result of this exposure?

A Sudden death.... There was reports of cardiac arrest in at least two of the cases.

Q Doctor, in this paper it indicates that the patients who were studied, it was a mother and three children referred to the clinic by a hospital where they had already treated for 17 days.

A I believe that was stated, yes.

Q Okay. So it had been at least 17 days, you would agree, from the time of exposure to the time that these people present to the hospital?

A They were presenting as a neurologic problem. I have no idea how long the cardiac problem was going on.

Q Do you know the mercury blood levels in the people who were studied in this paper?

A I don't.

Tr. 2 at 467-68.

Comparing the Cinca paper to the case at bar, Dr. Connor could not render an opinion comparing the blood levels in the Cinca paper's subjects with those contained in a thimerosal-containing vaccine, but agreed that among the fatal cases reported by Cinca, "there was evidence of demyelination of the cranial nerves and myocarditis." Tr. 2 at 469. When asked if pattern was repeated in Thomas Kolakowski's autopsy data, Dr. Connor pointed to the brain, heart, and liver enlargement discussed in Thomas Kolakowski's autopsy report. *Id*. To Dr. Connor, this implied that death was vaccine-related, in the absence of other known factors:

> [T]here was a significant abnormality that was developing in this child, and clearly out of the examination that was done they basically looked at the whole body, found nothing to explain any type of disease process. There was no congenital abnormalities. There was a study done on the genetics chromosomes,[81] and that was basically, I believe, negative. They did gross specimen or gross anatomy of the case and found nothing particularly abnormal in the child. There was no congenital

[81] No records of genetic analysis performed on Thomas' DNA were filed.

> diseases that they could deal with. So what they were left with are organ enlargement, brain, heart, liver, in which was basically a normal birth, normal Apgar, normal child. They even brought in people, forensic pathologists in. They couldn't find anything wrong. The only thing that they could find at that time that brought up a question was the injection to Hep. B vaccines within about 32 days, 33 days of each other, and thought that needed further evaluation, further consideration.

Tr. 2 at 469-70. However, Dr. Connor did not know, and could not opine, whether "there was any finding of interstitial myocarditis in Thomas Kolakowski," and he deferred to the pathologists on that point. Tr. 2 at 470. When asked if heart enlargement was the same, or different than myocarditis, Dr. Connor again noted that he would "have to defer to the pathologists." Tr. 2 at 470-71. In contrast to his far-ranging opinion given in his expert report, Dr. Connor's reticence on this last point seemed singular, since the differences distinguishing heart enlargement observed postmortem and myocarditis would seem like something a cardiologist could explain.

Lastly, Respondent's cross-examination of Dr. Connor focused on the specific facts and circumstances of Thomas Kolakowski's course. In a nutshell, Dr. Connor seemed ready to offer an opinion on "did it," even without proffering a theoretical mechanism on the "can it" question:

> Q Can you tell me what happens when thimerosal-containing vaccines are injected into the body?
>
> A ...Basically, vaccines are basically injected into the body with the Hepatitis or with the antigen. It is basically bound to the aluminum hydroxide. Thimerosal basically is an agent that is supposed to be as a preservative, but it has the ability of acting as an activator to help distribute the vaccine into the tissues.
>
> Q Thimerosal helps to distribute the vaccine into the tissue?
>
> A It is supposed to act as a catalyst.
>
> Q What happens when thimerosal enters the body?
>
> A I think you have to talk to an immunologist. You're getting beyond my scope. I'm a cardiologist.

Tr. 2 at 472-73.

Dr. Connor was less cautious about rendering an opinion outside of his expertise when concluding that the vaccination was to blame for the death of Thomas Kolakowski:

> When you start to discuss abnormal startle responses, you're then possibly dealing with either a peripheral injury to the nervous system, to the nerves or to the extremities, or you're dealing with a central problem, which would be more in the brain, brain stem. At that point that would be a second warning flag. The fact that the child died in 108 hours of the second vaccination, it was already too late to do anything because the warning signs were not recognized, and I would say those would be the thing I looked at and said, this is where I believe this child is different than other children. You can still have children that die, even if they've had vaccinations, but these are a couple of the signs or subtle signs that I have to look at

with suspicion to explain why this child would be different from another child who might get a vaccination. This child is different because basically what should have gone away didn't. Now I'm trying to figure out was there any place along the way that things changed to make this child different than another child, and those were the parts that -- those were the particular two items that caught my attention.

Tr. 2 at 475. In fact, Dr. Connor believed his discussion of the tremors was correct, even when it was incongruent with the opinion of Petitioners' retained (but non-testifying) pediatric neurologist:

Q [Y]ou're relying on the mother's affidavit for the evidence of any tremors in this child.

A Well, I have to rely on the mother's affidavit because she is basically a person who is involved with the care of the child on a day-to-day basis. I have to rely on the fact that many of these particular phone calls were made around Christmas and New Years, and several of the calls were made on Saturdays, that the pediatricians themselves would have not been in their office. There was no way they would have put the notes into the chart. What I found in addition to that is there is a grandmother or a mother-in-law who is also an interested party who says that she basically had seen something that didn't look right to her, and the mother followed up on it. The fact that I don't see the information in a chart, I would have preferred to, but I thought the notes were very limited.

...

Q Okay. And then I tried to keep track as you were testifying, it seemed to me there were at least four times in which you noted the mother approached the doctor or called the doctor about what she considered to be tremors.

A Correct.

Q Yet did it strike you at all that none of that information made it into the medical records?

A Not when it was done on a Saturday night in one case, and on New Year's Eve on another case, no.

Q What about an office visit?

A I don't know. I can't explain that.

...

Q Doctor, is it your opinion that the tremors that you're relying on in the mother's affidavit are a sign of mercury toxicity?

A I'm saying that it is unusual to have tremors after a few weeks. Now I'm looking back 10 years later as Monday morning quarterback, and saying they have the likelihood that it could have been mercury toxicity or thimerosal toxicity.

> Q If you relied on the medical records alone, without the mother's affidavit, is there any evidence that there was unusual signs that you say could have been symptoms of thimerosal toxicity?
>
> A Yes, the autopsy is going to have to be where I have to go.
>
> Q So the medical records don't provide it in terms of his pediatric care records?
>
> A No.
>
> Q Doctor, you would agree that you are not qualified to diagnose mercury toxicity based on the description in the mother's affidavit?
>
> A No.
>
> ...
>
> Q Petitioners asked Dr. Kinsbourne to review this case, particularly with an eye toward trying to further elucidate what mother was talking about in her affidavit of tremors.... So a pediatric neurologist looked at the record. He looked at the affidavit. He presumably had all the medical records in the case, and he couldn't arrive at a conclusion what those movements that mom described were.
>
> A But he's, once again, looking at it from a neurologist's point of view without looking at the autopsy and the pathologist's point of view.

Tr. 2 at 475-79.

Dr. Connor stated that he did not see any evidence of cardiotoxicity in any of Thomas Kolakowski's records. Tr. 2 at 480. But to him, that would not preclude an conclusion of cardiotoxicity in Thomas:

> What we have to rely on is what possibly could happen to explain the enlargement of the heart, and the possibility that this child had an event whereby there could be a setup for a significant cardiac arrhythmia that could come into this child and cause a cardiac arrest.

Tr. 2 at 480. Dr. Connor stipulated that this was the first case in which he had "attributed sudden cardiac arrest to mercury toxicity," and that he had never "diagnosed anyone with cardiotoxicity as a result of exposure to mercury." Tr. 2 at 480-81. Dr. Connor could not say what would be "the amount of ethyl mercury exposure required to cause mercury toxicity to the heart," because "that would be in the realm of the toxicologist." Tr. 2 at 481.

Continuing on in discussing the specific facts of Thomas Kolakowski, Dr. Connor was questioned on his opinion regarding the lividity noted in the medical records. To Dr. Connor, the other symptoms noted (asystole, apnea) would fit a classic cardiac arrest paradigm, "but the lividity would be a little more of an issue." Tr. 2 at 481. He described lividity as "blood pooling in a very particular area falling to gravity," and agreed that its presence indicates that "blood is not circulating normally in the body," which is caused by "[d]eath, but over a period of time." Tr. 2 at 481-82. He

clarified his opinion, stated on direct examination,[82] saying that, at the time of the EMTs' arrival, Thomas was in cardiac arrest, but was not already dead, although he could not say with any particularity when Thomas actually died. Tr. 2 at 482-83.

Commenting on the EKG strips and EMT notes, contained within the EMT records, Dr. Connor summarized that, "They called it asystolic. It could have been a fibrillation." Tr. 2 at 484. Also, according to Dr. Connor, when further EKG readings were taken, "it looked like there was some rhythm activity ... it was chaotic and it was still an abnormal heartbeat ... [it c]ould have been a slower form of ventricular tachycardia." Tr. 2 at 485. Dr. Connor agreed that the observation of Thomas by the EMTs at 7:32 that morning was that "he had no pulse and no respirations," and that they recorded his EKG as "asystolic." Tr. 2 at 485-86. Likewise, he agreed that their additional EKG notes record Thomas as being asystolic at 7:40, 7:43, and 7:46 as well. Tr. 2 at 486. Dr. Connor did not see in the medical records any indication that medical personnel tried to shock Thomas' heart, because "[y]ou need to have a course fibrillation in order to make a shock effective, and there wasn't enough here to show a course fibrillation." Tr. 2 at 487. Dr. Connor thought Thomas' heart rate of 21 could be "consistent with an agonal rhythm," because "you can still have electrical activity without actually having functional activity." Tr. 2 at 489.

Respondent asked Dr. Connor to label the identifiable wave segments in Thomas' EKG strips as taken by the EMTs and at the hospital. Tr. 2 at 490-92. Interestingly, though, after describing one such curve, Dr. Connor added:

> But once again, it has no value. It has no value whatsoever.... Because we're dealing with a dying heart. It does not tell me anything about what happened that set the thing in motion. I have no information there. Agonal rhythms basically are agonal rhythms. Everybody will have a prolonged QT in that, even if it was from a heart attack. It does not necessarily tell me anything about a setup for a dysrhythmia. I can't extrapolate backwards, and if you're expecting me to, that would be an error. I'm just making a comment that you have an agonal rhythm. It's slow, and basically what I can see there is small complexes, prolonged QT, but it does not tell me anything about the preceding event....
>
> ...[Regarding an earlier statement,] I made an observation. I'm not stating that that was the cause of it because it being agonal rhythm I wouldn't state it was a cause. If it was, it was unintended.
>
> Q So your observation that on arrival his EKG showed an agonal rhythm with a heart rate at 21, very small QRS complex, prolonged QT interval and inverted T-waves has nothing to do with the cause of --
>
> A Not for the precipitating event. This is after the event is over with, because that would get me into a problem of saying that that would be a valid reason for saying that that was the cause of it. I'm looking at the after effect. I'm not necessarily looking at what set it off. I have no documentation of that.

[82] "[T]he child may have died just shortly before mother awoke, or may have had a cardiac arrest shortly before mother awoke." Tr. 2 at 367.

Tr. 2 at 492-93.

Dr. Connor's dismissal of the EKG strips from the morning of Thomas' death extended to any comparison with the EKGs measured in the Dahhan article:

> Q Is it reasonable to compare the EKGs in the Dahhan article to the EKGs in Thomas Kolakowski?
>
> A There were no EKGs in Thomas Kolakowski.
>
> Q Based on our earlier discussion, Doctor, you would not take these EKGs that we have on Thomas Kolakowski and say that any of them demonstrate a prolonged QT interval?
>
> A Of any value?
>
> Q Of any value.
>
> A No. This is a dying heart. Even if it's prolonged, it doesn't prove anything. It cannot be applied.
>
> Q So the authors of the Dahhan article state that everyone who died had prolonged QT intervals, we can't relate that in any way to Thomas Kolakowski?
>
> A They were in a pre-arrest -- they were taken at a pre-arrest state. In the post-arrest state where you're dealing with residual electrical activity from the cardiac muscle where you may have electrical/ mechanical dissociation.

Tr. 2 at 495-96.[83]

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[83] On this same point, Dr. Connor explained in further detail:

> In the case where you have a heartbeat which is registered on the electrocardiogram, you have to have two items. One is the electrical activity where you have the PQRST seen, as you do in this end stage heart here, but you then must have the muscle functioning in such a way as to contract and push the blood from the right heart to the lungs, from the left heart to the aorta. So you have both electrical and mechanical action.
>
> Once the child, the person has died or has been declared dead it's because they can no longer determine that there is any mechanical action on the periphery from this person, yet the heart muscle will give you the impression that electrical activity still exists, but we now have electrical/mechanical dissociation, which is what you see in a dead heart, and this is what you're looking at here in the agonal rhythm.
>
> You're looking at the dying heart with electrical activity in the muscle parts that are still alive, but they are not capable of producing a mechanical effect of moving the blood from the right heart to the lungs, from the left heart out. This person has passed the point of no return. So you really are looking at electrical/mechanical dissociation right here, and the fact that you still see some electrical activity doesn't mean that the person is alive nor does it mean that the person can be brought back, but that the person died within a certain timeframe, and you asked me that before, and I can't give that to you, but by the same token this is not a person that's been dead for several hours. That's why I have a little problem with the lividity on the statement. I don't know that electrical/mechanical dissociation could go on to the point where lividity would have been seen. I know pathologists can answer that better than me, but generally speaking I don't know what they saw or what they were interpreting.

Lastly, Respondent questioned Dr. Connor regarding the potential for alternative causes of death. Tr. 2 at 498-99. Dr. Connor did not believe that a prolonged QT interval was "a specific finding" for mercury toxicity. Tr. 2 at 498. He was unsure whether a prolonged QT interval was a typical finding in asphyxiation cases, because he does not work with asphyxiation, and suggested that the pathologists in this case would be better suited to opine in that regard. Tr. 2 at 498. He did say that all other potential causes of death, besides the Hepatitis B vaccine, had been ruled out, in his mind, and that the only possible cause, or "triggering event" leading to Thomas Kolakowski's death was his thimerosal-containing Hepatitis B vaccinations. Tr. 2 at 498.

3. John J. Shane, M.D.

Dr. Shane went to Lehigh University and Hahneman University College of Medicine before engaging in a rotating internship at Wilkes Barre General Hospital, followed by a four-year residency in Clinical and Anatomical Pathology at Hahneman in Philadelphia, during which, he studied forensic pathology in Philadelphia's Medical Examiner's Office. Transcript of Proceedings convened on 11 June 2008 (Tr. 3) at 588. Thereafter, he practiced at St. Agnes Hospital as Chief of Pathology and Director of Laboratory Medicine ("responsible for the entire clinical laboratory and the entire Department of Anatomic Pathology") and taught at Hahneman for eight years. Tr. 3 at 59. Eventually, in 1974, Dr. Shane became Chairman of Lehigh Valley Hospital's Department of Pathology, which became "the largest acute care hospital in the state of Pennsylvania," a position he held for the following 28 years. Tr. 3 at 589. Thereat, the pathologists specialized their practice based upon physiologic system; Dr. Shane chose to specialize in neuropathology and cardiovascular pathology. Tr. 3 at 590. He continued to teach at various universities, and oversaw a fellowship at his hospital, until the year 2000. Tr. 3 at 590-91. At that time, he also went into his own private practice, where he remains, performing 60 to 80 autopsies per year, as well as consulting on forensic pathology issues[84] with other pathologists. Tr. 3 at 591-92. His practice has never limited to any particular age bracket, and he has performed autopsies on children and adults of all ages. Tr. 3 at 592.

Dr. Shane's direct examination turned to focus more particularly on his experience over the years with the issues presented by the case(s) at bar. Regarding whether Dr. Shane's teaching experience involved discussing mercury toxicity, Dr. Shane said that it formed part of his discussion on liver disease as a toxic threat to the liver. Tr. 3 at 595. Regarding whether he had ever

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Tr. 2 at 496-97.

[84] Dr. Shane explained this aspect of his practice thusly:

> Forensic pathology has to do with abnormal death. Death due to any unnatural cause. Many of these cases, the bulk of the cases that we did were impact, they were motor vehicular. And there were all ages that came through because all ages of people die in motor vehicular accidents. We also did the standard run of suicides, homicides, and asphyxial death is a coroner's case, as this case, actually died in one of the Pinnacle hospitals but was referred to the local coroner because it was an unexplained death. Unexplained deaths come to the coroners. This case fell into the unexplained category so it was sent to the forensic people.

Tr. 3 at 593-94.

encountered a case of mercury toxicity as a pathologist, Dr. Shane regaled that he could recall two, the most recent of which was a slow, steady poisoning by a wife of her husband with mercuric salts (inorganic mercury). Tr. 3 at 597. Dr. Shane expressed that he was familiar with the effects of mercury exposure on the nervous and cardiovascular systems. *Id*.

Moving on to the facts of this particular case, Dr. Shane focused, much as Dr. Connor had, on the relative good health of Thomas from his full-term birth until his death, and Mrs. Kolakowski's reference to "tremors" in her affidavit. Tr. 3 at 598. He described symptoms demonstrated by Thomas following his second Hepatitis B vaccination, such as "GI gas, decreased appetite, some respiratory difficulties, and a skin rash." *Id*. Dr. Shane referred to Thomas' EKG activity as "pulseless electrical activity" in that "he didn't have a pulse, but he had cardiac electrical activity." Tr. 3 at 599. Dr. Shane found it "very important" that Thomas was "warm to the touch when the EMTs arrived ... because babies, particularly in December going outdoors, even in a 70 degree room, babies have such a surface area that they lose heat very rapidly." *Id*. The recorded body temperature of 97°F meant, to Dr. Shane, that this was "a very recent death from the time he was examined." *Id*.

Later during his direct examination, Dr. Shane was asked whether the description in the EMT records of "cardiac arrest" was consistent with his analysis, and he explained that this was only partially correct:

> If we're to take cardiac arrest as pulseless, this child was pulseless. If we're to take cardiac arrest as there is no myocardial activity, no. This child had myocardial activity. This child had electrical activity on the EKGs from the EMT. Certainly had electrical activity on the EKGs in the emergency facility.

Tr. 3 at 627-28.

Dr. Shane next brought his pathology experience to bear on the pathology slides made pursuant to Thomas' autopsy. Tr. 3 at 600. Dr. Shane related that in his first examination of those slides, his attention was drawn wholly to the encephalic data, but that his second look at the slides focused on the "very subtle" changes he observed in the cardiac slides. Tr. 3 at 601. He concluded that both systems manifested adverse effects "directly related to the mercury exposure." *Id*. Dr. Shane heaped praise on the thoroughness of the examining pathologist and the slide preparation performed by the appurtenant laboratory. Tr. 3 at 602. The first finding to which Dr. Shane directed his focus was gliosis, followed by "dropout" of the granular cells of the cerebellum:

I saw the gliosis.[85] The gliosis spanned the glial cell population which include astrocytes, microglia and oligodendroglia.[86] When I use the term gliosis I mean proliferation as well as activation. A dormant glial cell has a particular appearance microscopically when it becomes reactive and it's now doing what it's intended to do. It gets a different appearance -- there is some enlargement of the cell, there are some nuclear changes, and I use the term "turned on" is the term I use with fellows and residents that I teach. This is a "turned on" glial cell which means it's a reactive glial cell. So we had both. We had the proliferation, we had the change in appearance of the glial cells.

The cerebellar findings were interesting in that we had drop-out of the granular cells. The granular cells occupy a very narrow area in the cerebellum. So if you have enough drop-out it can actually appear that there's a separation occurring there. It was my interpretation that this separation was due to the granular cell drop-out. There was a decrease in the number of granular cells, and there were also Purkinje cell[87] changes. The Purkinje cell changes were, again, changes of degeneration. These were the findings that I reported in addition to the cerebral edema.

This child had some perivascular, periglial, perineuronal clear spaces. These clear spaces were more prominent than one would see with normal tissue shrinkage. I refer to them as halos. There were halos around these cells, but they're really perivascular, periglial, perineuronal clear spaces that are associated with edema. In

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[85] Dr. Shane contextualized gliosis by introducing the nature of the glial cells:

> Glial cells are the inflammatory reactive cells of the brain. Yes, you can get migration of other inflammatory cells from the peripheral blood into the brain, but locally in the brain the brain has its own population of inflammatory reactive components. The astroglia, they respond quickly to a local process. And if you're to make an analogy, the analogy would be to the neutrophils which are the acute phase reactants elsewhere. The more chronic reactants, sub-acute chronic reactants, is the microglia. They're like the lymphocytes and they come in a little bit later with their changes. I view the oligodendroglia as the fibroblasts of the brain. They come in and they can make scar tissue in an area of injury. They're a more chronic reactant. I use the analogy in my teaching to a fibroblast. All three types of glial cells in this case were reactive, they were turned on, and they were proliferated. There was an increased number of them.

Tr. 3 at 604.

[86] Oligodendroglia are "the non-neural cells of ectodermal origin forming part of the adventitial structure (neuroglia) of the central nervous system; projections of the surface membrane of each of these cells (oligodendrocytes) fan out and coil around the axon of many neurons to form myelin sheaths in the white matter. With microglia, they form the perineuronal satellites in the gray matter." DORLAND'S, *supra*, at 1305.

[87] Purkinje cells come in two forms: "1. large neurons in the cerebellar cortex that have piriform cell bodies in th Purkinje layer ... and large branching dendrite trees going through the outer (molecular) layer towards the surface. 2. cells of the Purkinje fibers of the heart; they are large, clear, tightly packed cells with many gap junctions between them and thus conduct impulses rapidly." DORLAND'S, *supra*, at 325.

> a few sections there was a little bit of a extravasation of erythrocytes.[88] That's also part of the cerebral edema process.

Tr. 3 at 602-03.

When Dr. Shane stated that he observed edema in the brain, the Court interjected to ask whether he viewed that edema as vasogenic[89] or cytotoxic,[90] to which he replied: "There was some of each. There was some vasogenic edema and there was some cytotoxic edema, so it was one of those places where we had an overlap of edema." Tr. 3 at 605. Dr. Shane stated the significance of this conclusion:

> The significance is that this is a process where there is within the brain cytoinflammation. A cytoinflammatory process that is making these cells reactive. And as part of that reactivity there are changes in the blood vessel permeability, allowing fluid to escape into the perivascular and periglial spaces. And in addition, as part of that cytoinflammatory process, we have what's called spongiosis[91] or some intervening edema within the cerebral white matter.

Tr. 3 at 605-06.

Dr. Shane stated that, much like the presence of white blood cells in the bloodstream, the presence of glial cells indicates an inflammatory response in the brain. Tr. 3 at 606. He went on to say, though, that this response of gliosis is non-specific, which means it does not implicate a specific stimulus as causative:

> [T]he brain kind of is a unique organ because it reacts to a whole number of things by doing the same thing. You get cerebral edema, albeit of differing types with toxic involvement, you get it with injury, you get it with hypoxia,[92] you get it with tumors, you get it with infarcts or strokes. The brain gets edematous almost with any stimulus. So you have this broad spectrum of things that causes cerebral edema.

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[88] An erythrocyte is "one of the elements found in peripheral blood ... a non-nucleated, yellowish, biconcave disk, adapted by virtue of its configuration and its hemoglobin content to the transport of oxygen." DORLAND'S, *supra*, at 640.

[89] Vasogenic edema is "a type of cerebral edema seen in the area around tumors, largely confined to the white matter; it often results from increased permeability of capillary endothelial cells and less often is due to toxic injury to the vessels." DORLAND'S, *supra*, at 590.

[90] Cytotoxic edema is "cerebral edema caused by hypoxic injury to brain tissue and decreased functioning of the sodium pump, so that the cellular elements take in fluid and swell." DORLAND'S, *supra*, at 589.

[91] Spongiosis is "intercellular edema of the spongy layer (malpighian layer) of the skin." DORLAND'S, *supra*, at 589.

[92] Hypoxia is the "reduction of oxygen supply to tissue below physiological levels despite adequate perfusion of the tissue by blood." DORLAND'S, *supra*, at 890.

> When we deal with neurolysis,[93] it happens under many, many conditions. There's a lot of overlap. When you see something in the brain and you ask yourself the question what caused this, there's always some degree of overlap with what caused it. It's when you put together the complex of findings that you begin to narrow down the causes and you can develop some timing. So you have findings that are overlapping, but the more findings you put in there, the more specific you can become about what caused it, so it's a matter of looking at the findings collectively rather than individually, number one. And the more individual findings that you have the more specific you can become about interpreting the causation.

Tr. 3 at 606-07. In the instant case, Dr. Shane said he viewed the conjunction of both "turned on" oligodendroglia and fibroblasts[94] meant that Thomas' injury was "sub-recent rather than very recent," adding, "Oligodendroglia don't get turned on immediately, they get turned on a little bit later." Tr. 3 at 607.

Moving on to the more "subtle" cardiovascular findings he made from the tissue slides, Dr. Shane stated:

> Those findings in the heart were, number one, diffused interstitial edema in all the sections, and in particular in the sections of the septum, the interventricular septum. I had an inflammatory cell infiltrate, and it was a mixed inflammatory cell infiltrate. There was some acute, some sub-acute, some chronic elements. Not a big time infiltrate, but it was there. There were inflammatory cells, albeit you had to look for them. But when you looked for them, they were there....
>
> And in the septum, the septum is unique because it is going to be very, very susceptible, as it is in adults, to vascular changes. And that's where I saw some early myocardial muscle cell shrinkage. Again, a subtle finding, but on studying the sections of heart I am convinced that there was myocardial cell shrinkage and some earlier myocardial cell degeneration occurring in that septum. I had to be persuaded of those findings before I would call them. I was persuaded, I did call them as such. It ties in with the findings of congestive heart failure[95] in this child, and those findings were the liver congestion, the spleen congestion, and of course the pulmonary congestion which was very, very marked.

Tr. 3 at 611. When asked what the significance was of "early myocardial cell shrinkage and degeneration," Dr. Shane elaborated:

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[93] Neurolysis is "1. release of a nerve sheath by cutting it longitudinally; 2. the operative breaking up of perineural adhesions; 3. the relief of tension upon a nerve obtained by stretching; 4. destruction or dissolution of nerve tissue." DORLAND'S, *supra*, at 1255-56.

[94] A fibroblast is "a connective tissue cell; a flat elongated cell with cytoplasmic processes at each end, having a flat, oval, vesicular nucleus. Fibroblasts ... form the fibrous tissues of the body." DORLAND'S, *supra*, at 695.

[95] Congestive heart failure is "a clinical syndrome due to heart disease, characterized by breathlessness and abnormal sodium and water retention, often resulting in edema. The congestion may occur in the lungs or peripheral circulation or both, depending on whether the heart failure is right-sided or general." DORLAND'S, *supra*, at 672.

> The significance of that is that the blood supply to that septum is the anterior descending branch of the left coronary artery, and there really isn't a lot of overlap. That's it for blood supply. That's why anterior descending occlusions in older people are called widow makers. Don't have a lot of overlap in blood supply. With the edema that was developing, that edema would reduce the amount of blood supply to the cellular components of that septum, and that resulted in the early, again, early, beginning, subtle changes in those myocardial muscle cells.

Tr. 3 at 612-13. He added that his assessment of diffuse interstitial edema is "a type of edema that you see in an interstitial myocarditis." Tr. 3 at 613. Continuing on, he went into further detail on his notation of "interventricular changes" to the cardiac septum:

> Interventricular septal changes which are significant because those changes will produce arrhythmias. The interventricular septum houses what's called the Purkinje system. The heart has its own nervous system. It's called the Purkinje system. Purkinje fibers are modified myocardial muscle cells that are modified to conduct neurological signals. The SA node, the AV node communicates via the Purkinje system. The Purkinje system, the conduction system of the heart is housed exclusively in the interventricular septum. All impulses emanate out over the wall of the left and right ventricles from that Purkinje system and the septum, so I felt those subtle changes would be very consistent with disarrhythmias.

Tr. 3 at 613-14. When asked if these changes might affect the heart's conduction system, Dr. Shane responded:

> Yes, that is the conduction system. It is housed exclusively in the IV septum. It's single vessel blood supply is going to certainly have, even small amounts of edema are going to begin to impact on those vessels emanating from that LAD and cause these muscle fiber changes.

Tr. 3 at 614.

Dr. Shane believed that his analysis of the pathological findings dovetailed with the clinical record:

> This child in that hiatus time between receiving the second injection and dying and the autopsy did have respiratory involvement. There were respiratory symptoms. This child did have loss of appetite. Loss of appetite is the preeminent finding[] in liver congestion. Patients with congestive heart failure, what do they complain of when they come in? Doctor, I have no appetite. It's because of that congestion of the liver. So this child had decreased appetite. This child had the respiratory findings before death. Now we have this prominent congestion. In addition, this child was complaining of gas, and I'm pretty sure the gas would correlate to this child having some visceral passive congestion as well. So there were symptoms that tied in with these findings in these organs that are part of the medical record that are explained by the pathology findings and they correlate with the pathology findings.

Tr. 3 at 612.

Dr. Shane also made much of the rather nonspecific pathological finding of increased weight of certain organs:

> There was increased organ weights. And the increase organ weights involved the brain, very prominently. The brain weight was increased, the liver weight was increased, the spleen was increased, the kidneys were increased somewhat. A heart had an increased weight....
>
> I think the slight increase in weight of the heart was due to the edema, but the heart is small, hence the amount of edema fluid is going to be a matter of a few grams, that's all. The brain is much bigger. The capacity of the edema fluid or to accommodate edema fluid is much greater. So they're going to have this very dramatic increase of about 100 grams. The kidneys, again, can have some increased weight due to congestion, due to edema. And there we have a very significant 20 percent increase about in the weight of the kidneys. The spleen was increased very substantially. The liver was increased in weight. This is due to congestive changes....
>
> The combined weight of the lungs was 180 grams. Normal is 68 grams. So this is very, very dramatic. The only thing that gives you that kind of weight increase is pulmonary edema. There was severe congestion here, some escaped fluid is going to be obvious. That fluid, again, might be extracted during the processing, but nonetheless it was there because that kind of weight increase is congestion and edema which is the pulmonary passive congestive changes that I described.

Tr. 3 at 615-16. The Court queried Dr. Shane on whether the increased organ weights was caused by whatever also caused Thomas' death, or if it was just "part and parcel to the agonal process," to which Dr. Shane responded that the difference was one of magnitude and degree:

> That is far more than you could possibly accumulate in the short timeframe of the agonal process. You're not going to get that much in that short a time. The cerebral edema, there's no way with the agonal process that you are going to collect 100 grams of edema fluid in this baby brain during the agonal process. So this was something that was going on before the agonal process.

Tr. 3 at 616-17.

Dr. Shane agreed with the statement made by Petitioners, that pulmonary edema is "also known as congestive heart failure," a patently false proposition,[96] and in an attempt to elaborate, said, "if you're not putting that blood out of the left ventricle you can't put the blood in from the lungs and

[96] Pulmonary edema is "abnormal, diffuse, extravascular accumulation of fluid in the pulmonary tissues and air spaces due to changes in hydrostatic forces in the capillaries or to increased capillary permeability; it is characterized clinically by intense dyspnea and, in the intra-alveolar form, by voluminous expectoration of frothy pink serous fluid and, if severe, by cyanosis." DORLAND'S, *supra*, at 589. Congestive heart failure is "a clinical syndrome due to heart disease, characterized by breathlessness and abnormal sodium and water retention, often resulting in edema. The congestion may occur in the lungs or peripheral circulation or both, depending on whether the heart failure is right-sided or general." DORLAND'S, *supra*, at 672. The two share attributes, and one may be a (nonspecific) symptom of the other, but to say that they are equivalent is quite incorrect.

they congest." Tr. 3 at 617. Also, Dr. Shane believed, unsurprisingly, that the edema in the myocardium was related to the edema in the lungs, liver, spleen, kidneys, and viscera, all "part and parcel of right and left heart failure." *Id.*

When asked if Thomas' death was "quick" or "drawn out," Dr. Shane replied, "drawn out," but then proceeded to cite as his evidence for this answer nothing about his analysis of the agonal process at all, and to focus entirely on clinical features that (ostensibly) preceded the night of Thomas' death by days and weeks. Tr. 3 at 619-20. In doing so, Dr. Shane displayed the professional, analytical and explanatory skill of a medieval barber.[97]

The next point addressed by Dr. Shane was that infants' brains are qualitatively different from adults' brains in some ways, and in some ways they are similar. He explained that the brain continues to mature and develop after birth in fundamental ways, and that, for a time, the blood-brain barrier is more porous and less restrictive. Tr. 3 at 618-19. In fact, he noted that, with infants, "everything goes through," as they receive nutrients and antibodies from their mother while *in utero*. *Id*. He noted that the barrier progressively closes as the child grows older. Tr. 3 at 619.

The next major phase of Dr. Shane's testimony considered some of the medical literature filed in this case. Specifically, Dr. Shane stated that "there is only one article that is very, very good on the histopathologic findings, what we see under the microscope," and that was the Cinca[98] article; due to Dr. Shane's area of professional expertise, he focused on it. Tr. 3 at 621. He saw it as relevant to this case in the first part, because it "talks about neuronal necrosis," and Dr. Shane saw neuronal necrosis in viewing Thomas Kolakowski's autopsy slides, including "nerve cells that are showing nuclear fragmentation and degeneration" and "cytoplasmic changes." Tr. 3 at 622. This was significant to Dr. Shane "because neuronal degeneration takes time to develop [and does not] happen instantly in a very sudden death." Tr. 3 at 622. He continued:

> We have the [gliosis] that they talk about. I talked about the global [gliosis]. That is a finding that they relied upon. In that article they talk specifically about the cerebellar changes, the granular cellular drop-out, which we have. We have the granular cell degenerative changes. And the Purkinje cell changes, which we also had in this case. So when we look at the cerebellar findings, which are unique to the

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[97] Specifically, he said:

> If we take this child had respiratory difficulties, if we take into consideration that this child had decreased appetite, the decreased appetite I tie into the liver. The respiratory changes I tie into some congestion of those lungs. The feeling that this child had a lot of gas, I tie that into gastrointestinal congestion. So this child was symptomatic over a period of time and just happened to die very very sub-recently to going into the emergency room. This death occurred very very close to that child being found unresponsive and that's based on the factual things. The fact that this child had some PEA, pulseless electrical activity, still remaining in that heart during transit, still remaining when the child arrived to the hospital. And this child even in a hospital was 97 degrees, the child was warm.

Tr. 3 at 619-20.

[98] Pet. Ex. 18, Tab V, I. Cinca *et al.*, *Accidental ethyl mercury poisoning with nervous system, skeletal muscle, and myocardium injury*, 43 JOURNAL OF NEUROLOGY, NEUROSURGERY, AND PSYCHIATRY 143-49 (1979).

> mercury toxicity,[99] those cerebellar findings are present in this case. The cerebellar findings are the granular cell drop-out, the Purkinje cell changes, and we had that.
>
> The critical findings are the edema, the gliosis, the neuronal drop-out, and we have that. Again, these are changes that individually can occur in a number of situations. The cortical changes, three cortical changes of neuronal necrosis, the edema, the gliosis, that could occur in a host of different conditions so long as you take into consideration the time involved for this to occur. But then when we put into the mix the cerebellar findings, the cerebellar findings begin to narrow massively the choices and the best choice of a causative factor would be the mercury, we have the mercury exposure with the thimerosal. The mercury changes are described in this literature article. We have them here. It's a fit. So overwhelmingly, more probably than not, I believe the central nervous system changes were the result of mercury toxicity.

Tr. 3 at 622-23. Dr. Shane has testified before this Court enough times to know that generalized reference to conclusory opinion, without explanatory detail of what and how, remain unpersuasive. He pointed to no specific datum from the Cinca article, and did not relate the article in any specific way to the issue at bar, except to exclaim that the two were "a fit." He in no way described how a specific cerebellar finding was more related to mercury toxicity or exposure, or how it would make mercury the best choice as causative factor. What other choices are there in the differential diagnosis? What would lead him to exclude or demote the other possibilities? Dr. Shane never explained.

Another point from the Cinca article upon which Dr. Shane seized was a mention of satellitosis,[100] which, as Dr. Shane interprets the autopsy slides, was present in one instance within Thomas Kolakowski's autopsy slides:

> [T]hat [] is a nerve cell [] undergoing degeneration and the neuroglial comes into that cell and is about to undergo transformation to assist in the removal of that cell as it is degenerated, so you get this neuroglial cell that is attached to the neuron like a little satellite. We did have one cell that I'm convinced was satellitosis.

Tr. 3 at 624. Dr. Shane never explained how his putative observation of one of those cells related in any aetiologic way to the cause of Thomas' death, or how it connected Thomas' case with the data in the Cinca article–as if the similar development of one such cell, without further explanation necessary, proved that the thimerosal in a vaccine dose caused the death of Thomas, just the same as the consumption of pork contaminated with a poisonous dose of mercury would cause death. To Dr. Shane, even cells he did not observe prove his (unexplained) theory:

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[99] If this was so, Petitioners never put on evidence that it was so, which would have covered some distance in proffering a theoretical mechanism with specific characteristics on the "can it" question, which would then have been useful to check against specific cases like this one, in answering the "did it" question. Also, for something that, if true, might work to prove Petitioners' theory, Dr. Shane never once described how "the cerebellar findings" that he found were so specific to mercury toxicity and worked to align the Cinca study with the case of Thomas Kolakowski.

[100] Satellitosis is "accumulation of neurological cells about neurons; seen whenever neurons are damaged." DORLAND'S, *supra*, at 1658.

> Well, you get satellitosis, you get these glial cells, these neural glia that are going to phagocytize these degenerated nerve cells. You get them concentrated in an area and it forms a neuronophagic[101] nodule. That's something that occurs down the pike somewhere. I didn't see neuronophagic nodules, but you don't have to. That is a later stage in the process. I didn't have it here, but I did have the satellitosis and I certainly did have the neural glial proliferation. And I did certainly have the neural degeneration.

Tr. 3 at 624-25. "Timing" was all that separated Thomas' pathological findings from the Cinca case, said Dr. Shane:

> It takes time for these neurons to undergo degeneration. It takes time for the satellitosis to occur. Then if we add more time to the system, which we didn't here, you will have the neuronophagic nodules.... Things are not going to happen exactly as they would in an older child. So I wasn't that concerned with the fact that I did not see neuronophagic nodules, but I was very reassured in my opinion that I did have the other findings, the neural degeneration, the gliosis, the microglial changes as well as other glial changes, oligodendroglia. And we did have at least one cell with some satellitosis so they're starting to come in and clean it up.... The satellitosis is the beginning of that process. He didn't, in my mind, live long enough to develop the nodules.

Tr. 3 at 625-26.

Aside from the neuronal findings, Dr. Shane also noted that he saw similarities between Thomas' case and the Cinca data as they related to "myocardial findings" as well. Tr. 3 at 626. Dr. Shane described the following indicia he perceived in Thomas Kolakowski's case as finding an analog in the Cinca article:

> [T]he interstitial edema..., a subtle finding in this infant heart. The scattered inflammatory infiltrate of a mixed nature. And the early myocytic[102] changes in that septum. And these again are findings that correlate with the findings in the article. And I think they're significant, that we do have this correlation with this, the article describes an interstitial myocarditis.... The article correlates very, very nicely and describes the findings that I saw.

Tr. 3 at 626.

Dr. Shane concluded his direct examination in chief by summarizing the conclusions of the examining pathologist(s):

> Dr. Ross, prior to arriving at a conclusion, ... did engage three other forensic pathologists, a pediatric pathologist and another neural pathologist to review this case with him. The conclusion after those reviews is that these pathologists had great

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[101] Neuronophagia is "the destruction of nerve cells by phagocytic action." DORLAND'S, *supra*, at 1257.

[102] A myocyte is a muscle cell. DORLAND'S, *supra*, at 1213.

> concern over this child receiving the Heptavax. This seemed to provide for them a link to this child's death and they recommended further study.

Tr. 3 at 628. When pressed by the Court for the basis for such a conclusion, Dr. Shane explained:

> I believe their basis was that they did not have another basis for this child's death. They had symptoms, which they report the symptoms of the gas, the decreased appetite, the respiratory findings, and then they have findings they saw at autopsy which would correlate with that, but they didn't have a cause. And I think they were again, searching for something beyond what they had and the one link that they saw that caused them concern was the Heptavax administration. This was something that was a link that they were seizing upon.

Tr. 3 at 629.

Finally, he summarized his own opinion, that "the ethyl mercury which was contained in [Thomas Kolakowski's] hepatitis B vaccine contributed to his death," saying:

> [M]y reasons for that is when I looked at the central nervous system findings, they correlate very, very, very closely with what is reported in the article by Cinca. They correlate very, very closely in what I have seen in mercury deaths, and I have examined two of those. And it also correlates with this child's cardiac involvement which caused the visceral involvement, which relates back to the symptoms between the time of that second injection and the time of death. So we can correlate the clinicals with what we have. The literature reports the myocardial findings very close to what we have seen. And it's inescapable that this child had the visceral congestion, and you're not going to get that amount of congestion in a matter of minutes or even an hour. It takes time for the pulmonary edema to occur. It takes time for the cerebral edema, to this degree, to occur. So I believe this child had an ongoing process. I believe this child died very, very sub-recently to the time that it was discovered. The baby was still warm. The baby still had PEA, pulseless electrical activity. The PEA continued into the hospital. The baby continued to maintain temperature into the hospital. I'm sure as a result of the PEA. And I think the clinical pathology goes together very, very nicely in relating the causation to the mercury containing compound.

Tr. 3 at 629-30.

At least at the outset, Respondent's cross-examination of Dr. Shane consisted largely of challenging Dr. Shane's credentials, and the accuracy of his filed *curriculum vitae*. The first line of such questioning focused on his expertise in toxicology, as Dr. Shane is board-certified in Chemistry and Toxicology by the American Board of Clinical Pathology, which he said certifies him to perform laboratory work if necessary, although he delegates lab work by contract to laboratories. Tr. 3 at 631-32. His certification, he related, was "primarily focused on the interpretation of laboratory testing including, in this case toxicology and chemistry testing." Tr. 3 at 633. Dr. Shane acknowledged that he was not certified by a Ph.D. Toxicology board, and that he had not undergone the training for the Medical Toxicology board that he understood to be part of Emergency Medicine

expertise. Tr. 3 at 632, 634. Respondent challenged his expertise in forensic pathology, and Dr. Shane explained that he has training and experience in that area, but is not board-certified in the specialty. Tr. 3 at 634. Additionally, Dr. Shane admitted that he was not board-certified in Pediatric Pathology or Neuropathology, although, he stated, he has training and extensive experience in both areas, having begun his pathology practice before board certification had emerged as a widespread or popular credentialing process. Tr. 3 at 635-36. Dr. Shane likewise conceded that he did not possess board certification or special expertise in the fields of immunology, hepatology, nephrology or cardiology, although he said he did possess special expertise through training and professional experience in cardiovascular pathology, and did have experience in immunologic test interpretation, liver pathology, and kidney biopsy. Tr. 3 at 663-664. Regarding the two cases of mercury toxicity in his experience to which he had referred on direct testimony, Dr. Shane conceded that the poisoning case he had referenced involved (inorganic) mercury salts, not (organic) ethyl mercury, and that he did not recall the details of the other case. Tr. 3 at 662-63.

Respondent's questioning revealed that Dr. Shane did not actively teach any students, even though he maintains a relationship with one medical college, and was not deriving income from any university for teaching. Tr. 3 at 637-641. His private practice retains consulting physicians when needed, but only employs Dr. Shane directly, although he does maintain an office staff presence. Tr. 3 at 642-43. Dr. Shane's work as a litigation consultant and expert witness fills twenty to twenty-five percent of his time, and composes the same approximate share of his income. Tr. 3 at 650-51. He testifies in hearings, depositions, etc. between ten and fifteen times per year. Tr. 3 at 658-59. Respondent also questioned Dr. Shane about his publications and his testimony within specific cases, but these are of *de minimis* relevance to the matter at hand, and the Court does not summarize that here. In one particular case raised by Respondent, Dr. Shane wrote conflicting, apparently contradictory opinions for opposing parties to the same lawsuit, based on differing records provided to him by each side. Tr. 3 at 713-724.

Dr. Shane noted that his opinion does not rely upon or acknowledge the expert opinions of Dr. Lucier or Dr. Connor, and that he did not communicate with either, or read their expert reports, prior to composing his own. Tr. 3 at 666. Dr. Shane did agree with Dr. Connor's hearing testimony that the EKG readings from the EMTs and the hospital were "meaningless for determining the root cause of Thomas' cardiac arrest." Tr. 3 at 667. He also stipulated that cardiac arrest is part of dying, not necessarily a cause thereof. *Id*.

Dr. Shane admitted that he was not aware of cardiac effects of mercury toxicity until he began research on the instant case(s). Tr. 3 at 667. The only medical literature of which he was aware that demonstrated ethyl mercury toxicity affected the heart was Cinca, and he was not aware of any medical literature regarding the cardiac effect (if any) of thimerosal. Tr. 3 at 667-68. Nevertheless, he thought the form and dosage of the mercury studied in the Cinca study was sufficiently analogous to apply in analyzing thimerosal-containing vaccines. Tr. 3 at 668-69. When Respondent asked Dr. Shane to compare the blood levels of mercury between a vaccinee like Thomas Kolakowski and those recorded in the subjects in the Cinca study, Dr. Shane stated that to do so was "completely irrelevant in the context of this case," because, "You can't take the blood levels from older children and apply them to blood levels in an infant." *Id*. To Dr. Shane, the differences between infants and older children or adults were so qualitatively distinct, the same dose

response curve could not be seen as useful from one context to another, because of the greater permeability of the blood-brain barrier in infants. Tr. 3 at 669.

Dr. Shane's first expert report filed in this matter did not address adverse effects in the heart, focusing instead entirely on the brain; it was only in his second report that he noticed aspects in the heart tissue which he then attributed to the thimerosal in the Hepatitis B vaccine. Tr. 3 at 671-72. Dr. Shane explained this apparent disparity by stating that his first analysis considered seven different cases, and that such review focused on the brain slides of each; it was only when this case was selected as "exemplar" that his closer look revealed the cardiac effects. Tr. 3 at 672.

When asked again later whether he thought "dose determine[s] the toxic effect of an agent," he was reluctant to agree:

> Again, you have to be very, very careful with dose and how you apply it. I'll use an example of a very common drug, acetaminophen. Acetaminophen in normal adults has a high dosage range. You need to take 30 times the dosage to exceed the capacity to create the glucuronide which is excreted. If you exceed that, the acetaminophen is changed to a drug that is liver toxic and very liver toxic....
>
> ...In infants, you can trigger liver damage in children with the normal dose for children. It's because they don't have the presence of glucuronide manufactured by the liver to detoxify and excrete acetaminophen. So here is a drug with a normal pediatric dosage that can cause liver damage. The same thing in adults. An adult with liver impairment of any sort can trigger liver cell necrosis with regular therapeutic dosages of acetaminophen. So when you talk about a dose relationship you're talking about, number one, children; you're talking about here an infant. So you again, like acetaminophen, normal or very small dosages can precipitate catastrophe. So you can't rely on, if it's not a major dose it's not going to cause any damage. That doesn't apply. It doesn't apply in children and it doesn't apply in some adults....
>
> ...There isn't a specific dose, particularly in infants, where you could say this dose is okay and this dose is toxic. You can't do that....[103]
>
> Acetaminophen in normal dosages can cause liver necrosis in children and even in adults with liver cell damage. Normal dosage can be fatal. So dosage isn't the final word, particularly when dealing with infants. And it's not a change in pharmakinetic properties, it's a change in how infants absorb the compound, how infants deal with the compound, how infants pass the compound into the brain.

Tr. 3 at 699-701. When Respondent challenged Dr. Shane's statement in his expert report that age was a greater determinant of toxicity than was dosage, Dr. Shane replied:

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[103] This statement would appear to contradict Petitioners' toxicologist, Dr. Lucier, who went into some detail describing how he and several others collaborated to arrive at a reference dose, which is an average daily dose that is putatively "safe" for almost anyone.

> [T]herapeutic dosages of some substances like commonly used substances, acetaminophen, can be toxic, can be fatal, at recommended dosages.... A little bit can cause physiologic damage. Again, it's a very, very individual thing and when you're dealing with infants it's a highly individual thing because infants are not small adults.

Tr. 3 at 702. However, even applying this rule could not explain why Thomas Kolakowski died in response to receiving the Hepatitis B vaccine. When Respondent asked Dr. Shane, "Why isn't the normal child experiencing significant granular loss whenever they get vaccinated with a thimerosal containing vaccine," considering "the blood/brain barrier is not up and running," his response was simply that, "They're different. Each child is different." Tr. 3 at 703.

This led Respondent to question Dr. Shane as to why more children were not damaged in the manner Thomas was:

> Q Similarly, wouldn't you expect if the blood/brain barrier is not up and running, that thimerosal containing vaccinations would just ravage normal children's Purkinje cells?
>
> A I can't say that. Each child is an individual. Each child will respond differently. We know what we have here, and that's what's important. In Thomas Kolakowski we have a child who has tremors of some type, we have a child who after he receives the second shot has clinical symptoms of abdominal problems, decreased appetite, respiratory problems. This child is symptomatic.... This child then goes on to a death and at autopsy this child had the findings that we're talking about. And the whole thing comes together with a very, very nice clinical pathologic correlation supported by the literature.

Tr. 3 at 704-05.

When asked, Dr. Shane did not know the amount of ethyl mercury contained in the vaccine doses administered to Thomas Kolakowski, and, furthermore, he did not think that such knowledge was relevant: "I don't think quantitative toxicologic determinations of tissue levels of ethyl mercury would change anything." Tr. 3 at 707-08. Respondent carried this defenestration of the dose relationship to a *reductio ad absurdum*:

> Q I'm asking you, Doctor, at what point would you see the signs in Thomas Kolakowski, because you say dose doesn't matter. So .1 micrograms? .001 micrograms?
>
> A It doesn't apply. You haven't heard my testimony that precise dosage levels don't apply in individuals with the toxic effects. So .1, if it's 1, whatever it is, we know we have these effects so we know at that level in this infant it was toxic.

Tr. 3 at 708.

Dr. Shane stated that he had no dispute with the conclusions made in Thomas Kolakowski's autopsy, which he summarized as diagnosing "Sudden Infant Death" (but not Sudden Infant Death Syndrome, or SIDS), and noting a concern about the Hepatitis B vaccine. Tr. 3 at 670. Dr. Shane

was very laudatory concerning the sampling of the tissues from Thomas Kolakowski's autopsy, calling it "wonderful" and "beautifully worked up." Tr. 3 at 672.

Dr. Shane also made much of the clinical symptoms experienced by Thomas Kolakowski before his death:

> [T]he clinicals are that this child was symptomatic between the time of receiving the vaccine and dying. This child had respiratory difficulties. This child had gastrointestinal problems. This child had decreased appetite. And this child had a rash.

Tr. 3 at 674. This prompted the Court to question "the significance of the rash," to which Dr. Shane replied that he thought there had been a "suggestion that this rash may have been ... pediatric acne [and that t]he significance of the rash could be a cutaneous manifestation of hypersensitivity." Tr. 3 at 674-75. When Respondent questioned what the hypersensitivity was directed at, whether at the mercury in the thimerosal or to an antigen, Dr. Shane replied, "Hypersensitivity to anything," but could not be more specific, he said, because "it's a paucity of information." Tr. 3 at 675.

Dr. Shane agreed that lividity was detected by the EMTs, which increased before Thomas was pronounced dead at the hospital. Tr. 3 at 676. Dr. Shane related this to his interpretation of the EKG strips:

> [W]ith pulseless electrical activity the circulation of blood throughout the body is very much diminished. That's why you don't have a pulse. That's why it's PEA and not a pulse.

Tr. 3 at 676.

When asked whether the finding of cardiac edema was consistent with hypoxic ischemia, Dr. Shane said it was not, based on his review of "hundreds and hundreds and hundreds of hypoxic hearts." Tr. 3 at 676. Instead, he views edema to be more common in cases of inflammation, based on his experience with hundreds of cases of myocarditis of various aetiologies," leading him to conclude that edema would be more consistent with "myocarditis rather than an ischemic process." Tr. 3 at 676-77. Later, Respondent asked whether he thought oligodendroglial swelling was a finding that would be consistent with hypoxic ischemia, to which Dr. Shane replied:

> It's not a finding I would rely on with hypoxic ischemia, and I'll tell you why. Because these are reactants that come in later. This would have to be a very, very long period of hypoxia ischemia. If this is a hypoxic encephalopathy due to ventilator brain then I see oligodendroglia. In an acute episode I don't think it's very consistent at all, and these oligodendrocytes or oligodendroglia are turned on, they are reactive. That's why we have the enlargement. They are proliferated. And for that to occur takes time. Time is the essence of that pathologic change.

Tr. 3 at 686. Dr. Shane added that the timing he envisions was "certainly longer than three or four hours." Tr. 3 at 687.

When asked if the pathological findings in the heart had been specific or nonspecific to mercury toxicity, Dr. Shane conceded that, "They're non-specific findings until the medical literature tells you that's what happens, then they become specific because you tie that finding to the medical literature." Tr. 3 at 677. The implication of that concession is that the same cardiac findings could be present in a case with no mercury involvement whatsoever, which Dr. Shane said would lead him to "search for another cause of myocarditis." *Id*. Unfortunately, Dr. Shane exposed the illogic of his perspective regarding the entire case in explaining this point:

> [W]hen you have myocarditis, you have literature that tells you this happens with ethyl mercury chloride toxicity. You have the administration of thimerosal. One plus one plus one is three and that takes it out of the realm of unknown or undetermined aetiology and places it into the framework of this plus this plus this. We have the clinicals, we have the pathology, and we have literature, and we have what we see.

Tr. 3 at 677. Dr. Shane's explanation gives the appearance of scrounging for supportive indicia to bolster the conclusion he desires as an *idee fixe*, instead of approaching the indicia in an inductive, empirical, diagnostic approach that could guide the Court's analysis helpfully. This problem was recurrent in Dr. Shane's testimony.

Dr. Shane acknowledged that the case of Thomas Kolakowski, and others like it, were provided to Dr. Shane to determine whether mercury toxicity had been a causative factor, which led him to focus on the central nervous system. Tr. 3 at 678. He focused initially on the cerebellar, granular cells, because, in his "in-depth examination of the brain ... the changes in the granular cell layer and the changes in the Purkinje cells [were] certainly present." Tr. 3 at 678. He explained his analytical approach as examining the brain slides and noting any "abnormalities," before comparing those abnormalities with his findings so as to "come to a conclusion as to what caused these abnormalities in the context of what else is present in this body and what's the clinical history." Tr. 3 at 679. Specifically, regarding those granular cells of the cerebellum and his estimate of twenty percent "drop out," Dr. Shane admitted that, due to their high amount of chromatin,[104] those cells are very dense and difficult to differentiate, and that his estimate of twenty percent is not an exact count, but a "semi-quantitative" estimate. Tr. 3 at 680. Respondent produced a printout of microscopic magnification of granular cells, and challenged Dr. Shane to pick out granular cells therein, to which Dr. Shane responded:

> I can't see granular cells but I do see that separation, and that separation to my eye is a pathologic change and it is due to a contracture in one of the layers that you're seeing, most likely the intensely cellular area is actually pulling away from the adjacent tissue so that would indicate to me that there has been some loss of cells or stroma in that area causing this separation. So I think the separation is a pathologic change. The pathologic change in the cerebellum. I don't know what the cause is,

[104] Chromatin is "the more readily stainable portion of the cell nucleus, forming a network of nuclear fibrils. It is a deoxyribonucleic acid attached to a protein (primarily histone) structure base and is the carrier of the genes in inheritance. It occurs in two states, euchromatin and heterochromatin, with different staining properties, and during cell division it coils and folds to form the metaphase chromosomes." DORLAND'S, *supra*, at 359.

> but it is abnormal. Again, a possible cause is again, tying in with what I saw when I looked at these under my own microscope. It could be the cell loss in that granular layer, that inner-granular layer, is what's causing that shrinkage.

Tr. 3 at 682. Dr. Shane thus opined that his assessment of the granular cells in Thomas' slides remained consistent with the presumed effects of ethyl mercury toxicity, and stated the basis for that opinion was "the publications that support that type of change occurring in the brain of individuals who have been poisoned with ethyl mercury." Tr. 3 at 686.

Respondent questioned Dr. Shane about another autopsy finding that he had noted–degeneration and necrosis of the Purkinje cells, which Dr. Shane had attributed to cases of ethyl mercury toxicity. Tr. 3 at 695. He based his opinion, "that mercury toxicity affects Purkinje cells," entirely on the Cinca article, which had published photographs of "abnormal Purkinje cells with smaller outlines with abnormal cell shape and with shortened exomes." Tr. 3 at 695-96. However, when asked if necrosis of those Purkinje cells was a characteristic trait of asphyxia, Dr. Shane demurred, stating:

> No, it's not. It's a cerebellar change and it's not a marker that I would use for hypoxic encephalopathy. The section of the brain that's most sensitive to hypoxia is the hippocampus and I don't see any specific changes in the hippocampus here beyond some of the neuronal necrosis that I see elsewhere that would suggest hypoxia. So no, it's not a change that I would say is characteristic in any way, shape or form of hypoxic encephalopathy.

Tr. 3 at 696-97. Dr. Shane conceded that Purkinje cell necrosis was not at all inconsistent with asphyxia, just that it was "not a finding that particularly, in any particular way, points to asphyxia." Tr. 3 at 697. He explained his thinking on this subject:

> The cerebellu[m] is not extremely sensitive to asphyxia and hypoxia. The cerebral cortex is far more sensitive, and as I mentioned, the most sensitive area of the cerebral cortex is the hippocampus. If I was looking for hypoxia I would not be going to the cerebellum for these changes, I'd be going to the cortex, and particularly to the hippocampus for those changes.

Tr. 3 at 697.

Respondent raised the distinction between karyorrhexis[105] (necrotic fragmentation of cell nuclei) and apoptosis (acceleration of preprogrammed cell death) with Dr. Shane, as had been done with Dr. Connor. Tr. 3 at 697. Respondent challenged Dr. Shane that one would not be able to distinguish between the two by "viewing pathological slides like we have here, in the manner in which it was done," but Dr. Shane disagreed, insisting that one could identify "the beginning of nuclear fragmentation" which he believed represented karyorrhexis. Tr. 3 at 697-98.

[105] Karyorrhexis is "rupture of the cell nucleus in which the chromatin disintegrates into formless granules which are extruded from the cell." DORLAND'S, *supra*, at 970.

Moving on to Dr. Shane's analysis of Thomas' cardiac effects, Respondent asked Dr. Shane to label the EKG strips with what he saw as heart wave segments, and he labeled a rather extensive portion as all consisting of small QRS segments, although he had difficulty doing so with great precision "because the pattern [was] slurred a little bit." Tr. 3 at 690-693.

When challenged by Respondent to cite peer-reviewed medical literature in support of his theory of sudden death caused by mercury toxicity, Dr. Shane did not comply, but restated his theory of insult:

> Well, mercury toxicity can lead to myocarditis. Myocarditis can result in a dysrhythmia, and many dysrhythmias are inconsistent with life. So that's how it would cause sudden death.

Tr. 3 at 698. He disagreed with the suggestion that such a process would have to occur "[o]ver a long period of time," and insisted a person "could die the very next day" as a results of mercury toxicity-related myocarditis, because "when you trigger a dysrhythmia is not predictable, but you're at high risk for dysrhythmia every second you have interstitial myocarditis." Tr. 3 at 699.

Dr. Shane agreed that "ischemic, hypoxic injury causes edema in the brain," and that "edema in whatever organ it might occur in causes the weight of that organ to increase from its pre-edema state." Tr. 3 at 729-30. However, he contended, in the case of Thomas Kolakowski, it was mercury that caused the increased organ weight:

> It did in this case by causing the central nervous system changes that were present in addition to the cerebral edema which was present here. It did in this case by causing edema in the heart which added to the heart weight, although it's a very, very small heart in an infant. And the passive congestive changes in the lungs and viscera are certainly related to the mercury and the interstitial myocarditis that the mercury caused.

Tr. 3 at 730. When questioned about the support for such a contention in the peer-reviewed medical literature, Dr. Shane referred to the Cinca study, which "does talk about the cerebral edema" and "the interstitial myocarditis that we see," and "does talk somewhat about the organ congestion that occurs certainly if you have heart failure." Tr. 3 at 730-31. Finally, though, Dr. Shane admitted that these indicia were "not specific to mercury, but heart failure due to any cause will cause the organ congestion that we see in the lungs and viscera." Tr. 3 at 731. When pressed further to comment as to whether "findings of congestion in organs significant in determining cause of death," or whether they were nonspecific, Dr. Shane answered:

> They can be specific when you have the extensive congestive changes that you see in these lungs. This tells you a lot. It tells you that this is most likely a heart failure, and it tells you most likely this is heart failure over a prolonged period of time because of the amount of increased weight that we have. And you correlate that further with the clinical history of respiratory symptoms, it correlates with a sub-acute, a semi-remote congestive heart failure that caused the clinical respiratory insufficiency and resulted in this massive increase in lung weight to 180 grams.

Tr. 3 at 731-32. Given the fact that Dr. Shane reiterated these points so frequently, the Court might have expected him to explain how those findings or conclusions support a theory (never proffered) of how thimerosal-containing vaccines caused sudden death. When asked the same question a second time, Dr. Shane stated that congestion in the organs was not specific to mercury toxicity: "Of course not." Tr. 3 at 732.

When asked if congestion in the organs was a finding consistent with hypoxic ischemia as a cause of death, Dr. Shane replied that "congestion would be caused by heart failure," regardless of what caused the heart failure. Tr. 3 at 732. But then Dr. Shane parried by stating that "in this case we know what caused the heart failure or dysrhythmia because we have the interstitial myocarditis." *Id*. When asked if alveolar dilation or capillary dilation in the lungs was consistent with hypoxic injury, Dr. Shane said no, that it is "far more consistent with heart failure," and was inconsistent with hypoxic injury, because, "With hypoxic injury you would not have congestion of the lungs until you had heart failure, then the congestion would begin and be ongoing for some period of time." *Id*. When asked if oral and nasal bleeding, such as was observed in this case, was consistent with hypoxic ischemia, Dr. Shane insisted that it was not, but that such a finding would be "consistent with heart failure," and that "the bloody mucous, the bloody edematous fluid is frequently... found in the nose and mouth respiratory tract [as] part of the failure." Tr. 3 at 733. When pressed as to whether "oral-nasal bleeding [was] commonly found in SIDS cases," Dr. Shane did not view that as a common finding in SIDS cases, or "a finding on which a diagnosis of SIDS would be based." Tr. 3 at 733-34. He added that in the case of Thomas Kolakowski, Dr. Shane perceived "no other cause of oral nasal bloody material other than the heart failure." Tr. 3 at 734.

Respondent queried Dr. Shane as to why, since the medical examiner for Thomas Kolakowski's autopsy was board-certified in both Neuropathology and Forensic Pathology, why the medical examiner did not note or record any of the pathological findings or conclusions reached by Dr. Shane. Tr. 3 at 737. Dr. Shane responded by pointing out that the medical examiner did not perform a microscopic analysis of Thomas' case, but relied solely on the gross findings, which Dr. Shane reported was "not uncommon for forensic pathologists or medical examiners. They will prepare the slides but they won't necessarily do a microscopic study." Tr. 3 at 737-38. Dr. Shane said this was indeed the case, even though the medical examiner retained several pathologists to review the case. Tr. 3 at 738. When Respondent challenged Dr. Shane as to why no microscopic analysis was performed, Dr. Shane replied:

> Not in the forensic arena. In the forensic arena very frequently you keep the tissues, you prepare the slides in some cases, in some cases you don't even prepare the slides, you just keep the tissues. And you opine, as Dr. Ross did, Dr. Ross did what I would expect the medical examiner to do. As I said, he's very, very, very good at what he does, and very cautious.

Tr. 3 at 738-39.

Respondent challenged a portion of Dr. Shane's written expert reports which postulated that "thimerosal is ethyl mercury, and the neurotoxicity of ethyl mercury exceeds that of methyl mercury, about which abundant literature exists from accidental methyl mercury poisonings throughout the world." Tr. 3 at 708. When Respondent questioned how methyl mercury poisoning studies could

prove that ethyl mercury was more neurotoxic than methyl mercury, Dr. Shane could not provide any supportive reference in the filed medical literature, but asseverated that "there is literature out there ... so much literature in this case, that would indicate that ethyl mercury is more toxic and achieves higher tissue levels than methyl mercury." Tr. 3 at 709. He added that he did not derive that point from Dr. Lucier's opinion, but gleaned it from medical literature that is "buried in the large number of publications that have been submitted in this case as supportive." Tr. 3 at 710. However, he deferred to Dr. Lucier on the matter of toxicology, and deferred to Dr. Connor on the interpretation of EKG results. *Id*.

Dr. Shane hedged his deferral to Dr. Connor, at least as it related to diagnosing congestive heart failure:

> We have a lot of lung problems pathologically, and this child had respiratory problems before death. The congested liver is certainly there pathologically. We had decreased appetite. So those correlations, I don't need anyone else for.

Tr. 3 at 710-11.

Respondent also challenged the apparent *non sequitur* in Dr. Shane's expert report, where he stated that, because ethyl mercury is especially lipophilic, it is particularly toxic to the brain, which contains many fatty components, such as myelin. Tr. 3 at 711. Respondent asked Dr. Shane whether he thought ethyl mercury was comparatively more lipophilic than methyl mercury, in a way that distinguished the two, or was otherwise relevant to the discussion. *Id*. Dr. Shane could not answer specifically, but said his impression "taken off the top of [his] head," was that "they would have similar tissue minding affinities." *Id*. He conceded that "to the extent [he was] distinguishing methyl mercury [from] ethyl mercury," he did not intend to indicate "that ethyl mercury is especially neurotoxic because it's lipophilic and methyl mercury is less so." *Id*.

When challenged on his statement, made in his expert report, that methyl mercury's half life is variable, with certain individuals experiencing "a profoundly longer half life," Dr. Shane could not cite to a supportive source of this information, but stated that there were "so many papers" that said so that were "part of that general literature." Tr. 3 at 712. He said that such variability was based on "physical condition," "metabolism," and "age." *Id*. When challenged as to why he did not provide supportive reference in his expert report for this formulation, Dr. Shane replied that it was "such boilerplate of good toxicology that a citation wasn't necessary." Tr. 3 at 712-13.

Finally and tellingly, the Court recites the following interchange, during which Dr. Shane admits that he can proffer no mechanism of injury by which ethyl mercury from a thimerosal-containing vaccine could cause arrhythmia, interstitial myocarditis, cardiac arrest, or sudden death:

> Q ...[D]o you know what actually caused his death? Specifically the mechanism?
>
> A Yes. I think that this was a death associated with ethyl mercury toxicity. I think there are central nervous system manifestations that we talked about over and over. I think there are cardiac manifestations that we talked about. I think there are clinical findings that go along with those cardiac manifestations that occurred prior

to the evening of his demise. He was symptomatic. He did have these symptoms that correlate with heart failure. And this is, until prove otherwise, a heart failure death in face of interstitial myocarditis, and there is no other cause for the heart failure which was symptomatic prior to his going to bed that night before his death.

Q So is mercury in the brain, is that what caused death here?

A Mercury in the brain caused changes in the brain. That's certain.

Q Was it actual mercury in the heart that caused death here?

A Actual mercury in the heart? I don't know if that mechanism of interstitial myocarditis is directly due to the mercury. It is an associated finding with ethyl mercury toxicity. I don't know that anyone has worked out that precise mechanism. If they have, I don't know about it.

Q Was it an immune process that caused death?

A That's a very likely possibility. This could be an antigen antibody response. Certainly with antigen antibody responses you can have interstitial myocarditis. It works with that finding. And certainly the timing of these events, a dose and then a challenge later on, that speak for hypersensitivity. The skin rash may reflect hypersensitivity. And the myocarditis could be hypersensitivity. So yes, that would work.

Q So it could have been antigen hypersensitivity, but your opinion and your pathological review, you support the fact that it's mercury toxicity, correct?

A I talk about it being mercury toxicity. Again, in terms of the interstitial myocarditis, I don't know what the mechanism for that was. It is a finding. It's a finding that is reported in ethyl mercury toxicity deaths. I see it here. One and one equals two.

Q In either one of your reports, why didn't you draw, as a differential possibility, an immunologic process?

A I didn't address the immunologic process because I didn't address the further mechanism of the myocarditis. But as you raise it, it certainly is a probability.

Q So you don't really know the mechanism then.

A I don't know the precise mechanism of the myocarditis. Is there a strong possibility it's antigen antibody? Yes, it works.

Tr. 3 at 735-37.

4. Jeffrey Brent, M.D.

Dr. Brent worked at the time of trial as a professor of pediatrics and internal medicine. Transcript of Hearing on 24 June 2008 (Tr. 4) at 751. Dr. Brent has a Ph.D. in biochemistry and a Master's degree in molecular biology, and he attended medical school at the State University of New York at Buffalo. Tr. 4 at 752. After medical school, he interned as a junior resident at Harvard, and

"ultimately completed a primary residency in emergency medicine at Emory University School of Medicine, Grady Memorial Hospital in Atlanta." *Id*. Thereafter, he underwent "a two-year subspecialty fellowship in the area of medical toxicology." *Id*.

Dr. Brent has been awarded a distinction by the European Association of Poison Control Centers and Clinical Toxicologists, is a member of professional societies such as the American College of Medical Toxicology, the American Academy of Clinical Toxicology" as well as "the European Association of Poison Control Centers and Clinical Toxicologists." Tr. 4 at 753-54. He is actively engaged in peer review, reviewing approximately one article per week "for a variety of medical journals ... toxicology journals[,] ... the New England Journal of Medicine, [] the Journal of the American Medical Association, a number of journals, both in the United States and worldwide." Tr. 4 at 754-55. He "was Editor-in-Chief of a major review journal, the major critical review journal in the world in the area of medical toxicology ... called Toxicological Reviews," and has served as a senior editor of Clinical Toxicology for over a decade, "which is the largest peer-reviewed original research publication in the area in the world." Tr. 4 at 756.

Dr. Brent's private practice focuses entirely on medical toxicology, and has treated "quite a number" of heavy metal toxicity patients pursuant thereto, including several dozen patients with mercury toxicity. Tr. 4 at 761. He has advised treating doctors as a consulting doctor on two or three times that many mercury toxicity patients, where his expertise as a medical toxicologist was necessary for treatment. Tr. 4 at 761-62. Most of the cases in the United States of mercury toxicity come as a result of using mercury in industrial or commercial applications, not through intake of organic mercury, inasmuch as "organic mercury poisoning in this country is extremely, extremely rare. You don't really see many cases of that." Tr. 4 at 763-766.

Dr. Brent testified that there are between 300 and 400 medical toxicologists in the entire United States, and that there are a "several hundred in Europe[,] ... just a handful in South America and Africa[,] ... [and] a couple dozen in the Middle East." Tr. 4 at 771. An exact figure is only possible in this country because only here is there an official certifying body that registers their member tally. *Id*. Dr. Brent himself is quite acquainted with this fact, because he has served as "one of the elected board of directors of the American College of Medical Toxicology," and thus has "a very good sense of what the entire field is about." Tr. 4 at 773. "In addition, one of [his] duties at the American College of Medical Toxicology is ... Chair of the Practice Committee, so [he is] very aware of all the aspects regarding toxicology certification in medicine." *Id*. He also stated that the American Board of Pathology does not have a specific board certification for toxicology, nor does it "offer any credentialing in ... medical toxicology." Tr. 4 at 774.

Dr. Brent differentiated medical toxicology from the study of toxicology outside of the medical context thusly:

> [T]oxicology in general is just simply the science of the adverse effects of chemical substances on living systems. So you know, it could be anybody studying adverse effects of any chemical. It could be on plants, it could be on people, it could be on fish, and so on. So there's no actual formal requirements for calling yourself a toxicologist. In contrast, there are for calling yourself a medical toxicologist.

> Medical toxicology is a recognized subspecialty by the American Board of Medical Specialties, which is the oversight organization of all the medical specialties, and there are very defined specialties and subspecialties. And medical toxicology is a very defined subspecialty. And a medical toxicologist, therefore, is a physician who, by virtue of completing an accredited post-residency two-year fellowship, and completing an examination, which I have to sit for a recertification periodically, is subspecialty board-certified to act as a medical toxicologist, and to call themselves a medical toxicologist.

Tr. 4 at 770.

Dr. Brent discussed the clinical symptoms and sequelae of mercury exposure and toxicity:

> [F]or those people who inhale mercury vapors and get very sick very fast, we're talking there about just elemental mercury, or you know, quicksilver. The first thing that happens is it goes to your lungs, it's very active on your lungs, and causes a severe pulmonary syndrome. And then you can evolve into multiple-organ system failure very quickly. Your kidneys will fail, you can have seizures. Once you get past the acute stage, very often these people will, over a period of time, develop a more typical mercury-related syndrome, which would be consistent, which is typically a tremor, constriction of visual field, an articulation problem with regard to speech, some ataxia, difficulty walking, peripheral neuropathy involving the sensory nerves. ...Sometimes people are stuck with a little bit of peripheral neuropathy. It depends, you know, if you're really seriously mercury-poisoned, you can have more severe sequelae.

Tr. 4 at 766-67.

Dr. Brent confronted Petitioners' ultimate theory of causation for its ephemeral, and, he believes, almost impossible quality, especially as it relates to toxicology:

> I didn't really hear a lot of a sort of coherent hypothesis of exactly what happened. I know Dr. Lucier did not offer any specific opinion about Thomas Kolakowski. Dr. Connor, as I recall, and correct me if I'm wrong, on cross-examination when he was asked about that, said he thought it was an immunological reaction to the vaccine. And Dr. Shane, I don't recall him actually, he didn't actually articulate a specific hypothesis that I could put my finger on in terms of what actually killed Thomas Kolakowski, and how this was related to mercury.

Tr. 4 at 768. He concluded that reaching the conclusion urged by Petitioners "is impossible."[106] Tr. 4 at 769.

Dr. Brent discussed the qualities and history of thimerosal and its use:

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[106] "I would say as close as we can ever say in science -- you always learn never say always, never say never. But I would say it's as close to impossible as anything I've ever seen in medicine.... I would say it is neither plausible nor possible." Tr. 4 at 769.

> Thimerosal is a preservative. If you look at the history of thimerosal, it's been used in pharmaceuticals since about, as far back as I can tell, the 1920s. There was an original safety study done in 1931 by Powell and Jamieson. For its time, it was a very excellent safety study. It pales in comparison with the kind of safety studies that we would consider to be appropriate today.... [Its first widespread application was as a preservative of blood plasma, used during World War II for combat trauma wounds.] And the experience was, and it's been well published, it's very well tolerated.

Tr. 4 at 774-75. He countered Dr. Lucier's statement that the use of thimerosal in vaccines came to light only around 1999: "I guess that's when Dr. Lucier may have discovered that it was in vaccines, but certainly it's been -- it's been on the product label, in the package insert, for as long as package inserts have been around, to my knowledge." Tr. 4 at 777. Likewise, he countered Dr. Connor's depiction of thimerosal as "an activator to help distribute the vaccine into the tissue." Tr. 4 at 778, citing Tr. 2 at 472-73. "I'm not sure where he got it. But no, it doesn't do anything like that. Thimerosal is simply a preservative." Tr. 4 at 778. Dr. Brent clarified a point potentially clouded by Dr. Lucier's testimony, that ethyl mercury is one of two severable parts of the thimerosal molecule, but it does not constitute half of a vaccine; it is between one and five one-thousandths of a percent of the vaccine's constituent parts. Tr. 4 at 779

Next Dr. Brent focused on the central question of causation, and he began by addressing the difficulty of tying general or diffuse symptoms to specific chemical toxicity (a point also made by Dr. Lucier). Tr. 4 at 780. This difficulty is surmounted by a scientific process characterized by Dr. Brent as a "formula" and a "formal exercise that absolutely must be done." Tr. 4 at 780-81. This process "involves three steps ... the *what-can-did* approach." Tr. 4 at 781 (emphasis added). He started with the "what" question, which involves identifying the substances to which the patient has been exposed: "The first question is what was the person exposed to. And if they were exposed to a chemical, what were they exposed to, how much, what was the dose." Tr. 4 at 781. For the Court, this question is typically answered as a question of fact from medical records, and vaccination records in particular. Dr. Brent continued, to address progression to the questions of "can it" and "did it":

> [O]nce you've identified what the chemical condition is, the next question is can that particular chemical exposure cause the particular condition, the particular disease in question. If it can't cause that disease, then you can stop right there. If it can, then you have, then you have to go to the next step. ...I think the first is what you call in the general causation, if I understand that correctly. The next thing is if the chemical can cause the disease in general, did it cause the disease in this specific individual. I think that's what I believe is called specific causation. And that depends on the circumstances of the exposure. I mean, we're all exposed to chemicals all the time that can cause lots of problems. As we sit here in this room, we're breathing in all kinds of chemicals. So the question is, is the chemical capable of causing the condition; and if so, were the exposure conditions appropriate. Was it the right dose of the chemical. Was it a sufficient dose of the chemical to cause the problem that is being investigated.

Tr. 4 at 781-82. The Court, and the Undersigned in particular, has long relied on such an approach.[107] Part and parcel to the question of "can it," explained Dr. Brent, is taking into consideration the dosage of the exposure:

> [Dosage] is the most fundamental thing we look at.... This goes back to Paracelsus,[108] who was actually one of the individuals who provided the early detailed descriptions of mercury toxicity. But the axiom of Paracelsus is probably the most fundamental principle of toxicology today....
>
> If you want to make an assessment about whether a chemical caused an adverse effect, the fundamental question is did the person get the sufficient dose. As Paracelsus said in his translation, poison isn't everything, and no thing is without poison. The dosage makes it either a poison or a remedy....
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> And so the real question is what was the dose, was the dose sufficient to cause the condition. If it is capable, if general causation exists. And so we have this dose-response relationship. And as you see there, as we say, there's no such thing as poisonous substances, there are only poisonous doses. And that's true of all things.

Tr. 4 at 782-83. He added that dosage remains "the most fundamental principle" in medical toxicology. Tr. 4 at 784. He quoted from a well-respected Toxicology source (Casarett & Doull) to state:

> The characteristic of exposure and spectrum of effects come together in a correlative relationship customarily referred to as the dose-response relationship. This relationship is the most fundamental and pervasive concept in toxicology. Indeed, it is an understanding of this relationship that is essential for the study of toxic materials.

Tr. 4 at 786. Also, he quoted from a clinical toxicology textbook (*Clinical Environmental Health and Toxic Exposures*) to state:

> One of the most important concepts in toxicology is the dose-response relationship. The underlying premise is that any compound can be toxic if it is encountered in large-enough doses. No matter what the compound's potency or how little a

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[107] *See Pafford v. Sec'y of HHS*, No. 01-0165V, 2004 WL 1717359, 2004 U.S. Claims LEXIS 179, *16, slip op. at 7 (Fed. Cl. Spec. Mstr. Jul. 16, 2004), *aff'd*, 64 Fed. Cl. 19 (2005), *aff'd* 451 F. 3d 1352, 1356 (2006) ("this court perceives no significant difference between the Special Master's test and that established by this court in *Althen* and *Shyface*"), *rehearing and rehearing en banc denied*, (Oct. 24, 2006), *cert. den.*, 168 L. Ed. 2d 242, 75 U.S.L.W. 3644 (2007).

[108] Paracelsus is the pseudonym of Philipus Aureolus Theophrastus Bombastus von Hohenheim, who was "the 'Luther of Medicine,' 1493-1541, a Swiss physician and alchemist who defied the authority of Galen and Avicenna and condemned all medical teaching not based on experience. His alchemical researches led to the introduction of such substances as lead, sulfur, iron, and arsenic into pharmaceutical chemistry. Although he was far ahead of his time in many of his observations (*e.g.*, on metabolic and on occupational diseases), much of his thinking was made obscure by his mysticism." DORLAND'S, *supra*, at 1361.

> compound is necessary to produce an effect, its respective toxic dose threshold must be surpassed to produce toxicity.

Tr. 4 at 787.

Dr. Brent described the dose-response curve as "a bell-shaped curve," which means "different individuals will require a different dose to have the same effect. Most individuals will be someplace quite near the center of the curve, but ... about 95 percent of the population will be someplace in that curve." Tr. 4 at 788. This does not mean that individuals at the extreme of the curve's distribution will be "hyper-susceptible," however:

> Hyper-susceptibility to a chemical is something very, very different. Hyper-susceptibility is what you see here, where there is a population of individuals that have a dose-response curve that has shifted all the way over to the left, so that they respond to chemicals at doses well below what the general population responds to.... You would have a dose-response relationship, it would just be at much lower doses.

Tr. 4 at 788. Along the same vein, when asked whether "a genetically susceptible population to mercury [has] ever been identified," Dr. Brent responded, "No. There is no known accepted hyper-susceptible population to mercury toxicity." Tr. 4 at 789.

Discussing the scientific validity of Dr. Lucier's defense of "look[ing] at products that are similar, and extrapolat[ing] what is known to the unknown" (Tr. 1 at 47), Dr. Brent stated:

> It depends on what you are doing it for. If you are a regulator, and you're trying to come up with regulations, then, of necessity, you have to make assumptions. You have to make assumptions to fill in what you don't know.... Whether that's true or not is not known. It could be, they could be very different. But in the regulatory community, where you have to make regulations, where you have to come to conclusions, you tend to make a lot of assumptions.... So for those purposes it is an accepted thing to do.

Tr. 4 at 791. However, outside of an extra-precautious regulatory realm, Dr. Brent finds it important to realize the likelihood of large differences between compounds:

> In terms of asking the question, does this particular molecule act similar to another molecule that has some chemical similarity to it, on a scientific basis, that's very different. You cannot. Because one of the things we know, that very, very small changes in a molecule can cause it to have very, very different properties, toxicological properties.
>
> ...[For instance, if you] want to know what happens to methanol by studying ethanol, ... you would be profoundly, profoundly misled. Because methyl alcohol [is] what's found sometimes in contaminated moonshine. If you drink methanol, you are likely to die. And if you get insufficient dose to die, you are likely to end up permanently blind. It's a profound retinal toxin. That's methanol. Ethanol, on the other hand, as we were just talking about, has some actually not-so-bad properties. And it only takes really huge, huge doses before you get yourself into a lot of problems, before

> you get yourself into a lot of problems, or taking large doses over a very chronic period of time. So methanol and ethanol are dramatically different compounds in terms of their toxicological properties. And trying to study one to get an idea of what the other one does can be extremely misleading.... There are some similarities between methyl mercury and ethyl mercury, but there are also some profound differences.

Tr. 4 at 791-95. Additionally, he agreed that "significant scientific literature [] demonstrates that ethyl and methyl mercury are actually quite different" in their toxicologic properties, and that the main body of medical literature does not equate the two for purposes of toxicologic study. Tr. 4 at 795. He cited the Casarett & Doull text to say that "no other [metal] better illustrates the diversity of effects caused by different chemical species than does mercury." Tr. 4 at 796-97.

Moving on, Dr. Brent discussed the EPA reference dose that formed the foundation of Dr. Lucier's testimony. In contradistinction to Dr. Lucier's statement that exceeding the reference dose in a singular exposure threatens an individual with increased risk for neurological injury (Tr. 1 at 222), Dr. Brent clarified that the reference dose is an average daily dosage below which anyone is virtually sure to be unsymptomatic.[109] Tr. 4 at 797-98. Dr. Brent disagreed with Dr. Lucier's statement about comparability between organomercurials (as demonstrated *supra*), stating that the reference doses as between methyl and ethyl mercury are different, because "[t]hey are different compounds," and because the EPA assigns advisory reference doses[110] only for specific chemicals, and only when they are deemed to pose a sufficient risk; the EPA has never assigned a reference dose for ethyl mercury or for thimerosal. Tr. 4 at 798. Dr. Brent likewise argued that the EPA's reference dose is not a safe/unsafe demarcation which, if exceeded, would more likely than not lead to adverse effects:

> What the reference dose is, is it's a dose below which the EPA will say we don't think anybody is going to have any problem. It is generally set very far below the level at which anybody would say there is likely to be a problem. Because the position is always that we don't know.
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> ...[W]e start with a dose where we know that somebody will have a problem, that may be adverse to the population; and then we very dramatically lower that dose by accounting for uncertainty, by putting in what they call uncertainty factors. And they ultimately come up with a dose that we say, well, we're pretty comfortable saying if you're all the way down here, you're going to be pretty safe. But, and they don't say if you're above that, you're necessarily going to have toxicity.

Tr. 4 at 799. He said that this same logic was operative in designating the reference dose for methyl mercury:

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[109] Both the reference dose and the benchmark dose are distinct from the threshold dose: "For any given chemical exposure, you have to get to a certain dose before you're going to have an adverse effect." Tr. 4 at 838.

[110] "[W]hat is applicable to one is not necessarily applicable to the other. Generally you don't apply a reference dose from one compound to another compound, because you base the reference dose specifically on data on that particular chemical." Tr. 4 at 798.

> [The EPA researchers tried] to come up with the most sensitive end point they [could] find for an adverse effect on humans. That's where you start. And then you put in your uncertainty factors under that. And if you remember, we talked about Minamata disease, where it was shown that in the population that took methyl mercury, the individuals who tended to get sick were the children who were exposed *in utero*. And sometimes the mothers would be perfectly fine, yet the children would be profoundly affected by the methyl mercury when they were exposed to it *in utero*, through the mother. So they chose, therefore, as their most sensitive population prenatal exposures. And so, and then they looked and they found the most sensitive measure that they could find for adverse effect, based on prenatal exposures to methyl mercury, and they used that to set, as a starting point to set the reference dose. And then they put in all their uncertainty factors.

Tr. 4 at 800-801. More to the point of the central question in this matter, Dr. Brent did not believe that the EPA reference dose for methyl mercury was based on the potential for cardiac effects:

> The reference dose, the most sensitive end points that they could find were really based on some very sensitive neuropsychological testing that was done on a heavily mercury-exposed population in the Faroe Islands.[111] And so it was really based on

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[111] Dr. Brent went into some detail concerning the methods and data of the Faroe Islands studies:

> [W]hat they did was, at birth, they took a large number of parent-child, parent-newborn pairs. And they studied them at birth, and then they prospectively followed them over a period of a number of years. And the study is still going on. And at various points over the years, they evaluated them, they evaluated these children. And one of the things that they did at the time of birth is they got umbilical cord, they got umbilical cord blood, blood from the placenta that was going to the mother. And they measured the mercury concentration in the blood. And they also did hair. And so they were able to correlate any effects that they saw with the mercury levels that these children were exposed to *in utero*.... [T]he reason they used this group is because this is a much more heavily methyl-mercury-exposed population than in the U.S.... [T]he average concentration of mercury in the umbilical cord blood, in the Faroe Islands, was a little over 22 micrograms per liter. Now, that is a way, way high level.... To give you a point of reference, in the U.S. almost all blood mercury levels for people is under about 10 micrograms per liter. So here they have a population that chronically runs much higher blood mercury level related to methyl mercury than in the U.S.
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> They followed this whole cohort out for up to 14 years, and they've been studying them periodically.... What they have found over that period of time is that for all intents and purposes, these children are perfectly normal. There have been no clinically observed defects that they have been able to find that are at all related to -- because they look at each child with respect to what their prenatal exposure had been, because they had determined that. And they have not been able to find any clinically obvious effect that is related to either their prenatal exposure to methyl mercury, or their prenatal exposure to PCBs.... what they did is at various points along the way, they tested them with very sensitive neuropsychological testing techniques. And they saw that in some of the children with the higher cord blood levels, that they could find some abnormality on these tests that were correlated with how high the blood level was. It was probably related to the methyl mercury.
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> ...Another thing, by the way, and this is a bit of a confounder in that study ... is that the pilot whales also contain a good deal of PCBs. And PCBs are known to potentially have adverse effects on outcomes, intellectual outcome in children, as well.

Tr. 4 at 802-805.

> performance on a neurological test. There's nothing in the reference dose whatsoever about any adverse cardiac effects.

Tr. 4 at 801.

Dr. Brent also spent some time discussing the statistical analysis that went into assigning a safety reference dose for methyl mercury:

> They took a point where five percent of the children are likely, where five percent of the children had a substandard score on the Boston Naming Test. And they looked, and the average cord mercury level for those children was, as you can see there, 85 micrograms per liter. So that's pretty high. Remember, in the U.S. it's under 10.... [T]he 85 micrograms per liter was an average. And there was, of course, a bell-shaped curve, two standard deviations. And the way you can express two standard deviations is either in terms of a bell-shaped curve, or what's called confidence intervals ... and they took the lower limits of the confidence intervals as a safety factor; not just the average value, but the lower limit of the confidence interval. And so they found some children would be down as low as 58 micrograms per liter. And so they took that 58 micrograms per liter, and they put in then a safety factor or an uncertainty factor of 10. And so they got down to 5.8 micrograms per liter in blood. And they said basically, for the purposes of our reference dose, we think if anybody can get their blood level down in the 5.8 micrograms per liter, they're fine [meaning] no chance of any adverse effects, based on this Boston Naming Test.
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> And then they said well, how much methyl mercury will you have to ingest in order to get 5.8 micrograms per liter of blood. And you can calculate that, this data allows you to calculate it. And the calculation basically came out to be if you ingest 0.1 microgram per kilogram per day of methyl mercury, your blood level should be about 5.8.[112] So that's where the reference dose of 0.1 microgram per kilogram per day came from. It's based on a sensitive neurological indicator in prenatal exposures.

Tr. 4 at 806-07.

Next, Dr. Brent discussed the National Academy of Sciences' benchmark dose (BMD), and how that figure could have been confounded by the prevalence of PCBs in the Faroe Island data. The benchmark dose used for methyl mercury was based, said Dr. Brent, on "the level of cord mercury in the blood that was necessary to cause an adverse effect on ... the Boston Naming Test" among the Faroe Island subjects. Tr. 4 at 808. However, the researchers chose not to factor out the PCB effects from their data, as a precaution, which may have skewed their data findings:

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[112] The Court notes again that calculating an intake rate based on a certain blood level requires making standardizing assumptions about blood retention and clearance rates, which vary across a human population. Also, based on the Burbacher data, which demonstrate that methyl mercury is retained much longer in the bloodstream than ethyl mercury, it is quite clear that the same 0.1 μg/kg/day ratio set for methyl mercury is not transferably applicable to ethyl mercury.

> [W]hen they adjusted for PCBs, for the adverse effects of PCBs, then it's actually required a methyl mercury level of 184 micrograms per liter to have this adverse effect on the Boston Naming Test with a lower confidence interval of 71. But it was felt for technical reasons not to adjust for PCB. It was a regulatory decision; you always err on the conservative side on regulatory decisions. So they just used the unadjusted figure....
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> [The EPA] used this exact table from the National Academy of Sciences. And once again, for the EPA risk assessments, they used the unadjusted group....
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> [H]ad they taken it into account, then what would have happened is, if you'd look at that formalism, the actual value that would have been necessary on the average to cause the adverse effect they were looking at the Boston Naming Test was about 184 micrograms per liter in blood. Once again, remember, in the United States we tend to be under about 10 micrograms per liter.... [F]rom there they would have had to calculate a reference dose, which would have been higher than the current one.

Tr. 4 at 809-10. Dr. Brent explained the implications of the researchers' precautionary decision:

> Well, as it should be, the reference dose is a very conservative number. It's based on prenatal exposures. It's got these large safety factors involved, by taking the most sensitive test, and not taking the average value, but taking the lower end of the confidence interval, and then dividing it further by 10. So it has a lot of safety built into it, which is exactly what you want to do when you are coming up with a safety factor, with a reference dose.

Tr. 4 at 810.

Dr. Brent believed that most of the adverse effects measured in the Faroe Island studies could be attributed to the PCBs that were quite present in the diet of that study's subjects, but not in that of the Seychelles subjects:

> [T]he Seychelles study basically is running in parallel with the Faroe Islands study. And they're doing the same thing, you know, periodically testing the children based on their exposures prenatally. And they have not been able to find any adverse effects with methyl mercury exposures. There's pretty much the same levels as the Faroe Islands. Now, the one difference is that the Seychelle Islands, they don't have the compounding factor of the PCBs exposure. I'm not clear whether that's the reason or not, but they have not been able to find any adverse effects. So we see here that even in this population, that is taking very large amounts of seafood, certainly compared to the United States, and putting much more methyl mercury in their brain than in the United States, that they're really not having any adverse effects.

Tr. 4 at 819.

Another point made by Dr. Brent is that the reference dose was based on chronic, average daily intake over a lifetime:

> So if you look at people in the Faroes, ... [t]hey are always up at that particular level. On the other hand, in the U.S. as I said, we're almost always under 10. Very often we're around two or three, particularly in children, sometimes even less than that.... But they're almost always well under 10. And then goes quickly back down. Versus what happens in the Faroe Islands, where, unrelated to vaccination, these people are always up at a much higher level. So the Faroe Islands is more of sort of a chronic situation, where they're always up there. And that's what they based the reference dose on. Not, you know, these little blips, like you see with a vaccination. But the reference dose is really based on Faroe population, which is chronically running very high.

Tr. 4 at 812-13. To Dr. Brent, this means that it is incorrect to say that thimerosal-containing vaccines would expose children to mercury in a dose that "exceeds the safe level," to the point of potential adverse outcome, as Dr. Lucier had intoned:

> [What] he called a safe level, simply refers to the reference dose. It [does not mean] that if you exceed that level, you necessarily are going to have an adverse effect, because of the large difference between the reference dose and the data that shows where you'd have an adverse effect. And it's also, remember, it doesn't take into consideration that on a day-to-day basis, sometimes you'll have very little exposure.
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> ...[T]here are many, many people, particularly fish-eating populations, who are well above the 5.8 level. We saw in the Faroes, they live at about 22. Nobody has ever shown an adverse effect clinically, from the people living there.[113]

Tr. 4 at 813-14. This reasoning also undermined Dr. Lucier's claim that 60,000 children per year are born "at risk" due to maternal exposure to methyl mercury over the level of the reference dose. Tr. 4 at 816-17. More importantly to the issue at bar, Dr. Brent stated that there was "absolutely no data whatsoever that that kind of exposure [*i.e.*, exceeding the reference dose] would cause cardiac arrest" or cause a higher risk for it. Tr. 4 at 817. He discussed the paucity of evidence for any such increase in observed incidence or risk for adverse cardiac outcomes within the populations studied closely for mercury toxicity:

> The Seychelles population which, you know, which is being very well studied prospectively, there has not been a single report of any adverse cardiac effects, cardiac arrests in any of the children. The same thing in the Faroe population, with a much higher exposure. There has not been, just like the Seychelles, there has not been any reports of adverse cardiac events like cardiac arrests in the children.

Tr. 4 at 820.

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[113] Dr. Brent did qualify this statement as follows:

> Although clearly when you get to the higher cord blood levels, they, you know, and you test the children, they do find that you can find, in the Faroes, they do find some detriment in performance.

Tr. 4 at 814.

At one point, the Court raised a point made by Dr. Lucier to explain the difference in results between the Faroe Island study and the Seychelles study, the variability of intake schedule and amount consumed between the two:

> THE COURT: Did not Petitioner's experts discourse about the steady, consistent, chronic exposure in the Seychelles, versus the episodic exposure in the Faroes, because of the consumption of whale? Which I gather was staccato, was not as steady. And they made something about it, if I recall.
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> THE WITNESS: Yes, that's a very good point. It is true that in the Seychelles, it's a pretty much steady diet. In the Faroes, [] they get the whale periodically. But methyl mercury has a relatively long half-life in blood. So what happens is if you measure their blood levels of the methyl mercury in the Faroes, it doesn't go up with a whale feast, and then down and stay down, and then up with a whale feast, and then down. It stays pretty high all the time because of the long half-life of the methyl mercury in the blood.

Tr. 4 at 823.

There was one study in particular that Dr. Lucier had referred to for support of Petitioners' theory of ethyl-mercury related cardiac arrest, which Dr. Brent saw as an unreliable outlier that addressed a distinguishably different condition, myocardial infarction:

> [The Salonen study[114]] is a study that has been cited because it reports that there is an association between fish and mercury intake, and myocardial infarction, which is a heart attack. Now, it's important to note that a -- there has been no allegation, as best as I saw in this case, by anybody that Thomas Kolakowski had a myocardial infarction. So it's kind of a different end point. But that's what they did report in the Salonen study.
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> ...One of the things you can tell about a study in terms of how well it's regarded in the medical literature is how often people cite it.... And according to an Ovid database search, that Salonen study has not been cited once in the medical literature since it got published.

Tr. 4 at 821-22.

Dr. Brent believed that there existed no support in the medical literature for the proposition that the ethyl mercury in thimerosal-containing vaccines could cause death from an adverse cardiac outcome:

> [T]here is, in terms of anything I can find, no credible scientific support for the proposition that thimerosal exposure from vaccines can cause death. I know this literature, I think, quite well. I have looked specifically for anything that would support that position, and I could not come up with a single paper, with a single allegation, with a single suggestion anyplace in the medical literature to suggest that

[114] Neither Dr. Brent nor Respondent indicated where in the record such an article was to be found.

> toxicity from thimerosal in vaccines, in the doses you get from vaccines, is likely to cause adverse cardiac effects, cardiac arrest, death.
>
> And as a matter of fact, I went through all the studies that were cited and talked about by the Petitioners to see if they had anything that made that allegation. And none of it, none of the studies did. What they did is they relied on a couple of publications, primarily the Dahhan publication, which is very, very high exposure; the Cinca publication, which is very, very high exposure; and the Grandjean paper, which actually doesn't really refer to cardiac toxicity, and is a study from the Faroe Islands.

Tr. 4 at 822-23.

Dr. Brent went into greater detail concerning the Dahhan article,[115] which "dealt with one of several Iraqi episodes of poisoning by fungicides that were put on seed grain," a fungicide "generally referred to as EMPTS" (ethyl mercury paratoluene sulfanilate). Tr. 4 at 824. First he noted that, "EMPTS is different from ethyl mercury. It has some similarities to it, obviously ... the differences between EMPTS and ethyl mercury have never been systemically studied." *Id*. The Dahhan study studied the electrocardiograms of 42 people with poisonous levels of mercury toxicity (some of whom died). *Id*. Dr. Brent described the results and the conclusions that can be drawn from them:

> [I]f you look at the effects, they were rather varied and inconsistent. And they were not very specific. Anybody who takes care of a lot of very acutely ill patients, and these patients were certainly potentially acutely ill, knows that as you keep them on cardiac monitors during their illness, their EKGs are going to show a lot of non-specific changes. They're going to show a lot of changes in the so-called QT interval and the ST segment, and so on. And there was no real specificity of these changes. There was nothing really specific about them that said ah-ha, this is a pattern you only see in mercury poisoning. Rather, what it seemed to Dahhan is sort of very non-specific changes that people see all the time, if you were to, for example, walk into an intensive care unit at any hospital and look at the cardiac monitors. And it's unlikely that they actually represent the toxic effect of mercury on the heart.

Tr. 4 at 824-25. Dr. Brent stated that the same reasoning explains the results of a similar studies by Jalili,[116] a paper filed by Respondent, and Zhang,[117] a paper filed by Petitioner:

> Jalili explained exactly the same thing. He, in fact I believe it was Jalili who actually pointed out that most of the EKG changes were actually due to low potassium levels. It's classical, you see in acutely ill patients, which causes these kinds of EKG changes.

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[115] Pet. Ex. 18, Tab U, Shawkat S. Dahhan and Hussain Orfaly, *Electrocardiographic Changes in Mercury Poisoning*, 14 THE AMERICAN JOURNAL OF CARDIOLOGY 178-183 (August 1964).

[116] Resp. Ex. A, Tab 27, M. A. Jalili and A. H. Abbasi, *Poisoning by Ethyl Mercury Toluene Sulphonanilide*, 18 BRITISH JOURNAL OF INDUSTRIAL MEDICINE 303-308 (1961).

[117] Jimel Zhang, *Clinical Observations in Ethyl Mercury Chloride Poisoning*, 5 AMERICAN JOURNAL OF INDUSTRIAL MEDICINE 251-58 (1984).

Q And this would also then apply to the cardiac findings in the Zhang paper...?

A That's correct.

Tr. 4 at 826.

Also on the subject of the Dahhan paper, Dr. Brent distinguished the very limited dose of ethyl mercury in thimerosal-containing vaccines from the nearly-lethal or lethal doses of mercury consumed by the Dahhan study's subjects:

> I think here is a classic example of why, where dose is such an important distinction here. The doses in the EMPTS episode in Iraq were huge, and bear really no relationship, I mean, cannot be generalized to the tiny doses you get with a vaccine. EMPTS was 7.3 percent of the fungicide substance that was used in Iraq. The ethyl mercury and the two doses of the hepatitis vaccine that Thomas Kolakowski got, as we were saying before, contained 0.005 percent of ethyl mercury. So it was massively different. In fact, if you look at the estimates in the Dahhan paper, they believe that, they point out that about 35 milligrams of EMPTS per kilogram was necessary to cause a lethal effect. Thirty-five milligrams per kilogram. ...[T]here are 1,000 micrograms in a milligram. So that's 35,000 micrograms per kilogram of EMPTS, which, if you just look at the mercury component, works out to 15,000 micrograms per kilogram. Now, Thomas Kolakowski received in his two vaccines 5.3 micrograms per kilogram. There is a massive, massive difference in dose between the two. So clearly you can't generalize the Dahhan paper to what happened to Thomas Kolakowski.

Tr. 4 at 826-27. Dr. Brent added on this last statement that he could not see any relevance at all in relating the Dahhan paper to the issue at bar. Tr. 4 at 827.

Next, Dr. Brent addressed the Cinca article,[118] relied upon by Dr. Lucier:

> [I]n the three cases in which they measured the blood levels, they measured them in three out of the four individuals that were poisoned, the blood levels varied from 1,000 to 5,000 micrograms per liter. Once again, remember, in the United States we tend to be under about 10 micrograms per liter; the maximum you typically get from a vaccine is well under 10 micrograms per liter. So this is a massive, massive exposure, compared to anything that you could possibly get from a vaccine.

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[118] Pet. Ex. 18, Tab V, I. Cinca *et al.*, *Accidental ethyl mercury poisoning with nervous system, skeletal muscle, and myocardium injury*, 43 JOURNAL OF NEUROLOGY, NEUROSURGERY, AND PSYCHIATRY 143-49 (1979). Dr. Brent summarized the study thusly:

> The Cinca study is from Romania, and it reports four members of a family who [ate meat from a] hog [] accidentally poisoned with fungicide containing ethyl mercury chloride. And they, for reasons unbeknownst to me, ate the hog, and got themselves poisoned by ethyl mercury. Once again, this was an extremely high-dose exposure, as the data shows.

Tr. 4 at 828.

Tr. 4 at 828-29. The same study examined the mercury levels of various body tissues, to assess the level of toxicity in the tissue or organ:

> [H]ere you have the tissue levels in various tissues of mercury. Kidney, as you see, has the most, and kidney avidly will, is the organ that takes up most of the ethyl mercury. And what you can see, if you look all the way over on the right to the myocardium, that's the heart, you get relatively little into the heart compared to the other tissues.

Tr. 4 at 830. In sum, Dr. Brent did not find the study relevant to the issue at bar: "I see no way that this kind of massive, massive exposure to ethyl mercury could have any relationship to the kinds of doses you get in two hepatitis-B vaccines." *Id.*

The next article discussed by Dr. Brent was the Pfab[119] case study, about the individual who attempted suicide by ingesting a thimerosal solution. He compared the mercury levels of that individual to the mercury levels measured post-vaccination in infants:

> That solution contained five grams of thimerosal, five million micrograms -- five million micrograms. And as you see, his blood levels were also extremely high: 14,000 micrograms per liter. Now, there have been some studies where they have given vaccines to children, and assessed their blood level post-vaccination. The Stajich[120] study was one that looked specifically at [the] Hepatitis-B vaccine. And the children got about 12.5 micrograms of mercury in the form of thimerosal, just like Thomas Kolakowski got in his hepatitis-B vaccine. And as you can see, the peak blood level, what they measured, was on the average, in the term infants, of 2.2 micrograms per liter.
>
> There has also been a discussion of the Pichichero[121] paper from 2002, where they measured blood levels in children who got vaccines. The doses varied quite a bit, depending upon what vaccinations they were getting. It was up to 62.5 micrograms of mercury at a time. And as you can see, the blood levels in Pichichero were post-vaccination one to 1.6 micrograms per liter. Pichichero, by the way, published a second study. I don't know, I don't believe there's been any testimony on the second study; it just came out in 2008 in the Journal of Pediatrics, where once again he looked at a greater group, a bigger group of vaccinated children. They received up to 57.5 micrograms of mercury in the form of thimerosal at any particular vaccination. And the highest blood level obtained was a little under eight micrograms per liter.

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[119] Resp. Ex. A, Tab 42, Rudolph Pfab *et al.*, *Clinical Course of Severe Poisoning with Thiomersal*, 34 (4) CLINICAL TOXICOLOGY 453-460 (1996).

[120] Resp. Ex. A, Tab 47, Gregory V. Stajich *et al.*, *Iatrogenic exposure to mercury after hepatitis B vaccination in preterm infants*, 136 (5) THE JOURNAL OF PEDIATRICS 679-681 (2000).

[121] Resp. Ex. A, Tab 43, Michael E. Pichichero *et al.*, *Mercury concentrations and metabolism in infants receiving vaccines containing thiomersal: a descriptive study*, 360 THE LANCET 1737-1741 (2002).

> So if you compare Cinca, you know, with blood levels that are 1,000 to 5,000, or the other, or other cases of acute poisonings, like Pfab or what happened in Iraq, you see it's very, very, very different from what you get from vaccines. And Thomas Kolakowski received two doses of hepatitis-B, and so it would be very similar to what was seen in Stajich. Stajich just studied the newborn dose. But he probably would have developed blood mercury levels pretty similar to what Stajich's are in his term infants, because Thomas Kolakowski was a term infant.

Tr. 4 at 831-32. It was in the context of discussing these studies, and the orders of magnitude of difference in dosage between the poisoning studies and the amount of thimerosal in vaccines, that Dr. Brent responded to a point made by Dr. Shane–that "precise dosage levels don't apply in individuals with the toxic effects." Tr. 4 at 833, quoting Tr. 3 at 708. This statement makes no sense to Dr. Brent:

> How could Dr. Shane say that at .1 or .01 microgram you could actually have an adverse effect? I have no idea how he could say that. There certainly is not any reliable scientific data, or any scientific data of any kind, reliable or unreliable, that could support such a preposterous, frankly, assertion.

Tr. 4 at 833-34.

Next, Dr. Brent distinguished the measurement of heart rate variation in the Grandjean study from the studies of subjects who had sustained heavy doses of mercury:

> The 2004 Grandjean article ... was from the Faroe Islands study ... what they studied was something called heart rate variations.... They weren't actually studying the heart. Heart rate variation is not an effect on the heart, is not an effect of the heart. The heart is under great control of the nervous system, part of the nervous system called the autonomic nervous system.[122] Speeds it up, slows it down. And so what they were studying is the nervous system control of the heart. And they found that at children who had very high prenatal methyl mercury exposures in the Faroes, that they had decreased heart rate variability. Now, that's basically what they found. And it probably relates to an effect of the high levels of methyl mercury on the brain centers that control that. There have not been, once again, any reports of any adverse cardiac effects in the population of the Faroe Islands. These children, despite their very high blood mercury levels, prenatally and postnatally, are not having cardiac arrests, are not having sudden cardiac death, they're not having SIDS.

Tr. 4 at 834-35.

Also affecting this question is latency in the onset of adverse effect following an exposure to mercury. Dr. Brent used the latency interval to explain further that Thomas Kolakowski's death was not from mercury toxicity from vaccination:

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[122] The autonomic nervous system is "the portion of the nervous system concerned with regulation of the activity of cardiac muscle, smooth muscle, and glands; usually restricted to the two visceral efferent peripheral components, the sympathetic nervous system and the parasympathetic nervous system." DORLAND'S, *supra*, at 1841.

> There is [a latency period associated with mercury poisoning], primarily with the organic mercurials.... For example, in the Iraqi EMPTS episode, the latency was anything from nine to 100 days before any symptoms occurred.... [In a study of Iraqi mercury poisoning from grain with EMPTS,] there was a latent period of weeks to months after methyl mercury exposure before the development of poisoning symptoms....
>
> [In contrast,] Thomas Kolakowski had an unfortunate death, five days after his last vaccination.... I mean, even if you look back at his first vaccination, which is basically five weeks, even that is a fairly short latency.

Tr. 4 at 836. For this reason, Dr. Brent noted his disagreement with Dr. Shane's statement that a person "could die from mercury toxicity within 24 hours of exposure." Tr. 4 at 837, citing Tr. 3 at 698-99. Besides perhaps by inhaling mercury vapor, Dr. Brent did not think such an immediate toxic response would be possible, except in extremely high doses; he thought it entirely implausible that such could occur at a lower dose. Tr. 4 at 837. Moreover, Dr. Brent did not see any link between myocarditis and low-level mercury exposure from vaccination, inasmuch as myocarditis often accompanies "any acute severe illness," and is not specific to acute mercury toxicity, "[a]lthough certainly, somebody who's severely mercury-toxic can have a component of myocarditis." Tr. 4 at 837-38. Dr. Brent said the typical first (and at times only) manifestation of mercury toxicity is paresthesia (numbness) in the extremities caused by peripheral neuropathy, and that the Bakir study found the threshold dose[123] for such a sequela to be 25-40mg (25,000-40,000µg) of mercury. Tr. 4 at 838. That means that the first clinically manifest symptom recognized to be a sequela of mercury toxicity requires a dose, administered within a short span, of at least 25,000µg.

Dr. Brent stated that, in the Burbacher[124] study, methyl mercury levels were higher in the brain than were those of ethyl mercury following an equivalent dose[125] for each, which contradicted Dr. Lucier's negation of that proposition. Tr. 4 at 839-40, referring to Tr. 1 at 233-34. While conceding that following exposure to a given dose of ethyl mercury, the amount of metabolized inorganic mercury was slightly greater than it was following an equivalent dose of methyl mercury, Dr. Brent stated that "you get much less mercury in the brain after equivalent doses of ethyl mercury than you do from methyl mercury." Tr. 4 at 841-42. "So you get about roughly 107 at the end of the experiment for methyl mercury, versus maybe 15 for the ethyl mercury." Tr. 4 at 842. Dr. Brent

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[123] See *supra* at note 108.

[124] Pet. Ex. 18, Tab M, Thomas M. Burbacher *et al.*, *Comparison of Blood and Brain Mercury Levels in Infant Monkeys Exposed to Methylmercury or Vaccines Containing Thimerosal*, 113 (8) ENVIRONMENTAL HEALTH PERSPECTIVES 1015-21 (August 2005).

[125] Dr. Brent was quick to point out that the dosage given the primate subjects in the Burbacher study contained more thimerosal than would be contained within a vaccine dose:

> [T]hey had to push the dose up because they were concerned that if they used the actual doses that were used in the vaccine, they wouldn't be able to detect the thimerosal in the brain. It might be too low. So they pushed it up to this higher dose.

Tr. 4 at 840.

also contradicted Dr. Shane's claim that, at equivalent doses, ethyl mercury exposure is more toxic than methyl mercury, stating that the reason Dr. Shane could cite no medical literature source for this proposition is that none exists. Tr. 4 at 843.

Dr. Brent discussed the Magos study,[126] which he viewed as "probably the most relevant study that looked at that very question of which has more of an adverse effect at similar doses, ethyl mercury or methyl mercury." Tr. 4 at 843. Its purpose was "to compare total and inorganic mercury concentrations in selected tissues, including the brain, after the daily administration of methyl mercury and ethyl mercury, and to relate these findings to damage in the brain and the kidney." Tr. 4 at 843-44. The researchers concluded that "methyl mercury was more toxic than ethyl mercury at an equivalent dose," notwithstanding the fact that "ethyl mercury-treated rats had more inorganic mercury in their brain than methyl mercury ones ... because ethyl mercury becomes inorganic faster than methyl mercury does."[127] Tr. 4 at 844. And Dr. Brent contradicted Dr. Connor's statement that the Magos study followed an aberrant method to extract mercury for measurement, stating, "He got that totally backwards. The Magos method for mercury analysis is the well-accepted method used today all over the world."[128] Tr. 4 at 845.

Next, Dr. Brent defended the (first) Pichichero study against Dr. Lucier's earlier criticism that the first measurements of blood mercury taken in the study, occurring three to five days post intramuscular injection of vaccine, were too late to capture peak blood levels. Tr. 4 at 849-50.

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[126] Pet. Ex. 18, Tab YY, L. Magos *et al.*, *The comparative toxicology of ethyl- and methylmercury*, 57 ARCHIVES OF TOXICOLOGY 260-267 (1985).

[127] Dr. Brent added:

> Now, I will say that ethyl mercury affects the kidneys a little bit more, because ethyl mercury possibly affects kidneys. But in terms of the brain, clearly methyl mercury caused more toxicity than ethyl mercury. There is no study that I know of in the scientific literature that shows the contrary.

Tr. 4 at 845.

[128] Dr. Brent went into greater detail on why Dr. Magos was the world expert on this point, and why Dr. Connor was wrong to gainsay his method:

> Magos is basically a leader in this area. What Magos did, he developed this original method for the extraction of methyl mercury. And when he did this study, and he was studying the comparative amounts of methyl and ethyl mercury when he measured the amounts in the brain, he wanted to be sure that the technique for methyl mercury would also work well for ethyl mercury, because it's a different molecule.
>
> And what he found is he had to increase the stannous chloride concentration to be sure that he got a full extraction of ethyl mercury, so it was a valid determination that he got all the ethyl mercury out. And that's what he did. He modified his technique so it would work, specifically so it would work well for ethyl mercury, as well as methyl mercury. So no, there was no flaw in the technique. That is the technique that is still, that is still used today. It was an enhancement of the technique, so it would work well in this experiment.

Tr. 4 at 845-46.

> [T]hat makes the assumption, which is incorrect, that the vaccine was given intravenously. If you administer the vaccine intravenously, you will get a peak level immediately at the time of administration. However, vaccinations are given intramuscularly, and they are therefore slow-release kinds of preparations. They slowly release from the muscle. So that it really takes a day or two before you get, before you start getting the peak levels from the vaccine. So you can't extrapolate back to the day of vaccination. Probably the peak levels are about two days later.
>
> And the other thing that Dr. Lucier failed to mention was that, in fact, the very issue about which he was developing this hypothetical model about the back-extrapolation to high levels, has been very well studied by these same investigators.[129]
>
> ...[I]t takes about a day or so before the levels actually peak, because of the fact that it's an intramuscular injection. And the highest, the highest blood level in any of these groups that was observed in the study was less, was a little less than eight micrograms per liter. So that tells us that that's about what you can expect from a vaccination; something under about 8 micrograms per liter is what you would typically expect. And then as you can see, the levels fall, and by about 10 days or so they get very close to baseline.

Tr. 4 at 850-52. Dr. Brent agreed that "the levels reported in Pichichero 2008 are approximately, are less than half the levels of the back calculations testified to by Dr. Lucier," and that "the blood-level measurements by Pichichero following the administration of thimerosal-containing vaccines" are reliable. Tr. 4 at 852-53.

> Next, Dr. Brent spent some time contextualizing laboratory cell series studies:
>
> It's important to remember that when you just have cells in a petri dish in the laboratory, that is very dramatically different from, a very dramatically different environment from what you would see in the cells in an individual. Our cells, our bodies are highly regulated, metabolically regulated. We have all kinds of protective mechanisms in our body for all kinds of insults. We have profound systems in our body to keep it working correctly, to keep the cells working correctly. And there's a marked interplay between all these different systems. That does not, that is not the case in a test tube. That is not the case in a petri dish.
>
> ...You can kill a cell *in vitro* with just about anything. If I just put it in an environment of water, it will die. Cells are very, very highly vulnerable to chemicals *in vitro*, because they don't have all the chemical protective mechanisms that we have in our body. So in general, when you put a chemical in the incubation media

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[129] Dr. Brent mentioned one of these:

> [T]hey got a sample at what they call time-point-zero, which was just immediately before the vaccination, and then they got subsequent time reports starting as early as 12 hours post-vaccination. So that data doesn't require a back-extrapolation on some hypothetical models on the early Pichichero paper; the data is right there, in published form, by these same investigators in their follow-up study.

Tr. 4 at 851-52.

> with cells, say, in a petri dish, those cells are going to be very, very sensitive to doses of those chemicals that will have, that will be totally without effect *in vivo*, in a live person or an animal.
>
> ...We have metallothionein, we have glutathione, we have cysteine, we have various proteins. All of these, all of these bind and detoxify heavy metals, mercury and other heavy metals. That doesn't exist in a cell-culture environment.
>
> ...[E]ven that mercury in the brain is 99.9 percent bound, highly bound by these molecules we were talking about: the glutathiones, the thials, the metallothioneins, all these metal-binding proteins and other substances in the brain that are there with the specific intention of detoxifying against all the heavy metals that we come in contact with. Because it's only when the metal is in the free form, not bound to something else, that it can interact with cells. And once it gets bound by something else, the cell is protected from it.

Tr. 4 at 857-59. Therefore, toxic effects observed in a petri dish cannot be directly extrapolated to represent the same result or risk in a live human being. Tr. 4 at 857. The IOM has released a statement on the effect of mercury on precisely that very point. Tr. 4 at 860. Nevertheless, Dr. Brent stipulated that these sort of mechanistic studies are very helpful in the right context:

> They are useful, if I know that there is some effect of a substance on a cell type in the body, and I want to study that mechanism of effect, then what I would do is I would take that cell type and try to study it *in vitro*, because you can do more sophisticated studies on the cells. So they're good for sort of mechanistic studies, but they do not have really a lot of validity in terms of toxicity studies.

Tr. 4 at 860.

This understanding about *in vitro* studies dovetailed into Dr. Brent's critique of Dr. Lucier's reliance on the "Waly study":

> The Waly paper really doesn't have anything to do with the heart. It really was designed to look at neurological effects. And they used, they took a bunch of cells that are *in vitro*, and there are these things that are called cell lines.
>
> ...And very often these cell lines, in order to keep growing, have to be tumor cell lines, because tumor cells grow in an unrestricted fashion. And so he used a tumor cell line of neuronal cells, so-called neuroblastoma[130] type of tumor of neuronal cells. And then he incubated them with thimerosal and a bunch of other things, as well, and they all had adverse effects on the cell ... and he was able to demonstrate adverse effects from rather small amounts of thimerosal on the cells. Now, what was interesting is that he could only make this happen if he did this in a copper-free medium.

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[130] A neuroblastoma is "a sarcoma consisting of malignant neuroblasts, usually arising in the autonomic nervous system (sympatheticoblastoma) or in the adrenal medulla; it is considered a type of neuroepithelial tumor and affects mostly infants and children up to 10 years of age." DORLAND'S, *supra*, at 1253.

> ...Our cells are highly dependent on copper. We have large amounts of copper in our cells. And in fact, for some reason, Waly could only demonstrate this in a copper-free medium. When he reconstituted the medium so that there was copper in the medium, the whole effect disappeared. So it was strictly an artifact of the fact that there was no copper in the medium. I mean, you wouldn't see this kind of effect in the body, because we have lots of copper in our bodies. I think that's a typical example about the kinds of limitations of *in vitro* studies. So what he showed, when he did this in the copper-free environment, is that there is an enzyme in these cells called methionine synthase.[131] And he was studying the fact that thimerosal and all these other molecules that he added to the cells seemed to inhibit this enzyme, methionine synthase.
>
> And he tried to relate that to neurological adverse effects. It was sort of an unfortunate cell line to choose, because, as Waly himself, and Deth, who was a senior collaborator, ultimately point out is that the methionine synthase in this cell culture, this tumor cell culture line, is abnormal; it's not a typical methionine synthase. So we don't even know if it applies to other cells.

Tr. 4 at 861-63.

The rest of Dr. Brent's direct testimony was spent refuting claims on the topic of toxicology made by Dr. Connor and Dr. Shane, Petitioners' cardiologist and pathologist, respectively. Whereas Dr. Shane cited to acetaminophen as a classical example in support of his contention that children have greater vulnerability to toxicity than do adults, Dr. Brent disagreed. Tr. 4 at 864-65. Dr. Brent pointed out in the most recent edition of a book by Randall Baselt, mentioned by Dr. Shane in making his point, that "no place in that section does it say anything about the fact that children are more sensitive to acetaminophen than adults." Tr. 4 at 865. He continued, "As a matter of fact, as a matter of fact, children are markedly less sensitive to acetaminophen toxicity than adults."[132] *Id*.

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[131] Methionine is "a naturally occurring essential amino acid furnishing both methyl groups and sulfur necessary for normal metabolism," and methionine synthase is "5-methyltetra-hydrofolate-homocysteine S-methyltransferase." DORLAND'S, *supra*, at 1140.

[132] Dr. Brent explained this statement:

> Acetaminophen at very high doses, classical dose-related thing. You know, at therapeutic doses it's a good drug. At very high doses, it can cause liver toxicity. And adults, in adults it's one of the most common causes of liver failure, very, very high doses of acetaminophen. Usually overdoses.
>
> ...[T]he most common reason for admission, emergency admission to my service is an acetaminophen overdose. There is not a single case in the medical literature of a child who has taken a single overdose of acetaminophen -- and they do it, because they're toddlers, and they, you know, they find things and they have a lot of hand-to-mouth behavior, and sometimes they take a lot. There's not a single case ever in the medical literature of a toddler who has taken a single overdose of acetaminophen that have ever had any toxicity. So when Dr. Shane says that they are -- a great example of how small children are more sensitive, he's absolutely got it backwards. They're clearly less sensitive. And this reference that he cited says nothing at all about them being more sensitive. Now, if you look at another what I consider to be a well-regarded toxicology text, which is my book ... I didn't write the acetaminophen chapter. And here we have the chapter on acetaminophen. And

Another statement Dr. Shane had made was that Thomas Kolakowski manifested "signs of heart failure from mercury toxicity"–to wit: "GI gas, decreased appetite, a head cold, and a rash." Tr. 4 at 869. Dr. Brent countered, "These are the kinds of things that we see in children all the time. They're very non-specific." Tr. 4 at 870. Those symptoms may be present in the event of mercury toxicity, "[b]ut they wouldn't have anything to do with the mercury toxicity." *Id*. They "could be indicia for many, many things, but that would not include mercury toxicity." *Id*.

Dr. Connor had raised the specter of "an immunological reaction causing sudden cardiac arrest from a thimerosal-containing vaccine," but Dr. Brent responded that "nothing like that has ever been reported [or] discussed. I have no idea where he came up with that." Tr. 4 at 870. He explained why such a theory was so implausible:

> Thimerosal has never been shown to do anything like that. And in fact, when you think about it, think about the components of the vaccine. Components of a vaccine is designed to create an immunological reaction. If there was going to be a reaction, an abnormal reaction to anything, it would be to the major antigen in the vaccine, which is hepatitis-B antigen. I have no idea how he could have implicated the thimerosal. There's no evidence that this has ever happened in the history of thimerosal usage.

Tr. 4 at 871. On a slightly similar note, Dr. Brent conceded that one "can be allergic to thimerosal," but saw "no evidence that that was the case. And there's never been a thimerosal allergy causing cardiac arrest." Tr. 4 at 872.

Summating his direct testimony, Dr. Brent negated that "any scientifically valid evidence [existed] that thimerosal-containing vaccines can cause sudden cardiac arrest" or had "caused Thomas Kolakowski's death." Tr. 4 at 871-72.

On cross-examination from Petitioners, Dr. Brent stipulated that the amount of mercury in the environment is constant,[133] and does not decay, and that "if a person happens to be in an environment where there is more present, then they will have a higher exposure." Tr. 4 at 873-74. However, Dr. Brent disagreed that this meant "approximately eight percent of the population from the NHANES study already are at toxic levels of mercury;" that study indicated that such a figure represented the population who had exceeded the EPA reference dose. Tr. 4 at 874. He added that the reference dose was not even based on adult populations, but upon the most vulnerable

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> I draw your attention to this particular passage.
>
> "Children younger than six years appear to be more resistant to acetaminophen toxicity than adults and adolescents. Several large studies of young children have demonstrated that in this age group, a single injection of 200 milligrams per kilogram or less is highly unlikely to cause significant toxicity."
>
> And if you look at the sentence right above it, you can see that 150 milligrams per kilogram is the threshold for toxicity in adults. So they're clearly not more sensitive to acetaminophen.

Tr. 4 at 865-868.

[133] *Cf*. nuclear fission, fusion.

population–unborn children–even though it is the reference dose applied to all population groups as a precaution. Tr. 4 at 875.

Petitioners referenced the Stajich article to challenge Dr. Brent that prematurely-born children exceeded the EPA reference dose when they received their vaccinations; he replied:

> [F]or the peak after the vaccination, absolutely. But remember, the EPA reference dose does not talk about peak levels, it talks about average levels. And of course, the vaccine levels, as we saw, and we were looking at them, go up and come back down. So yes, on the day that they're vaccinated, or in two days or three days after they're vaccinated, they are, you know, roughly 7.4, a little higher, a little lower. But then it comes back down. There is nothing wrong with that. You would not certainly expect any adverse effect. And in fact, if you average it over time, they don't exceed the reference level.

Tr. 4 at 878-79. Petitioners challenged Dr. Brent on that explanation in the following exchange:

> Q Well, Doctor, if you take your example that way, you could actually have a toxic dose one day, but if you average it out over a lifetime, you're fine. Isn't that --
>
> A Well, well. But that I would say, yeah, I think that's a valid point, Ms. Chin-Caplan. But you have to realize that that's taking it to a bit of an absurd extreme. That's just not the way the reference dose was conceived of. They weren't thinking in terms of what happens if you're chugging along under the reference dose, and then all of a sudden you have this massive exposure: you fall into a vat of mercury, and then they pull you out, and your levels ultimately come back down. I mean sure, you can get mercury-toxic from that exposure. That's not the kind of thing that the reference dose is obviously intended to address.
>
> ...
>
> Q So at the time that these infants in Pichichero 2008 received their immunizations, they were already above the reference dose for EPA for at least that period of time, is that true?
>
> A Well, that's an incorrect application of the reference dose. But most of them were not. Most of them were not, if you look at them. The highest one was less than, it was a little less than eight. It might be one or two that were over 5.8. But that's not the way you apply a reference dose.

Tr. 4 at 886-87.

Following that discussion, Petitioners mentioned certain rat studies wherein infant rats retained mercury in their bloodstream without excreting it, apparently to argue for extrapolation to human children. Tr. 4 at 887-892. Dr. Brent responded to this line of questioning as follows:

> This is all based on the fact that the rat biliary system is very, very immature compared to the human biliary system at birth. And at birth, humans have a very well-functioning biliary system; and as a matter of fact, it has been well

> demonstrated, by Pichichero and by others, that there is a very active excretion through the biliary system of mercury from vaccines.

Tr. 4 at 892.

Another point of contention arose between Petitioners and Dr. Brent concerning his negation of the dose-independent "susceptibility factors" listed by Dr. Lucier; Dr. Brent characterized it as "a construct that Dr. Lucier came up with." Tr. 4 at 905. As to whether age is a susceptibility factor, Dr. Brent stated that "[p]renatal versus postnatal is a major susceptibility factor," but he did not think sex was "particularly a major one." Tr. 4 at 906. He added that no "genetic difference calculation" had ever been determined. *Id*. He did not believe that certain people "are more susceptible to the effects of mercury than others." *Id*. He stated that the population distribution for "differences in clearance rate" fit a bell curve distribution. *Id*. He did not believe that existing high body burden of mercury affected how susceptible a person would be to a certain dosage of mercury relative to someone who had a lower body burden. Tr. 4 at 906-07. He also did not think that a neurotoxin had been identified that, if present in the body, would make a person more susceptible to mercury toxicity, in the event of a concurrent mercury exposure. Tr. 4 at 907. At this, Petitioners mentioned PCBs, but Dr. Brent stated, "we don't know if they influence each other, or if they're just simply added to the effect. PCBs don't necessary worsen the mercury effect, they just add to it." *Id*. Lastly, he stated that any attempt to link diet as a susceptibility factor is completely speculative, and no scientific testing has been performed to isolate this possibility. Tr. 4 at 909.

Dr. Brent conceded that, "in sufficient doses," mercuric mercury (Hg_2) can damage the brain, but made very clear that the location and extent of the damages was dose-specific:

> [W]e're talking about specific doses. And it can harm different ... types of cells. And a lot of the damage that occurs when you get to very high doses tends to affect specific parts of the brain, mostly in the back of the brain, called the cerebellum, the calperene cortex, and specifically the granular layer of the cerebellum.
>
> ...That's the most vulnerable area. Now, as you get to higher and higher doses, I mean when you're talking about massive, massive, massive exposure, of course you can get spillover damage into other areas.

Tr. 4 at 913-14. He added that (inorganic) mercuric mercury does not cross the blood-brain barrier once it is in that inorganic form, "[o]r, if it does, it does very, very minimally." Tr. 4 at 914.

Later, Petitioners challenged Dr. Brent with one[134] of the Charleston studies filed by Respondent, which monkeys were chronically exposed to methyl mercury, but did not exhibit symptoms:

> [I]n fact, if you look, do you know what that exposure was? It was 50 micrograms per kilogram per day, which is ... a massive exposure. I think it was 50, I might have

[134] Resp. Ex. A, Tab 15, Jay S. Charleston *et al.*, *Changes in the Number of Astrocytes and Microglia in the Thalamus of the Monkey* Macaca fascicularis *Following Long-Term Subclinical Methylmercury Exposure*, 17 (1) NEUROTOXICOLOGY 127-38 (1996).

> that number wrong if you show me the article. I remember it was a very large -- it might have been 500 micrograms per kilogram per day, .5 milligrams per day. Massive exposure. The blood levels, the blood levels in those monkeys, Ms. Chin-Caplan, were approximately 1,000 micrograms per liter. A thousand micrograms per liter. Now, the monkeys themselves showed no clinical adverse effects, but this was a big, big exposure.

Tr. 4 at 915-16. The article's author, said Dr. Brent, concluded that it was only "at those kinds of very high doses, which are huge doses, that you can have adverse effects on brain function." Tr. 4 at 916. Dr. Brent conceded that, "at massive doses," inorganic mercury exposure resulted in microglia[135] activation, but thought the study's results proved that, most likely, "that's probably why the animals were clinically fine, because the microglia were activated as a protective mechanism." Tr. 4 at 917. Dr. Brent stipulated that, indeed, at a high enough exposure, "that compensatory mechanism [could] be overwhelmed," but added that the same could be said "no matter what the chemical substance is," it might "push a person or an animal to the point where you've overloaded their compensatory mechanisms." *Id*. However, he said, "That's fundamental dose-dependent toxicology." *Id*.

Later still, the questioning moved into the contrast between the Seychelles and Faroe Islands data, and the relative utility of the studies in drawing scientific conclusions. Petitioners challenged Dr. Brent with Dr. Lucier's reasoning that "when you have two well-done studies, one which has a positive effect and one which has a negative effect, that you should, prudence would dictate that you follow what's recommended, follow the results of the positive study." Tr. 4 at 931. Dr. Brent qualified that such an approach may be bureaucratically preferable, but that it was not a scientifically valid approach to the question now facing the Court here: the question of actual causation:

> I think what you have to do is, first of all, do everything you can to try to see if you can tell where the difference is, and the major difference between the two is the PCB exposures in the Faroe Island study. And secondly, ... [if] I was studying a new antibiotic, and I did two studies; and one was a positive study that the antibiotic works great to treat pneumonia, and one was a negative study, the antibiotic didn't work to treat pneumonia, I wouldn't say well, prudence dictates that I accept the positive study, and ignore the fact that one of my studies showed it didn't work ... and therefore assume that it does work, and give this antibiotic to people. I would reach just the opposite conclusion. I would say no. You know, at this point we have two conflicting studies; we don't know what the true answer is, but we can't reach conclusions about it.

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[135] Dr. Brent had previously described microglia thusly:

> Microglia are cells that kind of act as what we call phagocytes. They are cells that if there are bacteria in the brain, or debris, or something that shouldn't be there, they kind of gobble it up and digest it, and get rid of it.

Tr. 4 at 915.

> Now, if I were in Dr. Lucier's position, and I were a regulator, then regulators, remember, always take the most conservative position. That's their job. That's what they're supposed to do. I'm not criticizing for that. And so if you have one positive study and one negative study, then what you have to do is you make the assumption that what was in the positive study may be true. And you build your regulatory guidelines around that. For example, the EPA, when it came up with its reference dose, had to use Faroes. They couldn't use Seychelles, because there was no adverse effect in Seychelles. So there was no effect from which you can say here is the level where we see an abnormal effect, and then we put in our uncertainty factors.... I mean, that's what you do when you're a regulator. But that doesn't, that's different than saying you're trying to determine scientific truth.
>
> ...And you cannot make assumptions by saying well, we have a study that shows an effect, and a study that doesn't show effect; so I'm going to say scientific truth is there's an effect... I would fail terribly in my job as a scientist if I took that position.

Tr. 4 at 931-34.

From there, Petitioners' cross-examination of Dr. Brent moved to their theory of a causative link between ethyl mercury exposure and adverse cardiac effects, and his stated opinion that cardiac irregularity in those people was a non-specific symptom of failing health due to acute, severe toxicity, and nothing specific to mercury in particular:

> Q ...[T]he document by the Agency for Toxic Substances and Disease Registry, page 106 has cardiovascular effects of inorganic mercury ... [and] they talk about a 22-year-old who attempted suicide by ingesting approximately 20 milligrams of mercury per kilogram as mercuric chloride. And he indicated that an EKG showed no P-wave, prolongation of the QRS, and a high T-wave. That's a cardiac effect, isn't it?
>
> A Right. And I'm sure that was a cardiac effect of the fact that he was acutely ill. Yes.
>
> ...
>
> Q What is it about the acute illness that would lead to cardiac side effects?
>
> A Oh, it's just a characteristic. If you look, for example, at Dahhan, and you look, for example, at Jalili, you look at those EKG changes, they're very non-specific changes. And I note every day when I'm on service, which is now it's about eight days a month, nine days a month, if I walk into the ICU and look at the rhythm strips, you see these same changes on every acutely ill patient. They're very non-specific changes. STO abnormalities, QRS abnormalities, prolonged QT interval. I mean, that's just, that's just something that you typically see in acutely ill patients.
>
> Q Well, it says no P-wave... And if they see no P-wave, isn't that an indication there's no atrial contraction?
>
> A No, there could be atrial contraction. It shows there's no, it's not AV node-stimulated. For example, if you look in patients that have atrial fibrillation,

they're having contractions, but there are just lots of them and miscoordinated in the atrium. And you won't see any P-waves because they're not, the atrial beats are not beginning at the SA node.

Q What about prolonged QTs?

A Totally non-specific finding. I would say if you take my ICU patients, 50 to 60 percent of them have prolonged QT intervals.

Q Any dangers associated with prolonged QTs?

A Depends on how long.

Q And when you say it depends on how long, what's the danger with the length of time?

A Well, the QT interval refers to the relaxation of the ventricles, the depolarization -- the repolarization of the ventricles, causing relaxation. And if you have a prolonged QT interval, which now, a typical QT interval is about 400, 450 milliseconds. If it gets above about 550 or 600 milliseconds, you are at risk for a type of cardiac arrhythmia that we call *torsades de pointes*.[136] And so we don't like it to get that long, because it does put people at risk. But, you know, you do see, you do see elevations into the high 400s and 500s all the time. I even see them on outpatient EKGs quite a bit.

Q So the dangers of prolonged QTs are sudden cardiac arrest?

A Only if it is, only if it is very prolonged, and it's a *torsades de pointes* type of arrest.

Tr. 4 at 939-41. When queried, Dr. Brent pointed out a passage within another study raised in challenge by Petitioners: "There are no demonstrable cardiac changes in mild cases. Moderate and severe cases had tachycardia, occasional arrhythmias, and two cases had a moderate rise in blood pressure." Tr. 4 at 943. Petitioners also raised the Jalili article's mention of "cardiac involvement" regarding cases where individuals expressed an irregular pulse or EKG changes ("[f]requent ventricular ectopic beats," "prolonged QT interval," "depression of the ST segment," "and T-wave conversion"). Tr. 4 at 945.

Petitioners next raised the Dahhan article for comparison with the preceding studies:

Q And Doctor, when you look at this article, are the cardiac abnormalities noted here any different from the earlier articles which we looked at?

A Exactly the same pattern, a very non-specific pattern.

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[136] *Torsades de pointes* (from the French for "fringe of pointed tips") is "an atypical rapid ventricular tachycardia with periodic waxing and waning of amplitude of the QRS complexes on the electrocardiogram as well as rotation of the complexes about the isoelectric line; it may be self-limited or may progress to ventricular fibrillation. DORLAND'S, *supra*, at 1924.

> Q Okay. And Doctor, when you look at page 183 of this article, does it say that all fatalities from mercury poisoning show a prolonged QT interval? In the EKG.
>
> A Prolongation of the QT interval, all fatalities in the mercury -- yes.
>
> Q So in this author's mind, a prolonged QT interval was a problem.
>
> A Well, he didn't say the prolonged QT interval caused it. That was what he observed, that in the fatal cases the QT interval was prolonged, which is not surprising. You know, the sicker you are, the more likely you are to prolonged QT intervals.

Tr. 4 at 946. Shortly thereafter, Dr. Brent distanced the EKG effects from the ethyl mercury as a cause:

> I think it was Zhang who actually pointed out that most of the EKG changes were actually related to hypokalemia, to a low potassium level, which is very common in very sick patients. And he describes -- you asked me what the EKG changes were? Was that your question? Yeah, they're described up on the top of page 253. It said seven patients showed EKG abnormalities, also prolonged QT interval, five showing depression of the ST segment, flat or inverted T waves and large U waves, consistent with low serum potassium levels. After potassium supplementation, most values returned to normal or nearly normal levels.

Tr. 4 at 947.

When Petitioners suggested that a subject in the Cinca study, who had been mercury poisoned, had "died from cardiac arrest," it led to the following interchange:

> A Well, that's not -- let me point something out. You cannot, it's a meaningless statement to say that somebody dies from cardiac arrest, because everybody dies from cardiac arrest. That's usually how you define death. No matter what's happened to you, when your heart stops is when you define death. So for example, you cannot list on the death certificate cardiac arrest as the cause of death. That is not considered to be a valid cause of death. Everybody dies of cardiac arrest. So the question is, you know, what caused the cardiac arrest.
>
> Q Okay. So Doctor, what caused the cardiac arrest in these two children?
>
> A They were mercury-toxic.
>
> Q And in these children, they had EKG abnormalities?
>
> A Oh, I'm sure they did. With blood levels from 1,000 to 5,000 micrograms per liter, you're going to be pretty darn sick. And when you're that sick, you're going to have EKG changes, no question.
>
> ...And I think it's probably fair to say that anybody with a blood mercury level of 1,000 to 5,000 micrograms per liter, who ends up this sick and in a hospital, is going to probably have some non-specific EKG changes. Absolutely.

Tr. 4 at 951-953. Moreover, although the Cinca study's author(s) stated "that ethyl mercury poisoning seems to [cause] cardiac alterations more than methyl mercury poisoning," Dr. Brent "went back through all their references to try to verify where that came from, and [] could not verify that ... there is nothing in the literature that actually supports that statement." Tr. 4 at 954-55. He compared the Cinca study to the others discussed, saying, "[I]n all these cases, where there is a very massive exposure to mercury, to the point that it makes you very ill, you will definitely see some non-specific changes on the EKG. And that's true of any very ill patient, for any reason." Tr. 4 at 954.

Discussing yet another article shortly thereafter, when asked to discuss "the [cardiologic] significance of a prolonged, a prolonged evoked potential latency," Dr. Brent replied that there was none. Tr. 4 at 957-58. Also, addressing the observation of decreased R-to-R variation that occurs in patients with high prenatal exposure to methyl mercury, Dr. Brent stated that "it does show that the methyl mercury, at those very high levels, is having some effect on the brainstem ... it's a higher level than people should normally be exposed to. But ... you don't see any [actual clinical manifestations]. Tr. 4 at 962-63.

There was during cross-examination, yet another discussion of the Burbacher article:

Q Does ethyl mercury get into the brain faster?

A Ethyl mercury gets into the brain slightly faster, but accumulates in the brain much less. It goes out faster.

Q Okay. Now, you're talking about the organic species, correct?

A Correct.

Q Now, the inorganic mercury, the Hg_2 that we discussed earlier.

A Right.

Q In Hg_2 from ethyl mercury, was there a higher level in the brain from ethyl mercury than there was from methyl mercury?

A ... So if you look at the inorganic mercury for thimerosal, at the end of that time period, it's a little over 10. It's a little over 10, maybe 12, 13, 14 parts per billion. And it stays pretty constantly at that. And that's about the amount of inorganic mercury. If you look at the methyl mercury, it's a little lower. The inorganic mercury is a little lower. It's about maybe seven, and that's about where it is. Now, what's important to notice is, however, at the end of the experimental period all of the, if you go back to the thimerosal on the right, all of the ethyl mercury is just about gone. There's a little bit left there, maybe two parts, maybe two to three parts per billion. It's just about gone. If you look at the methyl mercury, there's still over 100, or let's say 100, parts per billion left there. So the total mercury for thimerosal is about 12, 13; total mercury for methyl mercury is about 107, maybe. And what's going to happen is, Ms. Chin-Caplan, is that the methyl mercury gets the, get demethylated a lot slower than the ethyl mercury gets deethylated. So over time what happens is all the methyl mercury will eventually become inorganic mercury.

And so in the long run, not only will you have more total mercury, as you have here at the end of the experiment, in the long run you'll end up with much more inorganic mercury from the methyl mercury.

Tr. 4 at 968-970.

Finally, Dr. Brent clarified and honed certain aspects of his opinion on redirect:

Q Dr. Brent, there was a lot of discussion on your cross-examination about the Seychelles and the Faroes, and whether fish is beneficial for neurological outcomes, or whether the Faroes showed a subtle neurological deficit. Although interesting, do either of those studies, are they relevant to what the question is today? Does it cause cardiac arrest in children, or sudden cardiac arrest in infants? Do any of those studies cover that as an outcome?

A No, no. They weren't looking at that. They were totally silent with regard to the issue of cardiac arrest or anything like that.

...And they have never, despite the very high mercury exposures, they have never reported that there's any increased incidence of cardiac arrest or sudden childhood death or anything like that.

...Q And Doctor, I know you testified on your direct, and probably on your cross again, what is the relevance of the Zhang, the Jalili, the Cinca, and Dahhan articles regarding the EKG abnormalities in this specific case?

A Yeah. You can't – those are massive exposures. Those are poisonings, mass poisonings.... And they've got no relationship whatsoever to the very tiny exposures that you see from the vaccines. And the EKG abnormalities were really, you know, they were the abnormalities of sick people. These were very sick people. They were mercury-poisoned people; they were very ill. And you do see these kinds of EKG abnormalities, it's just one of those things.

Q And do those EKGs have any relevance to, specifically to the findings for Thomas Kolakowski?

A No, absolutely not. You know, the doses in those were so huge that they didn't, that they were nothing at all like what Thomas Kolakowski got. Thomas Kolakowski got doses that are similar to what all children get, and there's never been a report of any allegation of cardiac arrest from the doses you get from vaccines. And in fact, if you look at Thomas Kolakowski's rhythm strips in the emergency department when they were attempting to resuscitate him, they were just, you know, even the Petitioner's cardiologist said they were just completely non-specific and you couldn't draw any conclusions from them.

Q And finally, you testified earlier that there is no reference dose for ethyl mercury, is that correct?

A There is no reference dose for ethyl mercury.

> Q And when we talk about exceeding the reference dose through thimerosal-containing vaccines, we're referring to the reference dose for methyl mercury, is that correct?
>
> A That is absolutely correct.
>
> Q And in your opinion, is there any applicability of the reference dose for methyl mercury to ethyl mercury?
>
> A No, no. If anybody was really concerned, they could do their own analysis for ethyl mercury and come up with a reference dose. But since most people's exposures to ethyl mercury are so tiny, I don't think anybody is ever going to bother to do that.

Tr. 4 at 979-82. His concluding remarks came at the prompting of the Court:

> I would say only that, you know, there has been a lot of discussion here today about a lot of issues that have taken us, I think, very, very far afield from the question of whether thimerosal in a vaccine can induce cardiac arrest in a child. And that, you know, we've covered all this ground. We've talked about all this stuff, and we've sort of gotten away from the fundamental question. And based on that fundamental question, though -- if we get away from the brain stuff and all the other stuff -- I think it just simply comes down to a question of dose, and the tiny dose that's associated in vaccines. And there's just absolutely no scientific support for the idea that it can cause cardiac arrest.

Tr. 4 at 989.

5. Richard Ringel, M.D.

Dr. Ringel is a pediatric cardiologist. Transcript of Proceedings convened on 25 June 2008 (Tr. 5) at 997. He attended SUNY Stonybrook, and then Albert Einstein College of Medicine in New York for medical school. Tr. 5 at 998. He completed his pediatric internship and residency, as well as his pediatric cardiology fellowship, at the University of Maryland in Baltimore. Tr. 5 at 998. He started teaching there as well, moving from assistant professor to full professor over time. Tr. 5 at 998. He currently serves as Director of Pediatric Cardiac Catheterization and Adult Congenital Heart Disease Catheterization at Johns Hopkins University Hospital, pursuant to which he sees approximately 20 patients a week, as well as "attend[ing] general pediatric cardiology clinics and inpatient service, ... some teaching responsibilities, and administrative responsibilities for the division." Tr. 5 at 998-999. As part of his work, he had been interpreting EKGs for 25 years. Tr. 5 at 1002. Dr. Ringel is board-certified in pediatrics and pediatric cardiology. Tr. 5 at 998.

Dr. Ringel began his opinion testimony by corroborating generally Respondent's position that dosage is the most important factor in assessing toxicity:

> Q And when you prescribe medications, is the dose of the medication important?

A It's very important. In infants and children, the dose of medications has to be weight-based.... [I]f you don't choose the adequate dose, you can not give enough of a medication based on a child's weight, and the medication can be ineffective. And of course, if you then give too much of the medication based on the child's weight, then the medication can be toxic. So you need to fall within the range of therapeutic dosing.

Q So, Doctor, would you agree ... that when prescribing medications, they can be considered beneficial at one dose, but toxic or fatal at another dose?

A Yes, of course.

Tr. 5 at 1006.

Dr. Ringel summarized Thomas Kolakowski's course based mostly on the medical records:

Thomas was born as a full-term well child. The only thing that was recognized or noted was that he was large for his dates, or LGA, large for gestational age. His mother had concerns on the second day of life about jaundice. It was felt that the jaundice represented the fairly typical or ubiquitous neonatal jaundice, and there were recommendations given to expose to sunlight, and to keep well hydrated. There was an examination a few days later, confirming the diagnosis of mild jaundice. The weight was found to be eight pounds, six ounces, a good weight for the infant. And some additional supplement of formula was recommended to make sure that the infant remained well hydrated, one of the ways to clear jaundice from the system. There was some concern or mention of tremors. There is not any record of these tremors in the medical record. There is the mention of the jaundice by the pediatrician, but nothing about the tremors. By the next weight check on December 24, the weight had already increased to eight pounds, 13 ounces, which is really quite good for an infant.

There was an examination on January 2. Apparently he was found to be growing and gaining weight well. The examination was noted as normal, without any abnormalities identified by the pediatrician, except for a mild eye discharge. And the second hepatitis-B vaccine was given on January 20. There's nothing else in the medical record about that. My understanding is that four days later, on the 24th of January, I think I have that date right, Thomas awoke around 3:00 a.m. His mother brought him to the bed to feed him, and then Thomas stayed with Mother and Father in bed. And when she awoke at around 7:00, 7:15 in the morning, she found that Thomas was not breathing. There was some blood in the mouth, nose, I believe some on the sheets. His father attempted or performed CPR, and emergency medical services were contacted.

...

My recollection is that the emergency medical technicians arrived on the scene; they found Thomas to be, I think they used the word "lifeless," but of course I can read the record. They found no spontaneous activity. They proceeded with CPR, including

> intubation in the field. They administered the first doses of epinephrine via the intratracheal tube, and they did not obtain intravenous access, and brought him to the emergency room. I believe they did obtain an electrocardiogram so-called rhythm strip, single lead, which demonstrated asystole, or no cardiac electrical activity.

Tr. 5 at 1008-14.

Narrowing in on the cardiologic matters within this history, Dr. Ringel noted that, during his first few weeks of life, Thomas "sounded as if he was a normal, healthy newborn," and did not notice anything in the medical records showing "signs or symptoms of cardiac dysfunction after his first hepatitis-B vaccination." Tr. 5 at 1011. He also did not "find any information in the medical records that indicated that Thomas Kolakowski was suffering any signs of cardiac dysfunction between December 19, when he received his first vaccination, and January 20, when he received his second vaccination[, nor] between January 20, 1999, when he received his second hepatitis-B vaccination, and January 25, 1999," when he died. *Id*. Dr. Connor stated that there was no evidence in the medical records of congestive heart failure, and he disagreed with Dr. Shane's assessment that Thomas showed indications of congestive heart failure. Tr. 5 at 1013.

In looking at the emergency records from 25 January 1999, Dr. Connor reiterated the notations that Thomas was pulseless upon the EMTs' arrival, and showed some signs of lividity, and that the rhythm strips from the EKG they took indicated "asystole, or lack of any cardiac electrical activity." Tr. 5 at 1014-15. He described the EKG rhythm strips recorded soon after their arrival, but before Thomas arrived at the hospital:

> In this, in this lead, described as LEAD II, which is a standard single lead used in emergencies, there is no evidence of cardiac electrical activity. There is some baseline motion, if you will, of the tracing. We call that baseline noise, or artifact. You'll get that from any system where you hook up an electrocardiogram to something that has fluid or moisture. Also there is some, what someone might describe as rhythmic motions in the second of the three tracings. This is all one continuous tracing that's been cut up, but in the second line. But the EMTs make sure to note that it's with CPR; so CPR is rhythmic, and that's why the artifact may appear rhythmic.

Tr. 5 at 1015. Dr. Ringel did not corroborate Dr. Shane's finding of pulseless electrical activity on those EKG strips, believing that they demonstrated "total asystole," with "no cardiac electrical activity" at all. Tr. 5 at 1016.

Dr. Ringel recited the EMTs' clinical findings as "pulseless apneic modeling, lividity, blood from the mouth, flaccid extremities, dilated non-reactive pupils, no obvious trauma," with a EKG reading of "asystolic." Tr. 5 at 1017. He added, "Lividity suggests that there has been inadequate pumping of blood for a period of time, so that the blood that is in the vascular space has begun to settle out to the, what we call dependent regions," meaning "whatever is the lowest point

gravitationally."[137] Tr. 5 at 1017-18. He was not able to ascertain with any particularity or certainty when death would have occurred simply on the basis of a lividity finding, except that it would take longer than 15 minutes following death.[138] Tr. 5 at 1018-19. This diagnosis was described "pretty much the same" when Thomas arrived at the hospital: "Patient presents pulseless, intubated, with lividity noted. Asystole, CPR in progress." Tr. 5 at 1019-20. At the hospital, the asystole was confirmed by a three-lead EKG. Tr. 5 at 1020.

> Dr. Ringel described the EKG readings from the emergency room as follows:
>
> It's mostly noise. I do not see anything that looks like a standard electrocardiogram pattern. You can't pick out the so-called P waves, you can't pick out regular QRS or the ventricular electrical activation or repolarization.[139] It looks primarily like noise to me, perhaps related to ongoing CPR. We know that CPR was being given at this time.

Tr. 5 at 1021. Dr. Ringel could not delineate any of the wave segments of normal heart wave function from Thomas' rhythm strips at the emergency room:

---

[137] Dr. Ringel later elaborated regarding the cardiologic significance of a lividity finding in this case:

> [L]ividity occurs when there is not enough blood being pumped around to keep the blood moving through the circulation. And then once the blood stops flowing through the circulation, the blood settles out. And as it settles out, it goes to the lowest areas gravitationally, and creates this kind of bluish hue, because the blood is pooling in the veins.
>
> The skin gets like a spongy feel to it, and the upper areas tend to look kind of pale or grayish. So the lividity, the way it looks, and again, it represents the cessation of any blood flow.
>
> And I repeat, I'm uncomfortable in being able to say how long it takes for that to occur, but I also have to say I'm not sure I understand the significance of the lividity if a child was found dead in bed, and whether the actual moment of death occurred 10 minutes before Mrs. Kolakowski woke up and found him, or 30 minutes before she woke up and found him.

Tr. 5 at 1028-29.

[138] He stated the reason for his discomfort about setting a firm time frame as follows:

> I can't tell you how long it takes. I can tell you that I have seen children present to emergency rooms after being found by parents, and have demonstrated lividity. I can tell you that if we've had a child die in an ICU, and we wait for parents to come in or that sort of thing, they will start to begin to show lividity. But I can't pretend to say I know how long it takes.

Tr. 5 at 1018.

[139] Dr. Ringel described in greater detail the wave segments of an EKG:

> The electrocardiogram pattern consists of a number of wave forms.... Atrial activation, or the electrical activation of the upper chambers of the heart, the atria, are described on the electrocardiogram by the P wave. Then there is the QRS complex, [which] represents the activation of the ventricles, or the pumping chambers. You then have a brief period of isoelectric, or lack of electrical charging. And then you have the T wave, which represents the repolarization or the recharging of the muscle cells of the ventricle becoming ready for the next electrical cycle.

Tr. 5 at 1021-22.

> No. Again, you see something that someone might say is rhythmic. In other words, there are spikes that occur in fairly regular intervals. But they're not perfectly regular, like a heart rate. They also don't have the typical wave-form patterns. They have, especially in the second tracing from the top, you see a lot of very sharp spikes, which look more like 60-cycle or electrical interference, like a lead being loose and then causing this interference, particularly in the middle of the second row. If you point to the middle of the second row -- yes, thank you -- you can see in between each of those large downward deflections, there's this noise in between.
>
> So this is a noisy record. It could possibly represent a coarse form of ventricular fibrillation, but I think it's more likely noise. If you don't mind, I refer you down to the bottom two tracings. Now, this is a pattern that some people would look at and certainly say represents ventricular fibrillation. Can you distinguish it completely from noise? No, you can't. You have to be cognizant of the clinical situation. It's easy to create a pattern that looks like that just by tapping on an electrocardiogram or performing CPR, but if you just showed this to me, I would say this could be ventricular fibrillation.

Tr. 5 at 1022-23. A later EKG taken at the emergency room fit the profile of an agonal–or dying–heart rhythm:

> Here you see what is often described as an agonal electrocardiogram pattern. You don't have normally formed P waves, the QRS patterns are often strange or bizarre. And there may or may not be well-formed T waves, and the rate is usually very slow. And one can think of it as the final gasps of a dying myocardium giving off some intrinsic electrical activity as the heart muscle dies.
>
> ...Again you see a very slow rhythm. The heart rate, if you want to call it that, is in the twenties. You do see a QRS pattern. You do see, when the noise dies down in the background, you can see some vague T-wave patterns. Again, we would consider this to be, you know, an agonal EKG rhythm at a rate far below anything life-sustaining.[140]

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[140] Dr. Ringel described what he meant by "agonal" heart rhythm, and related it to what he saw on the hospital EKG strips in this case:

> As heart muscle is dying, there still remain[] some cells in the heart muscle, particularly the ventricles, that have the ability to depolarize, or discharge themselves, and repolarize, or charge themselves up again. Which is the process of how heart muscle becomes activated to contract. So not every bit of heart muscle dies simultaneously, so there can still be some muscle that has still retained some ability to electrically activate itself and deactivate itself.
>
> So that's what you see in an agonal rhythm, is the last remaining parts of the hearts dying. It is recognized that not all parts of the body die, if you will, at the same time. It's one of the tenets that leads to the possibility of harvesting organs for transplantation. So that even after a patient is dead, if you will, there are still useable organs, because they haven't completely "died." So there are still living cells there.
>
> So this is just a reflection that there are some small patches of myocardium that can give off some electrical signal. There's nothing else you can say or do about it, as far as interpreting the

Tr. 5 at 1024-25. Dr. Ringel opined that none of these EKG readings "would be indicative of a normal heart pattern." Tr. 5 at 1025.

Regarding when Thomas Kolakowski actually died, Dr. Ringel stated his opinion that "when the EMTs arrived, I have no doubt that he was dead," adding "Resuscitative efforts were unsuccessful, and then he was declared clinically dead, absolutely unresuscitatable and dead." Tr. 5 at 1025. Also, regarding the utility of Thomas Kolakowski's postmortem EKG readings in assessing the cause of his death, Dr. Ringel was dubious:

> Q What information can you use from Thomas Kolakowski's, or can you use any information from Thomas Kolakowski's EKGs to provide information about his condition following receipt of his first hepatitis-B vaccine?
>
> A No. These are essentially post-mortem electrocardiograms, and have no value in interpretation of them in comparison to the literature that Dr. Connor referred to. And I believe in Dr. Connor's testimony here, he also made that statement, that these electrocardiograms are irrelevant.
>
> Q So is it your opinion, then, that the EKGs provide no information about Thomas Kolakowski's condition immediately prior to his death?
>
> A They provide no information.
>
> Tr. 5 at 1029-30. The Court asked a clarifying question on this point:
>
> THE COURT: Doctor, ... was his cardiac arrest the cause of the process that led to his death? Or was it part of the agonal process, and the death, the cause of death was actually something else? If you can say.
>
> THE WITNESS: I see nothing in here that points to cardiac arrest as the, if you will, cause of death.
>
> THE COURT: Precipitating factor.
>
> THE WITNESS: Correct. ... everyone in their field recognize that the heart stopping tends to be the end process of many, many, many, many causes of death. So there is

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> electrocardiogram, because at this point in time the heart is already breaking, the heart muscle is breaking down. It has liberated all sorts of enzymes into the bloodstream and into the milieu of the heart muscle itself. If you check things like CPK levels or triponin levels, these are high; these are components of heart muscle cells that have begun to leak out from the leaky membranes or the dying membranes.
>
> The body tends to be filled with potassium by this point, because as heart muscles and regular muscles and skin tissues begin to die, they release potassium into the system. So the potassium levels become very elevated in a dying patient. Potassium levels wreak havoc on electrical signals. Acid buildup in the bloodstream, as the body is unable to discharge the acid any longer, that, too, wreaks havoc on electrical signals.
>
> So these are just signs of depolarization, but nothing interpretable when comparing it to normal electrocardiograms.

Tr. 5 at 1026-27.

> nothing here that suggests that this child had an underlying cardiac problem that would lead to cardiac arrest, which is a sudden cardiac death, which is what we prefer to call it; lead to a sudden cardiac death as the cause for the child's ultimate demise. But yes, ultimately the heart stopped. I don't like to use the term "cardiac arrest," because I think in most of our minds that suggests that the cause of death was cardiac.
>
> ...[T]he heart had stopped beating some time before the EMTs arrived. They found absolutely no signs of cardiac function. They were able to resuscitate some minimal cardiac function, and apparently only electrical function, as I have displayed, by doing the CPR and by giving epinephrine. But they were never able to, even in the emergency room, resuscitate enough of the heart to develop meaningful heart function.

Tr. 5 at 1031-32.

Dr. Ringel agreed with most of Dr. Connor's assessment of the EKG strips, but disagreed strongly with Dr. Shane's foray into EKG interpretation:

> This is, this is incorrect. Unlike Dr. Connor's notations, which are clearly marked and you could see what we call a QRS pattern and T wave, there is no cardiac electrical activity here. This is background, baseline electrical noise, with perhaps some rhythmicity caused by the ongoing CPR, which again is noted on the strips itself. There's no cardiac electrical activity here.

Tr. 5 at 1034. Elsewhere Dr. Ringel contradicted another of Dr. Shane's labeling of EKG strips, noting that where Dr. Shane saw heart activity, Dr. Ringel saw only "noise" and "movement artifact." Tr. 5 at 1036.

Next off, Dr. Ringel addressed the medical literature touching on cardiologic sequela to mercury toxicity and/or lethal poisoning. First, he distinguished the Dahhan[141] article's findings from the EKG strips of Thomas Kolakowski in the medical record:

> [A]t the time of the electrocardiograms, all of the patients were alive. None of them were in the process of dying, although a few of the patients did die subsequent to the obtaining of the electrocardiograms.

Tr. 5 at 1038. Accordingly, Dr. Ringel saw "no sign of correlation" to compare the Dahhan article to Thomas Kolakowski's case. Tr. 5 at 1039.

Moving on to the Cinca[142] study, Dr. Ringel summarized the article as "mention[ing] the post-mortem cardiac findings on ... two or three of the patients, that demonstrated a chronic

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[141] Pet. Ex. 18, Tab U, Shawkat S. Dahhan and Hussain Orfaly, *Electrocardiographic Changes in Mercury Poisoning*, 14 THE AMERICAN JOURNAL OF CARDIOLOGY 178-183 (August 1964).

[142] Pet. Ex. 18, Tab V, I. Cinca *et al.*, *Accidental ethyl mercury poisoning with nervous system, skeletal muscle, and myocardium injury*, 43 JOURNAL OF NEUROLOGY, NEUROSURGERY, AND PSYCHIATRY 143-49 (1979).

interstitial myocarditis." Tr. 5 at 1039. However, when asked to compare the Dahhan and Cinca studies to a child who received two Hepatitis B vaccinations in the first month of life, Dr. Ringel stated:

> Well, I think both papers are important descriptive papers of what happens when people get exposed to huge doses of organic mercury, and the findings that can be described, and the recovery of some patients and the demise of others. They do point to a number of interesting findings.
>
> But from my readings and from my review, as I would review any article having to do with patients I care for, I don't see any sign of correlation between these papers and the Kolakowski case. They are just what we often say apples and oranges. These are, I don't see any reason to use these papers to signify what happened to Thomas Kolakowski.

Tr. 5 at 1039-40. Additionally, Dr. Ringel did not find that any of the medical literature mentioned by Dr. Connor actually supported Dr. Connor's opinion that vaccines caused or precipitated the death of Thomas Kolakowski *via* mercury toxicity. Tr. 5 at 1041.

Dr. Ringel briefly addressed a claim made by Dr. Shane, that Thomas Kolakowski demonstrated symptoms of congestive heart failure. First, he delineated that the symptoms of congestive heart failure "are very different between adults and infants." Tr. 5 at 1040. For infants, symptoms "develop[] gradually, but all over days to weeks," including "increased respiratory effort, which either means rapid breathing and/or deep breathing." *Id*. He added, "Another sign that parents might recognize would be gradual decrease, or an inability of feeding or nursing," leading to "poor weight gain or no weight gain ... [b]ecause so much energy is being used in the process of breathing and supporting the circulation, and the children are having decreased intake over the course of many days or weeks, that there is no longer enough energy left for weight gain." Tr. 5 at 1041. Dr. Ringel noted that he did not "see anything in the medical records[143] that indicated that Thomas had any of those signs or symptoms." *Id*.

Dr. Ringel concluded his direct testimony by considering potential alternative causes for Thomas Kolakowski's death:

> Well, Thomas obviously died tragically and suddenly. The coroner used the term sudden death in an infant, or sudden infant death. I'm not going to, even though I used that in my description in my statement, because that's what had been mentioned by the coroner, I don't know that I would necessarily know that one would classify it into a classic sudden infant death syndrome. That tends to be done by pathologists. But what I can say is that there is an increased risk of infants dying suddenly when they share a bed with two adults, in the age group under 11 weeks, such that the American Academy of Pediatrics has recommended against bed-sharing in that age group. So the finding of an infant who had been previously well, being found dead in the bed with signs of blood from the mouth and nose, and the inability to

[143] *See supra* at 4.

> resuscitate, leads to the other possible conclusion that this was a child who did succumb to inadvertent suffocation related to bed-sharing.

Tr. 5 at 1042.

Petitioners began cross-examination of Dr. Ringel by asking him to compare the dosage of ethyl mercury in the two doses of Hepatitis B vaccine to the EPA reference dose for methyl mercury:

> Q And do you know how much mercury is contained within those hepatitis-B vaccines?
>
> A I believe those doses, and at that time it was 12 and a half micrograms ... [and] he weighed approximately four kilo[gram]s.
>
> Q Which represents roughly three micrograms per kilogram of body weight?
>
> A Yes.
>
> Q And that would be 30 times the recommended reference dose for EPA?

Tr. 5 at 1045-46. Dr. Ringel abstained from answering this question, as it related to toxicology, and not cardiology. Tr. 5 at 1046-1047.

Dr. Ringel did not dispute Dr. Connor's testimony, "that prolonged QT leaves an individual at risk for cardiac arrhythmias and cardiac arrest." Tr. 5 at 1050. However, he differed once again with Dr. Shane's assessment of EKG readings that focused on pulseless electrical activity (PEA):

> Some of the EKGs shown to me had no electrical activity. One or two may have shown, or one of the traces may have shown ventricular defibrillation; that's not considered PEA. And then there were the strips with the agonal rhythm. That is, I think, a step beyond pulseless electrical activity. That's basically the spasms of a dead heart. So, no.

Tr. 5 at 1051.

Dr. Ringel resisted further attempts to pin down an exact estimate for the time of Thomas' death for the reasons stated earlier, and added that it ultimately was irrelevant for the issue at bar: "The child was dead when they arrived. And whether it happened one minute, six minutes, 24 minutes before, I don't see the purpose." Tr. 5 at 1053. He clarified his reading of the EKG readings taken at the hospital:

> I believe I said originally there was no rhythm. I felt maybe there was, perhaps, some ventricular fibrillation demonstrated, and then it looks like -- it's hard because the times aren't always on the strips. But it looks like in the time sequence, the final ones were agonal, which is the typical thing we see when we've finished with CPR and determined that there is no resuscitatability.

Tr. 5 at 1054.

Regarding potential alternative causes for Thomas' death, Dr. Ringel stipulated that SIDS is a "diagnosis of exclusion," used as a label when no cause is known, and that "the reviews of the

pathologists do not indicate a known disease process." Tr. 5 at 1056. Regarding the autopsy findings, Dr. Ringel only felt comfortable giving an opinion on its observations of the heart. Tr. 5 at 1057. Dr. Ringel categorized the heart's weight as "within the range of normal," because the average weight for Thomas' age is 28 grams, and Thomas' heart weighed 30 grams on autopsy, which he compared as "virtually identical." Tr. 5 at 1057. Petitioners challenged Dr. Ringel on this point, pointing out that a two gram difference from average meant Thomas' heart weighed nearly ten percent more than average, but Dr. Ringel refused that analysis:

> No, you can't do that, because average is average. Everything in medicine is considered essentially normal, within two standard deviations of the mean. So that any mean or average has two standard deviations. Now, with knowing the mean, you can't just say what the two standard deviations are on either side, but they're very wide. So there's no way that a 10-percent difference off the mean is ever two standard deviations off the mean. So 30 and 28 are virtually identical.

Tr. 5 at 1057. Dr. Ringel did stipulate that the autopsy report included "indications" of "increased brain weight." Tr. 5 at 1058.

When Petitioners questioned Dr. Ringel as to whether SIDS was "an appropriate diagnosis," he refused to offer an opinion, because it was "really a pathologic diagnosis, and that SIDS has been discussed and argued about for decades now as to what you call SIDS and what you don't call SIDS," and therefore saw the topic as outside his field. Tr. 5 at 1058-59. He did concede that the examining pathologist indicated in the autopsy report that he could not rule out the Hepatitis B vaccine as a causal factor. Tr. 5 at 1059-60.

Next, Petitioners challenged Dr. Ringel with the medical literature they saw as relevant to his opinion. Dr. Ringel, in responding to Dr. Connor's expert report, did not cite to the Dahhan and Cinca articles in his own expert report, which Petitioners found curious. Tr. 5 at 1060. Dr. Ringel did study the Dahhan and Cinca papers for their cardiologic import, and he "read enough of the toxicology discussions to realize that all ethyl mercury is not necessarily alike," which made him chary about agreeing to Petitioners' proposition that "ethyl mercury had a cardiac effect." Tr. 5 at 1061-62.

When asked, regarding the subjects studied in the Dahhan article, whether "there were changes in the entire electrical conduction of the heart," Dr. Ringel demurred, because of the wide variation in cardiac symptoms observed, but he agreed that several abnormalities were noted among the subjects, which varied in nature and degree. Tr. 5 at 1071. Among those, there were some whose EKG demonstrated QT wave prolongation, which presents a risk of "increased irritability, and potentially sudden death," but that depends on how prolonged the QT interval is and what caused the prolongation. Tr. 5 at 1072. He explained the distinction by reference to Dr. Connor's testimony:

> He brought up the familial long QT syndrome, which is quite different than other causes of QT prolongation. And familial long QT syndrome is the type of QT prolongation that is particularly high risk. Other QT prolongations are not necessarily that high risk.

Tr. 5 at 1072. Dr. Ringel did agree that the Dahhan study stated that "all fatalities from mercury poisoning showed a prolonged QT interval," but he pointed out that none of the subjects in the Dahhan study were infants; the youngest subject was fourteen years old. Tr. 5 at 1073.

This led Petitioners to ask Dr. Ringel whether infants were more susceptible to cardiac injury from mercury exposure and/or toxicity, to which he replied, "not necessarily." Tr. 5 at 1073. He elaborated, "There are some things that are much more dangerous for an adult with, say, borderline coronary artery disease, which has little or no effect on a healthy infant." Tr. 5 at 1073-74. In fact, he said, prolonged QT syndrome in particular, if fatal, proves lethal during the teenage years and thereafter, not during infancy or early childhood. Tr. 5 at 1074-75. He stated that this was a good example of how infants "might not be more susceptible" to mercury toxicity. Tr. 5 at 1075.

Moving to the Cinca[144] article, Dr. Ringel described the study as "not focus[ed] as much on being specific about electrocardiograms, but ... did mention EKG abnormalities and some microscopic changes to suggest interstitial myocarditis." Tr. 5 at 1078-79. He explained that interstitial myocarditis leads to dysfunction and weakness and potentially irritability of the heart muscle, which could lead to arrhythmias, which "increase the risk of death." Tr. 5 at 1079. As to whether the medical literature discussed indicates that "ethyl mercury has an effect on the cardiac tissue," Dr. Ringel thought that these studies proved that prolonged exposure to enormous doses of ethyl mercury could lead to mercury toxicity, which could include "cardiac changes and manifestations." Tr. 5 at 1079-80.

Dr. Ringel's cross-examination briefly addressed the Zhang[145] article, wherein subjects had been exposed to Serosin, which is composed of about 2 percent ethyl mercury chloride. Tr. 5 at 1076. As to the cardiac abnormalities noted in the paper, Dr. Ringel summarized, "Seven patients showed ECG abnormality[:] ... [a]ll showed prolonged QT intervals, five showing depression of the ST segments, flat or inverted T waves and large U waves, consistent with low serum potassium levels." Tr. 5 at 1077. He added, however, that, "After potassium supplementation, most values returned to normal or nearly normal values." Tr. 5 at 1077. He went on to state the researchers' conclusion that "perhaps the mercury was affecting potassium levels ... [a]nd perhaps that the EKG changes are related to potassium levels." *Id*.

Regarding the Damluji[146] study filed by Respondent, Dr. Ringel described the cardiac findings that, "The authors report there were no demonstrable cardiac changes in the mild cases; moderate and severe cases had tachycardia, occasionally arrhythmia, and in two cases a moderate rise of blood pressure." Tr. 5 at 1067. He stated, "The electrocardiographic findings were variable,

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[144] Pet. Ex. 18, Tab V, I. Cinca *et al.*, *Accidental ethyl mercury poisoning with nervous system, skeletal muscle, and myocardium injury*, 43 JOURNAL OF NEUROLOGY, NEUROSURGERY, AND PSYCHIATRY 143-49 (1979).

[145] Jimel Zhang, *Clinical Observations in Ethyl Mercury Chloride Poisoning*, 5 AMERICAN JOURNAL OF INDUSTRIAL MEDICINE 251-58 (1984).

[146] Resp. Ex. A, Tab 2, Salem Damluji, *Mercurial Poisoning with the Fungicide Granosan M.*, 4 (3) J. FAC. MED. BAGHDAD 83-103 (1962).

and showed multiple extrasystoles,[147] T-wave changes, and ST depression," adding that the paper is not specific as to the nature of those extrasystoles, "so the extrasystoles could be ventricular or atrial." Tr. 5 at 1067-68.

Moving to the adverse cardiac findings in the Jalili[148] article, Dr. Ringel summarized:

> In severe cases, the pulse is irregular, sometimes with bradycardia.[149] Electrocardiographic examination of severe cases showed frequent ventricular [ectopic] beats, prolongation of the QT interval, depression of the ST segment, and T inversion.... Twelve-lead records were made for 15 patients. Nine of these showed certain abnormalities.... [The abnormalities were i]mpaired conduction evidenced by prolonged PR interval, increased irritability of the heart with frequent [ectopic] beats, and severe myocardial damage, shown by prolonged QT segment and ST deviation with inversion of T waves, were present singly or in combination.

Tr. 5 at 1069. When, regarding this findings, Petitioners queried Dr. Ringel whether the authors of this study "thought that ethyl mercury played a role in these people's cardiac abnormalities," Dr. Ringel refused the proposition, stating, "You seem to use ethyl mercury always generically, and I have to see whether that's really the case, and what they describe here. So I can't answer just yet." Tr. 5 at 1069-70.

Petitioners challenged Dr. Ringel with the National Academy of Sciences monograph *The Toxicological Effects of Methyl Mercury*,[150] which Dr. Ringel stipulated was a reliable scientific source. Tr. 5 at 1085. Dr. Ringel did not dispute the contents of the work, and even stipulated that EKG abnormalities in both children and adults was discussed therein as a finding, but he disputed the relevance of such a source in the context of the issue at bar:

> [W]hat they say is that particularly high levels of exposure and chronic exposure can lead to toxic cardiac effects. They particularly point to hypertension, and they mention some hypertrophic cardiomyopathy. They also mention that the exposure sometimes takes many years. They refer to the Faroe Island experience, where the exposure took seven years to demonstrate an effect. They mention that there were some effects of prenatal exposure, and that again, I think it was the Faroe Island experience, where the changes were noted, subtle changes were noted at seven years. They talk about a wide range of things. I didn't see anything that suggested sudden

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[147] An extrasystole is "a premature contraction of the heart that is independent of the normal rhythm and arises in response to an impulse in some part of the heart other than the sinoatrial node." DORLAND'S, *supra*, at 659.

[148] Resp. Ex. A, Tab 27, M. A. Jalili and A. H. Abbasi, *Poisoning by Ethyl Mercury Toluene Sulphonanilide*, 18 BRITISH JOURNAL OF INDUSTRIAL MEDICINE 303-308 (1961).

[149] Bradycardia is "slowness of the heartbeat, as evidenced by slowing of the pulse rate to less than 60." DORLAND'S, *supra*, at 246.

[150] Pet. Ex. 18, Tab HHH, Committee on the Toxicological Effects of Methylmercury, *Toxicological Effects of Methylmercury* 169-172 (2000).

> infant death. I didn't see anything in there that talked about newborn infants or, you know, which we consider up to two months, and neonates dying from this. They talk a lot about older people, they talk about provoking coronary artery disease, they talk about, again, hypertension and things of that sort.

Tr. 5 at 1085-86. To this, Petitioners propositioned him again regarding whether "ethyl mercury has a cardiac effect," to which he responded with qualification, "[T]hat's being non-specific ... very high, long-term exposures of ethyl mercury-containing compounds [do] appear to have toxic effects on children and adults." Tr. 5 at 1086.

In sum, Dr. Ringel did not find those poisoning studies to be relevant to the issue of whether the amount of ethyl mercury in two Hepatitis B vaccines could cause sudden death in a newborn infant:

> I did not find any of these relevant to the Kolakowski case. We've been discussing these articles, you've been asking me questions. I don't dispute the findings of them at all, but we have no evidence that it bears any relationship to the Kolakowski case. There is no interstitial myocarditis on the microscopic examination of the heart. There is no evidence of congestive heart failure. There was no evidence of arrhythmia. We have no electrocardiograms to show us any of the findings that any of these authors presented. So I didn't find any reason to be correlating these articles to the Kolakowski case.

Tr. 5 at 1080-81. This exchange led to a fruitful and telling discussion on Thomas Kolakowski's autopsy:

> Q ...When you reviewed the autopsies, did you see any indication that the lungs were congested?
>
> A Yes, the lungs were congested. But congestion of the lungs will occur in a number of circumstances. I can feel comfortable saying limited things about congestion, because we deal with pulmonary congestion as a large part of our practice of pediatric cardiology. But they also occur in patients who suffer from suffocation. I've seen patients in my days of taking care of patients in the ICU, of kids who would develop what we call flash pulmonary edema from obstructive apnea. The airway obstructs. They make intense respiratory motions. They create a lot of negative pressure in the chest, get fluid accumulation, and get pulmonary edema. So I've seen it happen before my eyes, so I know it can occur just from blockage of the airway.
>
> Q And is it your opinion right now that Tommy suffered suffocation?
>
> A I was explaining that as an alternative explanation to congestive heart failure, saying that there are other ways of getting pulmonary edema. But I'm not going to diagnose his pulmonary edema. I would leave that to the pathologists who have looked at the slides, and might have additional thoughts about it, and to the pathologists who will be discussing the case. But I, I think it's fair, since I've made

> the statement, to say that what I've seen is consistent with things other than a primary cardiac death.
>
> Q So right now, as you're sitting in the stand, are you saying that Tommy suffered a suffocation, and that's why he died?
>
> A I'm saying, as I raised in my statement, that we don't have to evoke exotic stretches of imagination to explain how this child died, when we have a situation, which has been recognized by the American Academy of Pediatrics, to lead to this type of death. So when I read the case, the first thing that comes to mind is not that he is the one in hundreds and hundreds of thousands of patients who have been exposed to hepatitis vaccine, that this somehow caused his sudden demise, when we know that bed-sharing and suffocation related to bed-sharing is a risk factor for this type of death. So I'm not saying I'm making the diagnosis, but I've pointed to the must more likely explanation of his demise.

Tr. 5 at 1081-82. Later, Petitioners followed up on this point by asking the medical significance of the fact that Thomas was found to have blood from the nose and mouth, which was observed by the EMTs and mentioned by his mother's affidavit, to which Dr. Ringel responded:

> I believe I had seen in my career in critical care an occasional patient that seemed to have had some either suffocation or unexplained death, have some blood in the mouth. So I have an impression that that can be associated with an asphyxial death, but it's not more than that.

Tr. 5 at 1092.

In response to a question from Petitioners that actually related to Petitioners' purported theory of causation, Dr. Ringel stated that the autonomic nervous system "affects cardiac activity" in that it "helps to regulate the heart rate, speeding it up and slowing it down, depending on what is required," and coordinates "the respiratory drive with the heart rate." Tr. 5 at 1086-87. He described the nature and location of those nerves thusly:

> The main impulse for the [parasympathetic] nervous system, the negative effect, if you will, or the slowing effect on the heart, comes down from the brain, the mid-brain, to the vagus nerve into the chest. And the accelerant side of things, the sympathetic system, comes down through these paraspinal chains along the spinal cord from the mid-brain down into the chest.[151]

Tr. 5 at 1087. Dr. Ringel agreed that injury to the autonomic nervous system could potentially "affect the body's ability to respond to certain events, whether speeding it up or slowing the heart down." *Id*.

[151] The parasympathetic and sympathetic nervous systems together form the autonomic nervous system. See supra at note 121.

6. Enid Gilbert-Barness, M.D.

Dr. Gilbert-Barness attended medical school at the University of Sydney in Australia. Tr. 5 at 1094. She worked for a time in pediatrics, before training in pathology. Tr. 5 at 1094-95.As a pathologist, she worked at the University of Wisconsin, where she taught as professor of pediatrics and of pathology over a span of 23 years. Tr. 5 at 1095. She has since moved to Tampa, Florida, where she currently serves as Director of Pediatric Pathology at the University of South Florida, teaching as a professor in the areas of pathology, pediatrics, and Obstetrics & Gynecology. *Id*. She is board-certified in pediatrics, clinical pathology, anatomic (tissue) pathology, and pediatric pathology. Tr. 5 at 1096. She sat on the American Board of Pathology on the topic of pediatric pathology, where she was an examiner on that committee and wrote questions on that topic for the board examination for a period of eight years. Tr. 5 at 1097. She served as president of both the Society for Pediatric Pathology and the International Pediatric Pathology Association. *Id*. She has served on seven editorial review boards over the years, and, at the time of the hearing, was editor-in-chief of *Fetal and Pediatric Pathology*. Tr. 5 at 1098. One of her professional interests has been cardiogenesis and malformations thereof, pursuant to which, she has written for publication around 50 articles regarding the cardiovascular system. Tr. 5 at 1099.

Also on the topic of Dr. Gilbert-Barness' published articles, she wrote one "some years ago" having to do with mercury toxicity, and, in particular, Minamata disease, named after Minamata Bay in Japan, where several poisoning studies have been conducted due to the consumption of mercury-contaminated fish. Tr. 5 at 1097. Her task was to examine the cases from the pathology standpoint. *Id*.

Another area of professional interest for Dr. Gilbert-Barness, resulting in over three dozen published articles, has been Sudden Infant Death, and the circumstance commonly referred to (improperly, she maintains) as Sudden Infant Death Syndrome, or SIDS:

> Well, I've been very interested in sudden infant death, and actually the mechanism of sudden infant death, because for a long time, and even now, many people refer to SIDS, meaning Sudden Infant Death Syndrome. Well, what is a syndrome? It's ridiculous to call anything SIDS. And now I have, and hopefully most people are following my recommendation, that it should be called sudden infant death, and what the cause is. And in this particular case, I'm convinced that it is an asphyxial or anoxic death.

Tr. 5 at 1100-01.

Dr. Gilbert-Barness stated her conclusion in this case, that Thomas Kolakowski "died from asphyxia due to anoxic encephalopathy,"[152] a conclusion for which she has "no doubt whatsoever." Tr. 5 at 1103. The rest of her direct testimony was spent explaining her medical opinion, and the analysis by which she drew that conclusion.

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[152] An hypoxic encephalopathy is an "encephalopathy caused by hypoxia from either decreased rated of blood flow or decreased oxygen content of arterial blood." DORLAND'S, *supra*, at 611. The prefix "anoxic" used in place of "hypoxic" means that the decrease is to the point of absolute cessation.

Dr. Gilbert-Barness' introduction began with noting that she is "familiar with the procedures for conducting autopsies," so much so that she had "written a book on pediatric autopsy pathology," that was a "rather large book, close to 1,000 pages." Tr. 5 at 1103. She had conducted approximately 10,000 autopsies at the time of the hearing. Tr. 5 at 1104. She explained that organs are usually first preserved in formaldehyde (or "fixed in form") prior to taking slide samples, although sometimes the organs may be processed for pathological examination when they are fresh. Tr. 5 at 1104. She could not tell from the medical records which procedure was followed, because no microscopic examination was written up in Thomas' autopsy report. *Id*. Overall, her position on the quality of the slide presentation differed sharply from that of Dr. Shane, because she "thought the slides were [of] poor quality" and substandard clarity, although they were not so poor to be unintelligible. *Id*. For example, she observed "artifacts" on the slides, which could result from "dirt on the slide," "poor fixation," or other external influences. Tr. 5 at 1105. Specifically, on a slide with myocardial heart tissue from Thomas Kolakowski's autopsy slides, Dr. Gilbert-Barness pointed out "all those little black specks all over that slide is artifact, and that's, excuse me, disgraceful to even present a slide like that." Tr. 5 at 1106. Moreover, discussing the same slide, Dr. Gilbert-Barness stated:

> [Y]ou can see that there is no interstitial myocarditis. I think that's good to show that, because there's absolutely no inflammatory reaction there, which was described by Dr. Shane. And I'm sorry, but it just wasn't there.

Tr. 5 at 1106.

Moving back to discuss the autopsy report in general, Dr. Gilbert-Barness noted that the autopsy was only a description of gross findings and weights, but included no microscopic analysis. Tr. 5 at 1106-07. When asked whether she would expect to see a microscopic analysis in a case like this, Dr. Gilbert-Barness said that doing so was "all part of an autopsy." Tr. 5 at 1107.

Next, Dr. Gilbert-Barness began her discussion about her theory as to what caused Thomas Kolakowski's death. She believes "essentially all [of] the so-called SIDS cases, almost all of them, have turned out to be anoxic encephalopathy," due either to "the prone position, or overlying, or waterbeds an all these other devices that have resulted in anoxic death." Tr. 5 at 1107. In her opinion, her experience has shown that, in those cases where "medical examiners [classified] a case as SIDS," the microscopic evidence actually supports hypoxic/ischemic injury.[153] Tr. 5 at 1108. Moving to the instant case, Dr. Gilbert-Barness found it significant that "the medical records suggest that at the least, Mrs. Kolakowski was co-sleeping with Thomas at the time of death," inasmuch as "co-sleeping [can] lead to overlying, and result in an anoxic death." *Id*.

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[153] Dr. Gilbert-Barness provided a gloss for her terminology:

> Hypoxia is lack of oxygen, very low oxygen levels in the blood.... Anoxia means no oxygen. So in a way, that's a misnomer. It's better to call it hypoxia, but most people do refer to it as hypoxia/anoxia.... Ischemia is lack of oxygen in tissue. So if something is ischemic, it has lacked oxygen or blood supply.

Tr. 5 at 1107-08.

Dr. Gilbert-Barness compared that evidence with the evidence for mercury toxicity, weighed in the context of her professional experience with mercury poisoning. Tr. 5 at 1109. To her, the indicia of mercury toxicity are distinctive and many of them are specific, such that if exposure to mercury had been integral in Thomas' death, the record would reflect tell-tale signs.[154] *Id.* Not only did Thomas Kolakowski not evidence those indicia, he did not appear to manifest the ones claimed by Petitioners:

> Q ...[D]id you find any evidence that Thomas Kolakowski may have had dysrhythmia prior to the night that he passed away?
>
> A I don't believe he did. I think that the irregularity of the heart was probably agonal.... Which many patients have.
>
> Q So it's certainly nothing specific to mercury.
>
> A No.

Tr. 5 at 1110.

Dr. Gilbert-Barness differed with Dr. Shane's finding of "diffuse interstitial edema in all sections of the heart, and in particular, in the interventricular septum." Tr. 5 at 1110. She stated, "In fact, I was so concerned that I saw absolutely nothing, that I wondered whether I was thinking right or not. So I showed it around to six of my colleagues, pathologists. And they confirmed what I had said, that there was no evidence of a myocarditis."[155] Tr. 5 at 1110-11. She did not see any of the "inflammatory cell infiltrate," or the "mitochondrial cell shrinkage" in the cardiac septum or the "myocardial cell degeneration," all of which Dr. Shane said he observed in the slide samples. Tr. 5 at 1111. She did not see what he noted in the way of "chronic process going on in the heart." *Id.* Additionally, Dr. Gilbert-Barness thought Thomas' heart weight of 30g was well within the normal "standard deviation" range, and that a heart weight would have to be "at least 35 or maybe 40 [grams] before you would be concerned." Tr. 5 at 1112. Therefore, her opinion, based on both the

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[154] As Dr. Gilbert-Barness explained,

> Well, many years ago, and I hate to say how many years, but when I was a medical student attending the wards at the Children's Hospital there, there were many children who suffered from a condition which was called acrodynia. And that was due to mercury intoxication. These babies had been given a mercury compound, mercurous chloride I believe it was, that caused toxicity. And they had extreme photophobia. They couldn't stand seeing the light. And I'd go into these wards with, you know, 20, 30 patients, with their heads buried in the pillow because they couldn't stand the light. And they are examples of acute mercury toxicity. And I actually, as a medical student, wrote a paper on it, that was never published, but it was interesting to me, that finally they found that it was due to mercury.
>
> Q Would you say that the clinical manifestations were stark?
>
> A Oh, yes, absolutely.

Tr. 5 at 1109.

[155] Dr. Gilbert-Barness defined myocarditis as "an inflammation of the heart muscle ... usually due to a viral infection or some other type of infection that causes it." Tr. 5 at 1110.

gross findings and her own microscopic review, was that Thomas' heart was not "swollen due to edema." *Id*.

The autopsy's gross examination noted fine petechial hemorrhages on the left ventricle of Thomas' heart, which Dr. Gilbert-Barness stated was "typical of an anoxic death," and lends support for her conclusion. Tr. 5 at 1112-13. Another finding of the autopsy's gross examination–30cc of bilateral sanguineous pleural fluid–was not determinative either way, because it was not a large amount relatively speaking; however, it was consistent with hypoxic death. Tr. 5 at 1113. She explained how: First, "there is dilatation of capillaries, the small blood vessels," and then "they burst, and you can get these little petechial hemorrhages that we call them, or exudation of fluid, particularly in the pleura." Tr. 5 at 1113-14. Asked what she made of the diffuse consolidation[156] of the lungs, Dr. Gilbert-Barness referred to the microscopic data of "hemorrhages in the lung, and ... congestion of the capillaries of the air spaces, which she viewed as "also very good supportive evidence of a hypoxic or an anoxic death," inasmuch as "in an asphyxial death, ... there is lack of oxygen [during which t]he infant struggles for air momentarily, until it succumbs[, leading to] dilatation of capillaries, bursting of the capillaries, and hemorrhages." Tr. 5 at 1114. She added, "particularly in asphyxial deaths, you'll find areas of hemorrhage in the lungs [which are] so striking that I can just look at lungs, and ... I'm sure that that is an asphyxial death." Tr. 5 at 1114-15. She agreed with Dr. Shane's notation of "prominent alveolar wall capillary dilation and congestion," but viewed the finding as more supportive of asphyxial, and therefore hypoxic, death. Tr. 5 at 1115. She agreed that the lungs were heavy, but stated that this was also due to the asphyxial process she had been describing. Tr. 5 at 1116. None of these findings would have required a chronic process, she maintained, insisting that such effects could transpire "very rapidly," even "instantaneously, very soon after hypoxia or asphyxia is created," because of the struggle to breathe, such that "it probably could occur within a couple of minutes, or even less." *Id*. Moving on within the autopsy report, Dr. Gilbert-Barness thought the notation about congestion in the liver was consistent with her conclusion, because "in a case of an asphyxial death, all the organs become congested, because this is a struggle to get more oxygenated blood to the baby." Tr. 5 at 1117. Similarly, with the oral and nasal bleeding, Dr. Gilbert-Barness stated that such findings are "very commonly seen in asphyxial deaths," adding a moment later that pathologists "almost always see" nasal and/or oral bleeding in cases of bed-sharing-related asphyxia. Tr. 5 at 1118.

In sum, there was nothing in the pathological findings in this case that would lead Dr. Gilbert-Barness to believe Thomas Kolakowski's death resulted from anything else besides hypoxic or anoxic death by asphyxiation. Tr. 5 at 1117. She said the pathological evidence was not just consistent with such a conclusion, but diagnostic of it. Tr. 5 at 1118.

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[156] Dr. Gilbert-Barness saw that term almost as a term of art in pathology, used outside the manner it is typically used clinically:

> I think he's described it as consolidation. It's not really consolidation as we usually use that term, to mean pneumonia, or consolidation in that respect. But if you have hemorrhage in the lung, the lungs look fairly solid. And that's why he's described it as consolidated.

Tr. 5 at 1115.

Dr. Gilbert-Barness stated that even the description of the dependent lividity in the EMT and hospital records supported a conclusion of asphyxial death:

> Well, after death, and this is positional, blood seeps down to the pendant portions. And there are areas of what's called lividity. That's a bluish-reddish appearance of the skin. And that is because of the pooling of the blood. [I]n this case, the person who did the autopsy described it as purple. Well, that is another feature of an anoxic death. The lividity looks much darker and purple. And he actually described this, not realizing what he was really saying, but that was what was said.... [The lividity looks darker, or purple b]ecause before death, there must be some period of time -- and I can't tell you how long -- but when the infant is starting to have the effects of asphyxia, then there's a lack of oxygen. And so the blood, before death, is hypoxic; and therefore, the seeping of the blood after death tends to be more purple.

Tr. 5 at 1119. Later, on cross-examination, Dr. Gilbert-Barness stated that "lividity occurs within probably 30 minutes, or certainly within an hour of death," and reiterated that "if it's dark and purple, as this case was, one would strongly suspect an asphyxial death." Tr. 5 at 1132.

Dr. Gilbert-Barness disagreed with Dr. Shane's assessment that Thomas' temperature indicated his death had been very recent to when he was discovered unresponsive: She would not expect the body temperature to drop precipitously after death occurred, saying, "It drops slowly over a period of time ... usually said to be about one degree per hour." Tr. 5 at 1120. She stated that Thomas' temperature, recorded in the hospital as 96.4°F, correlates to having been deceased for two hours at that point. *Id*. This would place the time of death at 5:30 AM that morning. Tr. 5 at 1125. Also, regarding the clinical records, Dr. Gilbert-Barness found it relevant that Thomas was noted to have been experiencing a head cold in the period leading up to his death, inasmuch as seventy-five percent of sudden infant deaths show a preexisting history of upper respiratory infection. *Id*.

On cross-examination, Dr. Gilbert-Barness reiterated her opinion that Thomas Kolakowski died from asphyxial death, presumably from overlying.[157] Tr. 5 at 1121. She clarified that she meant that the medical record redounded to the conclusion of asphyxia, and Dr. Gilbert-Barness was presuming, based on the sparse evidence available on the subject (*i.e.*, co-sleeping), that maternal overlying was the mechanism of the asphyxiation; whereas she held her opinion of asphyxia as a cause of death beyond a preponderance of the evidence, she was not necessarily as committed to inadvertent maternal overlying as a mechanism. Tr. 5 at 1121-24.

The next relevant topic on cross-examination was the finding of left ventricular petechial hemorrhages, which Dr. Gilbert-Barness had said supported her conclusion of asphyxial death. Tr.

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[157] Dr. Gilbert-Barness explained what she meant by that term thusly:

> Well, when a baby is in bed with the mother, and she turns, she overlies and squashes the face. And it's well known that babies up to four to six months of age are obligate nasal breathers. If you obstruct the nose, they are asphyxiated. Even though their mouth may be open. And this is a factor that not many people realize, but these babies are obligate nasal breathers. So if this baby was lying with the mother, and the mother turned and obstructed the nose, then asphyxia will occur.

Tr. 5 at 1121.

5 at 1125-26. Dr. Gilbert-Barness stated that those hemorrhages could occur anywhere, including the heart, as they were in this case. Tr. 5 at 1126. Petitioners queried whether the simple act of performing CPR on an infant like Thomas could cause petechial hemorrhages, but Dr. Gilbert-Barness rejoined that such a result would be impossible if the infant was already dead by the time CPR was attempted, and that CPR "will not produce petechial hemorrhages." Tr. 5 at 1126. However, she said, "Petechial hemorrhages are such an absolute landmark for an asphyxial death" that they have their own term in that context ("Tardieu[158] spots"), and "have been known for centuries." *Id.* Dr. Gilbert-Barness explained that, although such hemorrhaging can appear anywhere in the body in the event of asphyxia, they are most common in the pleura, but also are known to appear in the heart, thymus, and eyes. Tr. 5 at 1126-27. In Thomas' case, they were only seen in the heart. Tr. 5 at 1129.

Regarding the 30cc of bilateral, sanguinous, pleural fluid, Dr. Gilbert-Barness opined that, like the petechial hemorrhages, the larger amount of serosanguinous fluid was a product of the asphyxia, and is a very common finding in asphyxial deaths. Tr. 5 at 1129-31. They are both caused by the enormous negative pressure caused by the suction of blocked inhalation. Tr. 5 at 1136. Fluid in the pleural cavities is common as an artifact of the agonal process, said Dr. Gilbert-Barness, and "most fluid has blood in it." Tr. 5 at 1130. When challenged by Petitioners, Dr. Gilbert-Barness agreed that visceral organ congestion is nonspecific and can be caused by many things. Tr. 5 at 1131. She did maintain that the finding of visceral organ congestion is certainly consistent with asphyxial death, though, as it is caused by "a dilatation of all the vessels, particularly the capillaries," leading to the accumulation of much fluid. Tr. 5 at 1139. "To a certain extent," the same can be said regarding the brain. Tr. 5 at 1140.

Petitioners challenged Dr. Gilbert-Barness on her estimation for time of death, asking, "if a child has been dead for two hours, how can there still be electrical activity in the heart?," to which she replied that, "the electrical circuit may not be entirely shut off," adding "Arrhythmias at the time of death are not uncommon." Tr. 5 at 1133-34. Pressing further on this point, Petitioners queried how the heart could still be active if the blood in the circulatory system is not moving, but pooled in one spot, to which Dr. Gilbert-Barness replied that the conduction system is really electric, and can be stimulated or active electrically, even if not mechanically operational to move blood. Tr. 5 at 1135.

The last topic of cross-examination was a crucial discussion about nomenclature, and the loaded term of "SIDS." When asked if her opinion was that Thomas Kolakowski "died from SIDS," Dr. Gilbert-Barness astutely clarified that "some people would call it SIDS," but that she was "calling it an asphyxial death," as part of "a hypoxic/anoxic type of injury." Tr. 5 at 1140. A moment later, she elaborated further:

> A lot of people still call this SIDS. Asphyxia. It's only recently, and I'm very proud of the fact that I'm one of the people who insisted that the sleeping position be changed, and that parents were advised when they left the hospital with a new baby

[158] Tardieu's spots are "spots of ecchymosis under the pleura following death by suffocation." DORLAND'S, *supra*, at 1745.

> that they have the baby sleep on ... the back. Because the prone position has now been strongly related to what has been called SIDS. And it's what I believe is an asphyxial death.

Tr. 5 at 1140-41. Petitioner followed up by noting that the medical examiner "did not assign cause of death to be an asphyxial hypoxic type of death," to which Dr. Gilbert-Barness responded that, "Well, he called it SIDS, which is, to me is saying the same thing." Tr. 5 at 1142. Petitioners rightly pointed out that the medical examiner termed the death "sudden death in infancy" which is just "a diagnosis of exclusion," and Dr. Gilbert-Barness clarified that "he didn't really find anything. So he's calling it sudden infant -- that's the same thing as SIDS, sudden death in infancy, yes. But that means nothing." *Id*.

This sparked a discussion about the major problem with using SIDS as a diagnostic tool: in some cases, it truly is just a diagnosis of exclusion–a medical throwing up of hands in bewilderment, while in other cases it is describing a specific constellation of symptoms that, in all likelihood, describes the effect of positional asphyxia. *See Perez v. Sec'y of HHS*, Case No. 05-1261V, 2008 WL 763301 (Fed. Cl. Spec. Mstr. Mar. 4, 2008). The following interchange is illustrative:

> Q So there's no other cause. They ruled out all the other causes, and that's what they believe it is, is SIDS.
>
> A Well, if it's SIDS as I believe it is, it's an asphyxial death. There is no evidence whatsoever that this could be anything but an asphyxial death. And I really stand firm on that. I've seen hundreds of cases, and I know this area.
>
> Q So Doctor, when you reviewed the autopsy report, you did see that Dr. Ross indicated that he could not draw a conclusion as to whether or not Heptavax was causally connected to the child's death.
>
> A Yes, I think he did say that. But he didn't even give a microscopic opinion, report.
>
> Q So you disagree with Dr. Ross, who did the actual autopsy?
>
> A Well, I disagree with that statement, yes.

Tr. 5 at 1142-43.

On redirect examination, Dr. Gilbert-Barness reiterated her opinion that Thomas' Hepatitis B vaccinations "had nothing to do with" his untimely death. Tr. 5 at 1143. She added that, since he died from "respiratory asphyxia," his death was unrelated to any toxic insult from mercury, which would have been "a metabolic effect." Tr. 5 at 1143-44.

7. Lucy B. Rorke-Adams, MD

Dr. Rorke-Adams received her Bachelor of Arts, Master of Arts, Bachelor of Science, and Doctor of Medicine degrees from the University of Minnesota, the last of which she was awarded in 1957. Tr. 6 at 1456. Her internship thereafter was at Philadelphia General Hospital, after which, she remained for her residency training in anatomical pathology, as well as a fellowship through the

NIH to study neuropathology. *Id*. She sat for her board certification in anatomical pathology and neuropathology in 1962 and 1963, respectively. Tr. 6 at 1457. Dr. Rorke-Adams remained at Philadelphia General on staff when her training concluded, taking the title Chief of Pediatric Pathology. Tr. 6 at 1460-61. Ever since she had begun her residency, she had been delegated the task of performing all pediatric autopsies, and was very experienced in that regard when she was named chief. Tr. 6 at 1461. Thereafter, she was named chief of Neuropathology, and then chief of the entire Pathology department before seven years had passed since her fellowship. *Id*. During that interval, she began serving simultaneously as the sole neuropathologist at Children's Hospital in Philadelphia. *Id*. Back at Philadelphia General, she also served as President of the medical staff, and, for a brief stint, as Medical Director for the hospital. *Id*. Eventually, she left Philadelphia General to work exclusively at Children's Hospital (where she again served as president of the medical staff) and joined the office of the Medical Examiner for Philadelphia. Tr. 6 at 1461-62.

Her current duties at Children's Hospital are composed of diagnosis and teaching residents, and she is often invited to lecture across the country and the world. Tr. 6 at 1462. Dr. Rorke-Adams has performed 800 to 1,000 autopsies, and has performed the central nervous system component of over 20,000 autopsies. Tr. 6 at 1473. Counted among the awards she has received have been "the bronze medal from the Brain Research Institute in Niigata, Japan, back in 1985," and, in 1999, "from the American Association of Neuropathologists, the bronze plaque for meritorious contributions," which is "basically the ultimate honor that an American or a pathologist could receive from their specialty group." Tr. 6 at 1465. She has served as President of the American Association of Neuropathologists. Tr. 6 at 1466. Also, she sat on the certification board for the American Board of Pathology, developing examination questions for the neuropathology examination component. Tr. 6 at 1468.

She described her editorial review board work for scientific and medical publications as "reviewing manuscripts that have been submitted for possible publication in the journal, and to make certain that the material in the manuscript is scientifically sound," which, she added, was "a very major responsibility, to make certain that foolishness doesn't get into the literature, [such that it could be] ultimately carried on and quoted." Tr. 6 at 1467. She performed this work, over the years, for the *Journal of Neuropathology and Experimental Neurology* (the American Association of Neuropathologists' journal), *Pediatric Neuroscience* (now *Pediatric Neurosurgery of the Child's Nervous System*), *Brain Pathology*, the *International Journal of Neuroradiology* (for which she was one of the original editors), the *Journal of Surgical Pathology, Histology, and Histopathology*, and the editorial advisory board for the Pediatric Cancer Treatment Group at NIH. Tr. 6 at 1468. In authoring her own articles, her particular interest lies in "[c]hildhood brain tumors, forensic neuropathology as it deals with children," and she had previously done much in the area of "central nervous system infections, primarily those caused by viruses." *Id*. One of her articles (her 147th) was entitled, "*Anatomical Features of the Developing Brain Implicated in Pathogenesis of Hypoxic-Ischemic Injury*." Tr. 6 at 1469. She wrote a book, for which she and another doctor spent seven years of research, discussing the myelination of the infant brain. Tr. 6 at 1470. She had "just contributed a chapter to a two-volume book that Dr. Gilbert had edited," for which she wrote the nervous system chapter, the longest in the book at 150 pages, and which is the standard in pathology laboratories: "I don't know about medical students; it's a little bit too detailed for medical students. But it's certainly used in the training of pathology residents and fellows." Tr. 6 at 1471.

She defined the field of neuropathology as "the study of the nervous system [including] the brain, spinal cord, the coverings, the peripheral nerves, and the muscles." Tr. 6 at 1457. She contradicted a point made by Dr. Shane, made when he said he would be qualified by his training and experience to sit for the neuropathology board certification examination, but just had never done so, because it was not the "in" thing to do at the time: She stated that a doctor that had not completed a special fellowship, such as the one she participated in, would not even be eligible to sit for the neuropathology subspecialty examination. Tr. 6 at 1458-59. She said the exception to this rule would have been in the case of a doctor who "had entered the field, and had distinguished himself or herself over a period of years" during the time before the establishment of the neuropathology board, which Dr. Rorke-Adams said were instituted "sometime in the fifties." Tr. 6 at 1459. She gave two examples, of which she was personally familiar, of neuropathologists who were top experts in neuropathology who nonetheless sat for the examination despite not having undergone such a fellowship and were thus "grandfathered in," which would have been, in any event, "a very unusual situation in which they've had extensive experience and are recognized by their peers throughout the country and the world." *Id*.

Next, Dr. Rorke-Adams described the specific difficulty of pediatric neuropathology:

> Evaluation of a baby brain is a rather difficult thing; most general pathologists find it too much of a challenge, and don't pay too much attention to it. Because the problem in pediatric neuropathology involves not only knowing the pathology, but you have to know the neuroanatomy. And in a baby, the neuroanatomy is constantly changing. So that what is normal development at, let's say 32 weeks' gestation, is not normal development at 38 weeks' gestation. So you have a constantly moving target, so to speak, when you're trying to deal with baby brains. And then there are many diseases that are unique to babies that one never sees in the adult population. So it's a very difficult and complex and challenging subspecialty. Most of the literature was in German. Fortunately, I read German, so I was able to tap into the experience of the German pathologists.

Tr. 6 at 1464. She added that all of her pathology practice is spent on pediatric pathology, and that, among the pediatric neuropathologists in the entire world, "probably five or six [are] in this country at the most; and throughout the world, maybe another 15 or 20." Tr. 6 at 1464-65.

Dr. Rorke-Adams began her testimony in chief by stating her conclusion that Thomas Kolakowski "died as a consequence of lack of oxygen to his nervous system," and that his death had nothing to do with mercury toxicity. Tr. 6 at 1473.

Dr. Rorke-Adams said it was typical for organs to be preserved in formaldehyde before samples are taken to be fixed for microscopic analysis, which she presumed had been done in Thomas' case, as "[t]here would be no reason to do otherwise." Tr. 6 at 1473. She thought that the pathology slides prepared from Thomas' autopsy–at least those of the nervous system–were "rather bad" (though still interpretable), as they "had considerable technical artifact, which suggested to me that perhaps the tissue wasn't too well fixed, or that the technician preparing the sections wasn't experienced." Tr. 6 at 1474-75. She described artifacts as "[t]ears in the tissue, and things that are really not part of the pathological process, but have to be contended with." Tr. 6 at 1474. She noted

that, although the autopsy contained "a fairly good description of the gross pathology," any description of a microscopic analysis was lacking, which she "certainly would have" expected to be done, "particularly in a case where the pathologist really couldn't come to a definitive conclusion about the cause of death, and used kind of a cop-out diagnosis of sudden infant death[159] or sudden unexpected death." Tr. 6 at 1475. She added that an "autopsy is not complete unless you do a microscopic" analysis. *Id*.

When she was asked "how often [she was] able to determine that [a] child died as a result of hypoxic or anoxic ischemic injury,"[160] Dr. Rorke-Adams answered, "Well, if it's there, we can usually determine it. Unless they die, you know, within seconds or something." Tr. 6 at 1476. She added that, far from being a rare finding, hypoxic ischemic findings were "quite common." *Id*. The reference in the medical records that Mrs. Kolakowski was sharing a bed with Thomas leading up to the discovery of his nonresponsive state was significant to Dr. Rorke-Adams, although she did not think an actual "roll-over" would have to occur to cause a co-sleeping-related asphyxia: "[T]he nose can be covered up by a pillow or a blanket or something, so somebody doesn't actually have to roll over on the child. But that's the general term we use in the office." Tr. 6 at 1478-79. She stated that

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[159] Dr. Rorke-Adams explained the problem of "SIDS" as a diagnostic term:

> Well, first of all, it's not a syndrome. It's not like some well-known clinical entity in which you have various components that all work together to produce the disease. Sudden infant death syndrome is a term that has appeared, and is part of the literature, to represent the fact that an infant dies for no apparent reason. And after extensive investigation of the clinical aspects ... leading up to the death, the completion of the autopsy, plus or minus other kinds of studies as are available, then you come up with no rational explanation for why the individual died. And this is why I used the term "cop-out" before. Because it's basically a diagnosis of exclusion. You've done everything you can to try to determine why the child died, and you haven't been able to do so, and so you call it SIDS. Or the term that we use now, preferably, is sudden unexpected death.
>
> Q And is it a frequent occurrence in your experience that a medical examiner will label a case a sudden infant death, or sudden unexplained death; and then, on microscopic, further microscopic review, it becomes evident to you that it's a hypoxic ischemic injury?
>
> A Yes.
>
> Q Or a hypoxic ischemic cause of death.
>
> A Right.

Tr. 6 at 1477-78.

[160] Dr. Rorke-Adams provided the following gloss for those three terms:

> [H]ypoxia simply refers to a decrease in oxygen. So it's less than what is required.... Anoxia is total lack of oxygen. That doesn't happen very often.... Ischemia refers to blood flow, so it refers to inadequate blood flow. And they're two words I generally use together, because if you have sufficient hypoxia, eventually the blood flow is going to be reduced as well, because hypoxia to the body is going to affect the heart, along with all of the other organs. So the heart will start failing. So that means that the contractibility of the heart is not sufficiently strong to maintain the blood flow. And so you then have the combination of the hypoxia with the ischemia.

Tr. 6 at 1476-77.

oral-nasal bleeding was a "quite common" and consistent finding in hypoxic ischemia deaths. Tr. 6 at 1479.

Still discussing the clinical findings, Dr. Rorke-Adams explained that the medical records' reference to "dependent lividity" meant that there had been "a pooling of the blood into the dependent parts of the body," going on to explain how it could be used to determine the position of the person when they died, or at least how they came to rest: [M]ost people die lying in bed, on their back. And so the lividity is usually on the back. But if you're in some other position -- for example, if you're on your face -- then the lividity is going to be on the front of your body. Tr. 6 at 1479.

With regard to the gross findings made by the medical examiner in the autopsy, the finding of "cerebral edema but no evidence of hernation or other gross abnormality" (Resp. Ex. E at 3) was of particular interest to Dr. Rorke-Adams:

> Well, he uses the term "cerebral edema" as an explanation or as a description of brain swelling. Now, brain swelling may be a consequence not only of increased water in the brain, but it's also a consequence of increased blood in the brain. And the increased volume of blood in the brain is a consequence of the fact that you don't have proper circulation, so the blood is pooling in the brain. The other thing that makes the brain heavier than normal -- and I presume he called it edema because the brain weighed 540 grams, in contrast to an expected mean of 441 grams -- is that when you have hypoxia -- that is, when the brain is not getting a sufficient amount of oxygen, there is a mechanism that we refer to as autoregulation. And autoregulation is a phenomenon which occurs in all of us if there are changes in the concentration of oxygen coming to the brain, or if there's a decrease in the blood flow. For example, if your blood pressure goes down to 50 or 40 or something, and you're not getting proper flow. So the phenomenon of autoregulation becomes active. And this involves dilatation of the blood vessels in the brain that, many of which are not ordinarily carrying blood, but all of the available blood vessels start opening up to take whatever blood is coming, and to extract from that blood whatever oxygen is being carried to it in order to supply the brain. So when you have a situation of hypoxia, then this phenomenon is going to kick in. So you're going to get more blood in the brain that you ordinarily have, which would then increase the weight of the brain. Now, the importance here, and I suspect that's why Dr. Ross mentioned this, he says, "Cerebral edema, but no evidence of herniation or other gross abnormality." Now, herniation refers to a phenomenon that is very important in terms of cerebral edema or swelling. And that indicates that this has gone on for a while, and that there's a displacement of the brain through the only opening in the skull that will allow for any increased expanse. However, this is a five-week-old baby, and the sutures are still open. So this brain could have swollen much more than your brain or my brain, under the circumstances, without any evidence of herniation. But since Dr. Ross is describing edema, he felt it necessary to point out that there was no herniation.

Tr. 6 at 1514-1516.

Moving on to the microscopic evidence, Dr. Rorke-Adams corroborated with aspects of Dr. Shane's analysis regarding gliosis,[161] which she thought was an acceptably valid finding, although she disputed what he said about the involvement of the oligodendra, saying, "They don't respond to injury in quite the way that the astrocytes respond[, a]nd when you have microglial activation, that's specified separately[; i]t's not under the general umbrella of gliosis." Tr. 6 at 1480-81. She continued:

> And oligodendroglia are a very special kind of cell in the nervous system, whose job it is to form the myelin sheath that I was mentioning earlier that forms the insulation of the axons. So in areas of injury, the oligodendroglia only respond in a very specific kind of way. And in particular, when there is brain edema, the oligodendroglia will become swollen; their nuclei will sometimes disintegrate. But you never use the term "gliosis" in connection with anything that the oligodendroglia are doing.

Tr. 6 at 1481. She herself did not find gliosis in her examination of the slides. *Id*.

The next point of contention between Dr. Rorke-Adams and Dr. Shane's prior testimony regarded the "drop-out" of cerebellar granular cells, which Dr. Shane described, but which Dr. Rorke-Adams did not believe was present. Tr. 6 at 1482. Viewing the same photomicrograph of Thomas Kolakowski's brain slide, which had been shown to Dr. Shane (and which he declared unintelligible), she stated that she could distinguish granular cells. *Id*. She elaborated:

> I can tell this is an infant cerebellum, because the infant cerebellum has a different anatomy than the adult cerebellum.... The infant cerebellum looks quite different from the adult cerebellum, at least up to one year of age. And we have, in the infant cerebellum, a layer of cells out here. This is outside the brain. That part is called the subarachnoid space. This layer of cells over here is called the external granular layer....
>
> ...Now, when you're dealing with a baby, the population of cells in the internal granular layer increases as the population of cells in the external granular layer decreases. Now, there is this peculiarity about the development of the cerebellum in the baby. And these cells out here are still immature, and they're undergoing proliferation; they're increasing in numbers. And their job is to populate the internal

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[161] Dr. Rorke-Adams explained the finding of gliosis, and its significance as a diagnostic tool:

> Gliosis is a reaction of the glial cells in the brain, in particular the astrocytes. And they represent some kind of reaction to injury. The astrocytes are the cells in the nervous system that provide the scars in the nervous system. And so there are certain kind of astrocytes in the brain that when there is some injury to the brain, the astrocytes are stimulated to proliferate. And when, if the individual comes to the attention of a pathologist, we can recognize the histological reaction. And the general term is gliosis.

Tr. 6 at 1480.

> granular cell layer. So for a baby of five weeks of age, this is a perfectly normal cerebellum in terms of population of internal and external granular cells.

Tr. 6 at 1483-84, 1486. Dr. Rorke-Adams then stated her opinion that there was no perceivable drop-out among the granular cells in Thomas Kolakowski's autopsy slides. Tr. 6 at 1487.

On the same section of brain slide, though at a higher magnification, Dr. Rorke-Adams said:

> [W]e can see the detail of the internal granular neurons, and you can see the Purkinje cells, which look quite fine. They are quite normal and show no abnormality.... This is the internal granular cell layer. There is kind of an artifactual cleavage here between these two layers. That's not uncommon in baby brains, because baby brains have a lot more water in them than adult brains. And when you put the tissue into formaldehyde, it dehydrates the tissue and sort of pulls it apart.

Tr. 6 at 1488-89. Whereas Dr. Shane had stated his opinion that the separation in layers of granular cells represented a disease process before death, Dr. Rorke-Adams dismissed that separation as merely "an artifact of the fixation" of the tissue onto the slide. Tr. 6 at 1490.

Regarding Dr. Shane's expert report, which had noted degeneration in varying degrees of the cerebellar granular cells based on loss of nuclear detail and obscure cell margins, while Dr. Rorke-Adams stipulated that, when present, such a conclusion could be made based thereupon, she disagreed that such was the case here. Tr. 6 at 1490. She did not believe the cell margins were obscure or that the nuclei lacked detail, and though that such a statement could not be made in any event without analysis with an electron microscope:

> As I indicated, the problem with these granular cells is that they don't have much of a cell body, so all you see is the nucleus. So that you can't really talk about the cell margins, because that usually refers to the cytoplasm. And since you can't see the cytoplasm very well, unless you look at it with the electron microscope, there's no way that you can make a statement about it.

Tr. 6 at 1491.

Regarding Dr. Shane's reference in his testimony to " perivascular, periglial, perineuronal clear spaces" (he equated the phenomenon with the term "halo"), which he thought "were more prominent than one would see with normal tissue shrinkage," and "associated with edema," Dr. Rorke-Adams had a different position:

> [I]n the brain of a baby the water composition is about 85 percent. And you'll get much more shrinkage artifact in a baby brain than you do in an adult brain. So that in my evaluation of these sections, I didn't see anything to concern myself in terms of what's being described here. There was, however, evidence of edema in these sections. And that was manifested by the phenomenon of the oligodendroglial reaction, which is called acute swelling of the oligodendroglia. And also some of those nuclei were disintegrating. So there certainly was edema there. But the important identification of the edema was not with the halos, but with the actual changes in the cells that were present.

Tr. 6 at 1491-92. The Court asked Dr. Rorke-Adams whether the edema should be classified as "vasogenic or cytotoxic," to which she thought it "very difficult to be absolutely specific," but replied that, "Under these circumstances, I would say it was a vasogenic kind of edema, because of the hypoxic injury to the endothelial cells, the congestion and the edema, the loss of what we call the tight junctions in the endothelial cells." Tr. 6 at 1493.[162]

Returning to the oligodendritic cells, Dr. Rorke-Adams thought "totally false" Dr. Shane's testimony that the oligodendroglia was the "fibroblast of the brain ... mak[ing] scar tissue in an area of injury [and being] more chronic-reactive." Tr. 6 at 1494, citing Tr. 3 at 604. She rejoined, "The scar in the brain is made by the astrocytes. The oligodendroglia do not make the scar." *Id*. She continued:

> As I said, the reaction on the part of the oligodendroglia is primarily in edema, where you have the acute swelling. As I said, their job is to make the myelin sheath, and the astrocytes are the scar tissue of the brain. They're equivalent to the fibroblast. And then the microglia, which were also mentioned, come from the walls of the blood vessels, and they may participate in the scar. But certainly the oligodendroglia cannot be equated to fibroblasts.

Tr. 6 at 1494-95. She added, more generally, that there was no "evidence that Thomas Kolakowski was experiencing a chronic pathologic process," noting that "there was no activation of any of these cells, except for the acute swelling of the oligodendroglia that was part of the edema." Tr. 6 at 1495.

Dr. Rorke-Adams also saw as incorrect Dr. Shane's statement that "turned-on oligodendroglia [indicate] an injury that is subrecent, rather than very recent, because these are fibroblasts. Oligodendroglia don't get turned on immediately; they get turned on a little bit later." Tr. 6 at 1495, citing Tr. 3 at 607. She explained that, "The oligodendroglia are very specific, and they may be activated fairly quickly, as a matter of fact, if you have cerebral edema. And they're not the fibroblasts." Tr. 6 at 1496. Indeed, she reiterated, "The scarring in the brain is a consequence of the activation and proliferation of the astrocytes." *Id*. Also, returning to the finding of edema in the brain, Dr. Rorke-Adams disagreed with Dr. Shane's finding of spongiosis,[163] as part of the

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[162] She elaborated on this same point:

> The brain blood vessels are unique in that they have some attachments between the cells lining the interior wall of the blood vessel, so that they don't allow the movement of a lot of substances which are exchanged easily in other organs of the body. And the major issue here is that in hypoxia, the endothelial cells, which are the ones that are stuck together, come apart, and they allow the movement of fluids and other things into the tissue. So this is primarily the vasogenic edema. The cytotoxic elements are much less obvious here.

Tr. 6 at 1493-94.

[163] Dr. Rorke-Adams explained spongiosis as follows:

> Spongiosis is a histological finding in the tissue in which you have little tiny holes in the tissue. They look like little round circles mostly. And they're a consequence of the fact that the water which was in the, either the interstitial space, that is the space between the cells, or within the cells themselves. And we known that edema can accumulate either within the cell processes, in the processes of the

cytoinflammatory process he postulated, although she did stipulate that there were oligodendroglial changes. Tr. 6 at 1497.

Dr. Rorke-Adams also disagreed with Dr. Shane's characterization of the infant blood-brain barrier, contrasted to that of an older child's, that "everything goes through the [infant's] blood-brain barrier" and that, "It's not as much of a barrier." Tr. 6 at 1498, citing Tr. 3 at 618. She explained her disagreement:

> Well, the blood-brain barrier in a five-week-old infant is quite well developed. I think the transfer of maternal, things from the maternal circulation to the infant circulation is more important, or refers more to other organs of the body, and not specifically to the brain. The blood-brain barrier in a baby allows for certain things to pass, but it has actually, even at the time of birth, a very well developed blood-brain barrier.

Tr. 6 at 1498-99. She elaborated further regarding the selectivity of the infant blood-brain barrier, in contrast to the development at later stages:

> Well, the structure is well developed. The blood-brain barrier in a baby in particular allows the passage for larger protein molecules to travel back and forth. For this reason, for example, the spinal fluid of a baby will have a higher protein content than the spinal fluid of an adult. But there are certain specific things that the baby's blood-brain barrier allows passage. But for all intents and purposes, it's a very excellent, well-functioning blood-brain barrier.

Tr. 6 at 1500. Dr. Rorke-Adams also demonstrated her expertise on this topic by referencing that she was personally selected to review a book specifically on the blood-brain barrier by the editor of the *Journal of Neurology*, a medical journal. Tr. 6 at 1499.

Moving on from her disputes concerning Dr. Shane's review of the autopsy slides, Dr. Rorke-Adams registered her contentions regarding his reliance on the Cinca[164] article, in that it was an inapposite comparison between the subjects of the Cinca studies and Thomas Kolakowski, although she had high praise for the Cinca article itself. Tr. 6 at 1501-02. She first described one distinctive finding, from a neuropathologic perspective, within the Cinca study:

> [A]ll over the cerebral cortex, but mostly in the calcarine cortex -- that's the part of the brain in the back that's involved in visual impulses -- it says there was nerve loss and diffuse proliferation of neuroglia -- that's basically the astrocytes -- on microscopic examination of the brain. And then it talks about neuroglial activation with nerve-cell satellitosis and neuronal loss in formation of neuronophagic

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> astrocytes, or in the myelin sheaths, or in the interstitial tissue. But when the water is removed by the dehydrating process, it leaves these little holes that we call spongiosis.

Tr. 6 at 1496-97.

[164] Pet. Ex. 18, Tab V, I. Cinca *et al.*, *Accidental ethyl mercury poisoning with nervous system, skeletal muscle, and myocardium injury*, 43 JOURNAL OF NEUROLOGY, NEUROSURGERY, AND PSYCHIATRY 143-49 (1979).

> nodules.... [W]hat this refers to is the fact that the cell is undergoing necrosis ... [and this] is a response on the part of the astrocytes and the microglia, which are the garbage-collecting cells of the nervous system, to come and take away the necrotic cell.... So it's engulfment, or the medical term is phagocytosis of the neuron. And that's done by these microglial cells, and then the astrocytes participate in this. It's a very specific histological reaction that is characteristic.... [I]t's unusual because, as I said, most of the time neuronophagia results from viral infections. This is my, the first time I've seen this description in conjunction with, with the mercury intoxication.

Tr. 6 at 1502-04. She was adamant that these indicators of a specific pathologic process were absent from Thomas Kolakowski's autopsy slides, saying, "there was nothing that looked like a neuronophagic nodule." Tr. 6 at 1503. She compared that specific finding to the more "normal phenomenon" of satellitosis:

> All right, the satellitosis is another phenomenon, and that's a normal phenomenon actually. And the cells that are involved in satellitosis are the oligodendroglia, and they collect around the neurons, most particularly in the cerebral cortex and in some deep nuclear structures called the corpus striatum.[165] That's not pathological; it's something that you would expect to see if you were looking at the cortex or the brain. So they somehow or other apparently didn't understand that. But certainly the neuronophagic nodules are quite pathological. But there was nothing like that in this infant's brain....
>
> ...[S]atellitosis, as I said, is a normal phenomenon. So I can pick up any piece of brain cortex or cerebral cortex, and I can see satellitosis. I see that every day.

Tr. 6 at 1504-05. Moreover, Dr. Rorke-Adams stated that satellitosis is unrelated to the neuronophagic nodules, and is not just a phase in what would ultimately result in them: "[T]hey don't have anything to do with each other. Because satellitosis is a phenomenon of the oligodendrocytes, and neuronophagic nodules are formed by astrocytes in microglia." Tr. 6 at 1505.

Another distinction between the Cinca findings and the case of Thomas Kolakowski was the difference in Purkinje cell changes: Whereas, to Dr. Rorke-Adams, Thomas' slides demonstrated "perfectly normal" Purkinje cells, in the Cinca subjects they were "more spared" from necrotic degeneration and pathological damage, although "in certain areas silver impregnation of neurofibrils showed empty basket cells and torpedo-shaped Purkinje cell axons." Tr. 6 at 1506. She described the Cinca findings on this point as follows:

> Well, this is a change that is seen in mercury intoxication in the cases that have, other than this one that we're dealing with, have been reported in literature, and that I've seen myself in the case of mercury intoxication. It's a finding that can only be identified by doing special staining. That is, you can't see these changes on a routine H and E section, which is all we had in this particular case.

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[165] The corpus striatum is "one of the components of the basal nuclei; specifically, a subcortical mass of gray and white substance in front of and lateral to the thalamus in each cerebral hemisphere." DORLAND'S, *supra*, at 423.

Tr. 6 at 1506-07. She provided a photomicrograph of brain cells in a case of mercury toxicity, and described the distinguishing characteristics:

> So in this particular case of mercury intoxication, this is the Purkinje cell layer here. There aren't any granule cells to speak of; maybe a couple over here, but for the most part they're gone. This is a Purkinje cell here, and then you can follow along, and it sends out a process here. And then there's this bulbous swelling here....
>
> ... This is the nucleus of the Purkinje cell here. This is a Purkinje cell here. This is a dying Purkinje cell, and that's a more degenerated one, as well.
>
> Q Okay. So what is the stark black in the middle that's, depending on the way the slide is turned, either above or below that Purkinje cell you were just speaking of?
>
> A This is the torpedo. This is what's described and what's shown in figure 2 of the paper that Cinca wrote, and describes the change in the cerebellum of the 15-year-old boy who died. So you see the connection of the Purkinje cells here, and it's coming down to this thing. And this is what we call the torpedo, and that's a change that is seen on the Purkinje cells in mercury. It also shows that there are no basket cells....
>
> Q Is it fair to compare the Purkinje cell findings in Cinca with the case, with the case of Thomas Kolakowski?
>
> A No, because a silver stain wasn't done to look for any kind of changes of this sort.

Tr. 6 at 1508-10. She reiterated that she did not agree with Dr. Shane's observation of "cell enlargement, indistinct and ragged cell borders, abundant smudgy eosinophilic cytoplasm, and focal nuclear fragmentation" of the Purkinje cells, stating that she did not "see any changes of that sort" nor, indeed, any "degeneration in the Purkinje cells." Tr. 6 at 1510.

Furthermore, on this same point, Dr. Rorke-Adams stated that, Dr. Shane's observations did not jibe with the signs of mercury toxicity anyway (*e.g.*, the "torpedo"), and that even if his observations were accepted, "What he's describing here, if they were present, would represent hypoxic ischemic changes." Tr. 6 at 1510-11. She went on to say that her findings of no effect on the Purkinje cells did not detract from a diagnosis of hypoxic ischemia, "because there are certain sensitivities of the cells through hypoxic ischemia that Purkinje cells may be damaged in hypoxic ischemia. They may not be damaged. It depends on the period of hypoxia, the severity and so on." Tr. 6 at 1511. Elaborating on this point, she stated:

> There are certain neurons in the brain that are particularly sensitive. The neurons in the cerebral cortex, in certain layers of the cerebral cortex, a certain population of cells in a part of the cortex called the hippocampus, and there are specifically the pyramidal cells. The Purkinje cells may be affected. There is a certain other population of cells in the cerebellum in a nuclear structure called the dentate nucleus, which in my experience are more sensitive than the Purkinje cells. So again, it's basically a cascade of cellular involvement, depending on the severity and the length of the insult.

Tr. 6 at 1511-12.

Contradicting the insistent claim of Dr. Shane, Dr. Rorke-Adams stated that it would be impossible to distinguish karyorrhexis (cell nucleus disintegration caused by "some kind of a pathological process") from apoptosis (normal, preprogrammed cell-death, also by nuclear disintegration) using only the H and E stain employed in fixing the brain slides in this case. Tr. 6 at 1512. She said that there is a special stain technique that would need to be used to identify karyorrhexis with particularity, as distinguished from normal apoptosis. Tr. 6 at 1512-13.

Summing up somewhat, Dr. Rorke-Adams stated that there was nothing in her pathological review of Thomas' case that indicated " changes due to a chronic process going on prior to the night of Thomas Kolakowski's death." Tr. 6 at 1513. Nothing in her pathological review suggested "abnormal changes which would have taken more than a few hours to occur." *Id*. Nothing in her pathological review was, taken individually or in concert with other findings, " would be diagnostic of mercury toxicity." Tr. 6 at 1514. In fact, the only toxicity of any kind that she thought the evidence supported was toxicity from "lack of oxygen." *Id*.

She concluded her direct testimony by stating the four points that she believed most strongly militated for a diagnosis of acute anoxic encephalopathy.[166] Tr. 6 at 1516-17. The first finding is "acute congestion and isolated perivascular hemorrhage," which she views as the effect of the process of autoregulation she had described, in which blood vessels dilate "which are not normally carrying the blood," and where " the heart function has antedated the problems" in the brain. Tr. 6 at 1517-18. This causes the circulation of blood to slow below normal levels, which indicates to the pathologist a problem in the circulatory system. Tr. 6 at 1518. "The perivascular hemorrhages indicate that the damage has affected some of the endothelial cells, and opened up the tight junctions that we talked about earlier, and allowed the blood to get out of the lumen[167] of the blood vessel into the surrounding tissue." *Id*.

The second tell-tale finding, for Dr. Rorke-Adams, is the "swelling of myelin sheaths, acute swelling of oligodendroglia, and some pyknosis[168] of these cells, and cerebellar white matter," changes which "describe the changes associated with the edema, or as a consequence of the edema." Tr. 6 at 1518-19. She elaborated on this point more fully:

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[166] Dr. Rorke-Adams explained what she meant by the term "acute anoxic encephalopathy" thusly:

> It means that this is a brain that did not receive sufficient oxygen before death, and produced these changes. Encephalopathy basically is a clinical term, and pathologists never use it unless they have some defining adjectives before the word "encephalopathy." Encephalopathy clinically means that there's some disturbance of higher cerebral function. What I'm saying here, and the manner in which a pathologist uses the term, is that yes, there's some brain dysfunction, and it's most likely due to lack of oxygen. And so that's the accepted term.

Tr. 6 at 1517.

[167] The lumen is "the cavity or channel within a tube or tubular organ." DORLAND'S, *supra*, at 1069.

[168] Pyknosis is "a thickening, especially degeneration of a cell in which the nucleus shrinks in size and the chromatin condenses to a solid, structureless mass or masses." DORLAND'S, *supra*, at 1550.

> And as I indicated earlier, the excess fluid in the brain and cerebral edema can either be intra-cellular -- that is, within the cells -- or between the cells, what we call interstitial. And one of the places where the water can accumulate is in the myelin sheath. And even with an H&E stain, you can evaluate this. Because what happens is that the myelin sheath gets swollen, it gets sort of acculated [*sic*]. And that's a characteristic feature.
>
> The acute swelling of the oligodendroglia, as I said, is the reactive feature of the oligodendrocytes under these circumstances. And the oligodendrocytes, which are normally just a little dark nucleus that are a little bit larger than those internal granular cells that we looked at, then acquire a little halo around them that looks like a fried egg. So you can recognize that, and sometimes you can see some little pinkish material surrounding the nucleus.
>
> Some of these, the reaction can produce damage to the cell, to the point where the nucleus then disintegrates, and the word pyknosis refers to the damage to the nucleus of these oligodendrocytes because of their involvement in this edema. And I saw that in both the cerebral white matter and the cerebellar white matter.

Tr. 6 at 1519-20. In conjunction with this point, Dr. Rorke-Adams voiced her disagreement with Dr. Shane's stated opinion that swelling of the oligodendroglia was "not a finding [he] would rely on" to diagnose hypoxic ischemia, because the oligodendroglia took longer to react, which would require "a very, very long process of hypoxia ischemia," and would be inconsistent with an acute hypoxic-ischemic event. Tr. 6 at 1520, citing Tr. 3 at 686. In contradistinction, it was Dr. Rorke-Adams' opinion that "hypoxia ischemia will provoke edema, so the edema then will lead to acute swelling of the oligodendroglial cells in reaction to that process ... a fairly rapid process." Tr. 6 at 1521. She added that by fairly rapid, she thought that it would only take a few hours for that finding to become evident. *Id.*

Her third diagnostic criterion was the "acute neuronal necrosis of cerebral cortex, especially in layer 2 in one section, corpus striatum and brainstem," which she viewed as "the neurons in various parts of the brain [] undergoing necrosis because of the lack of oxygen." Tr. 6 at 1522. Her fourth finding which she believed to be diagnostic of hypoxic ischemia was that the Purkinje cell layer had separated from the molecular layer and the internal granular layer, even while both granular layers, external and internal, and Purkinje cells, [were] within normal limits." Tr. 6 at 1522. She viewed this finding as "quite striking" as "an artifact that we oftentimes see in baby brains, particularly if they're swollen and have these hypoxic changes." Tr. 6 at 1522.

Looking at Thomas' case from a "global perspective," albeit from her expertise as a pediatric neuropathologist, Dr. Rorke-Adams said she did not see anything that would suggest Thomas Kolakowski's death was caused by anything other than hypoxic, ischemic injury. Tr. 6 at 1522-23. When asked if she would characterize her findings as merely consistent with, or diagnostic of hypoxic, ischemic injury, Dr. Rorke-Adams responded that they were "certainly" consistent, and that there was nothing in the record to lead to an alternate, competing conclusion given the pathologic data and the medical records. Tr. 6 at 1522-23. She added that, "this case taken as a whole is quite consistent with hypoxia ischemia secondary to some interference with respiration, which would fit

in with the history of the co-sleeping," a diagnosis she is "very familiar with," which she makes "all the time at the Medical Examiner's office." Tr. 6 at 1523. Regarding the mechanism of hypoxia, she maintained that the source of the hypoxia was external, and was not "somehow metabolic, or from within the body," adding that there was "no indication of any metabolic disorder or some other disease state." *Id*.

In her mind, the Hepatitis B vaccinations had no "causative role in the death of Thomas Kolakowski." Tr. 6 at 1523-24. In her perspective, "the hypoxic ischemic ischemia was more likely than not, by a preponderance of the evidence, the cause of the death of this child," that "more likely than not, by a preponderance of the evidence, that was brought about by asphyxia," and that the mechanism of the asphyxia is a separate issue. Tr. 6 at 1524.

On cross-examination, Dr. Rorke-Adams admitted that she could not approximate with any precision when Thomas Kolakowski died, and did not engage an invitation from Petitioners to estimate the time of death based on body temperature at the hospital. Tr. 6 at 1525-26. To do so, she opined, was a "very tricky business" that was "actually the realm of forensic pathologists," and that, even in her capacity as a forensic neuropathologist, she is not called upon to perform that analysis.[169] Tr. 6 at 1526. She added later, when Petitioners raised the subject again, that "the rules are very difficult, because there are many, many factors that have to be taken into consideration[, a]nd the bottom line in the forensic pathology textbooks is that it's a very unclear, unscientific kind of problem, and there's no way that you can really be certain." Tr. 6 at 1561-62. Another area upon which she admitted she was unqualified to opine was in answering the question whether persistent cardiac electrical activity was "consistent with a death that occurred two hours earlier." Tr. 6 at 1528.

When asked how she knew that Thomas Kolakowski had not died from mercury toxicity if she had not read much of the toxicology literature filed in this matter, she replied:

> Well, I'm familiar with mercury intoxication and what it does to the nervous system. I didn't review the [numerous articles], but I certainly have a knowledge of mercury intoxication. There is a very fascinating book that was written about the contamination of the Minamata Bay in Japan. I have a copy of that book. I've read it, I've read the textbooks. I deal with toxicology in the course of my work, so I have a general knowledge of what mercury does to the brain.

Tr. 6 at 1528-29.

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[169] Dr. Rorke-Adams added further explanation on this point as follows:

> [T]here are many, many different factors that are involved in using body temperature as a determination of time of death, one of which is the ambient temperature. Another factor is what was the temperature of the individual before he died. The third one is the amount of clothing on the individual. So there are many, many things. And in my reading of the forensic textbooks dealing with this issue, the bottom line is that it's a very risky business to try to be specific in terms of those, relating one to the other.

Tr. 6 at 1526-27. She added, when asked, that Dr. Gilbert-Barness is a pediatric pathologist and Dr. Rorke-Adams was unaware of "how much forensic work she's done." Tr. 6 at 1527.

Petitioners' next line of questioning challenged Dr. Rorke-Adams on the clinical symptoms that would be expected in a case of mercury toxicity, and how those might be identified in a newborn. The first discussed was cerebellar ataxia, a motor imbalance and lack of coordination that makes for imbalanced walking and unsteady, shaky movement of the arms and hands, which Dr. Rorke-Adams would not apply to Thomas since he was so young, and not mobile or developed enough to note a change. Tr. 6 at 1529. Another indicium would be subjectively-felt anxiety, which would be difficult to discern with any specificity or objectivity in so young an infant, only to note excessive crying and derive anxiety therefrom. Tr. 6 at 1531. The same difficulty in objective observation is even more present with the indicia of visual dysfunction, personality changes, memory loss, and dementia. Tr. 6 at 1531-32. Motor and sensory motor neuropathy could be measured clinically through a neurological examination to determine difficulty in moving extremities or sensory loss (*e.g.*, a pin-prick test). *Id*. The remaining indicium was tremors, which were never clinically described by a medical professional, but was the term used by Thomas' mother in her affidavits to describe a physical phenomenon she claims to have witnessed in Thomas during his brief terrestrial sojourn. Tr. 6 at 1530. Dr. Rorke-Adams made sure to point out that there was "no indication of the pediatrician being told that the baby had tremors," and that "in the hospital records, the mother said that the baby only had a head cold, and nothing else was going on ... she didn't say anything about tremors there." *Id*, citing Pet. Ex. 5 at 4. Dr. Rorke-Adams did concede that the mention of tremors was made "repeatedly" in Mrs. Kolakowski's affidavits, composed in preparation for this litigation. Tr. 6 at 1530.

Later, Dr. Rorke-Adams was called on again to discuss the term "tremors" that was mentioned in Mrs. Kolakowski's second affidavit, which sought to describe the actual physical phenomenon that she referred to by that term. Tr. 6 at 1556. Grouped in with the mention of tremors was what Mrs. Kolakowski perceived as a weak or nonexistent startle reflect in Thomas, which Dr. Rorke-Adams conceded could be a neurological sign, before noting that she was not a pediatric neurologist, and could not therefore opine with particularity on what was a normal startle reflex at five weeks old. *Id*. Dr. Rorke-Adams remained quizzical about the affidavit reference to "body tremors," because tremors "are usually in an extremity," whereas "if the whole body is moving, then I would interpret that to be that the child is having a seizure ... I've never heard of body tremors." Tr. 6 at 1557. She did agree that such symptoms could be neurological. *Id*. A little later, Petitioners returned again to the affidavit about tremors, which describes them as "episodes where his body would stiffen, and then the involuntary movements would begin," lasting a few minutes time, adding that, when Mrs. Kolakowski would try to comfort Thomas during these episodes, "the trembling would continue, and stop when it had run its course." Tr. 6 at 1560-61. Dr. Rorke-Adams reiterated that, to her, this phenomenon sounded "more like a seizure." Tr. 6 at 1561.

The next topic of cross-examination delved deeper into Dr. Rorke-Adams' prior discussion of autoregulation, with Petitioners asking whether the stressor of hypoxia is what triggers the autoregulation, to which Dr. Rorke-Adams responded:

> The hypoxia is the first thing to occur. And the heart needs blood, just as the brain needs blood. So the biochemical effect in the brain is to open up the blood vessels. But if the heart doesn't get adequate blood, then it can't pump efficiently to increase the blood flow. And its structure, its muscle cells are also not getting sufficient

> blood, so that the effectiveness of the heart is diminished, because it's also compromised.

Tr. 6 at 1533-34. As to how long this process would take to transpire, Dr. Rorke-Adams stated that it would depend on how severe was the hypoxia. Tr. 6 at 1534. When pressed to estimate how long the process took in this case, Dr. Rorke-Adams replied:

> I can't give you a specific time, but it probably took place over a period of time, because this wasn't somebody clapping their hands over his face and cutting off all of his oxygen. It was probably a partial interference with his ability to breathe. And so again, it would be purely a matter of speculation.

Tr. 6 at 1534. Dr. Rorke-Adams said it was not speculation to say that Thomas' airway was partially, not completely obstructed, because if his airway had been completely obstructed, she "would expect him to die immediately, and then we wouldn't see any of these changes," which "take a while" to develop. *Id*. Overall, however, she stressed that the actual duration could only be estimated as a variable if the degree of hypoxic obstruction was a known quantity, but that if both were unknown variables, neither could be estimated with any specificity. Tr. 6 at 1535. It certainly could have taken place completely within the four hours between when Thomas fell asleep and when he was discovered, but she declined to narrow her estimate more precisely. *Id*.

Next, Dr. Rorke-Adams was asked to identify any pathological findings in the heart tissue that heralded ischemia, to which she answered that no special staining procedures or analysis was done on autopsy, and that she could not render an opinion on that question since it had been so long since she had performed cardiac pathology 45 years previous, such that her cardiac pathology knowledge was not up-to-date. Tr. 6 at 1535-36. Asked whether cardiac arrest could antecede anoxic, ischemic injury, Dr. Rorke-Adams agreed that it could, but that several of the findings present in this case would not be present, because, "If the heart stops beating just suddenly and the individual dies, you don't see anything anyplace in the organs of the body, including the brain. It takes a while for the pathological features to develop." Tr. 6 at 1537. When asked whether a slow degradation of heart function could lead to hypoxic ischemia, Dr. Rorke-Adams described what is referred to as "near-miss SIDS" as the situation where "a baby [] stops breathing and whose heart stops, but somebody is there," and the baby is resuscitated, but who never regains consciousness and ultimately expires. Tr. 6 at 1537-38. She said that in those cases, the histological changes within the brain are observable on autopsy, because there was time for those processes to transpire, whereas in a shorter time frame, they are not. Tr. 6 at 1538.

Dr. Rorke-Adams again voiced her disagreement with Dr. Shane, regarding whether the myocardium demonstrated pathological changes, when she stated that "[t]here were no changes in the myocardium," not even any changes caused by the hypoxia she postulated. Tr. 6 at 1538. She did agree with Dr. Shane that the brain showed evidence of cerebral edema upon microscopic examination, saying that she observed such evidence on most of the brain slides. Tr. 6 at 1539-40. She also disagreed with Dr. Shane about the evidence of the chronic pathological process that Dr. Shane described. She said that an ongoing inflammatory reaction in the brain would cause pyknosis. Tr. 6 at 1541. When Petitioners asked whether she would expect to find microglial activation if the brain was undergoing an inflammatory reaction, she qualified:

> Depends on the time and the extent. One of the peculiar things about hypoxic ischemic injury in the brain is that you can have widespread disintegration of the cells, and very little in the way of any kind of inflammatory reaction whatsoever. I've never heard anybody give a proper explanation for this. I don't know why that's the case, but that's what we observe. So the reaction to the cell death basically is not on the part of the inflammatory cells, but it usually comes after the cells have died and the astrocytes come in to, and some of the microglia will come in to take care of the removal of the dead cells and the presence of the scar, or form the scar.

Tr. 6 at 1541-42. She stipulated that "sometimes in hypoxic ischemia, [she might not] see microglial activation, [and she] just [does not] know why." Tr. 6 at 1542. On the same general point, Dr. Rorke-Adams clarified an issue of classification. When asked by Petitioners whether the term gliosis would include the inflammatory activation of both astrocytes and microglia, Dr. Rorke-Adams stated that the term refers only to astrocytes, and that the microglia are separate. Tr. 6 at 1542-43. Dr. Rorke-Adams stated that neuronal necrosis takes "an hour or two" in order "to appear after a hypoxic ischemic event," but that did not mean that she would expect the appearance of microglia "[w]ithin a couple of hours." Tr. 6 at 1543-44. She explained that, most commonly, "you can have a tremendous amount of neuronal necrosis and not see very much microglia at all." Tr. 6 at 1544. When Petitioners tried to analogize microglia to the macrophages within the bloodstream, as they are "the macrophages of the central nervous system," Dr. Rorke-Adams was careful to distinguish that they are different in important respects:

> Well, again, the central nervous system is unusual, and you can't compare it with another organ of the body, because you have a different cell population. And unless you have some kind of an infectious agent, you don't get the same kind of inflammatory changes in quite the precise way in the brain that you do in another organ of the body. If you have some kind of injury, you have the outpouring of chemical substances called cytokines, which stimulate the activation of certain reactive cells. But when you look at the sections, you don't see all of the various cell types in the brain that you would see in the liver, or the lungs, or the spleen, or whatever. It's a different kind of a histological reaction. [I]n most of the acute anoxic encephalopathies that I see, there's not much of a reaction at all in the early stage. You see the cell undergoing the necrosis, the pyknosis, the karyorrhexis. And then they just kind of disappear. That's another peculiar phenomenon that I've never been able to understand. Then eventually, once the cells have disappeared, you start seeing the activation of the astrocytes from the histologic point of view, and you see the gliosis.

Tr. 6 at 1545-46.

Next, the cross-examination turned to the interpretation of the Purkinje cells, and the separation that Dr. Rorke-Adams noted. She described the cell-layer separation as "a finding that's very common in the baby, that we see in the baby brain when there is edema," but added that it was seen by her as an exaggerated artifact, "an artifact that's accentuated when you have an edematous brain." Tr. 6 at 1549. In plain terms, the brain tissue, especially of an infant, tears when it is dehydrated in formaldehyde, but typically does so in a predictable pattern, to a predictable degree;

when the tear is more severe, it is evidence of greater fluid content, as in the case of edema. In this, she was in agreement with Dr. Shane:

> Well, the dehydration is from the dehydration that is produced by the formaldehyde. So it removes the water from the tissue, and it accentuates the spaces. For example, in Dr. Shane's report he talks about perineural and perivascular halos. That's all because of the shrinkage artifact. And the more swelling you have, the more dramatic those things are.... It's accentuated in cerebral edema, but it can be just an artifact unassociated with edema in a baby brain.

Tr. 6 at 1550.

The next phase of cross-examination showed that Dr. Rorke-Adams possessed the erroneous understanding that either ethyl or methyl mercury were inorganic, when actually they are both organic forms of mercury. Tr. 6 at 1551-52. When asked to discuss the differences in organic and inorganic forms of mercury, in the relative harm they can cause, Dr. Rorke-Adams replied that the source she had read on mercury toxicity noted that they cause different neurotoxicity, but that in other contexts they were lumped together. Tr. 6 at 1552-53. As pointed out by Respondent's objection at that point, that topic is toxicologic, not neuropathologic, and, while it did demonstrate an error on Dr. Rorke-Adams' part, it did not vitiate her probative weight in the Court's mind, because she seemed very competent on the topic for which she was offered: neuropathology.

Dr. Rorke-Adams explained her disagreement with Dr. Shane concerning the blood-brain barrier, in that she believed it was "intact at the time of birth," even though "it allowed for the movement of various proteins in the baby brain, whereas the blood-brain barrier doesn't allow that in the adults." Tr. 6 at 1554-55.

Petitioners circled back around to asking Dr. Rorke-Adams about the clinical record, to determine if any of the symptoms recorded were neurological, or at least potentially so. Dr. Rorke-Adams said in response to the symptoms raised (reluctance to sleep or nurse, passivity, insomnia, anorexia, the "tremors" and the putatively muted startle reflex) that they could be neurological, or they could be related to many other things, because they were mostly nonspecific. Tr. 6 at 1558-59. Dr. Rorke-Adams also responded by reference to what is absent from the medical records:

> [I]f you go back to the Exhibit 5, where she's giving a history when the child comes into the hospital essentially dead, she says, "Mom says child had a head cold and nothing else going on." So if she was so worried about these things, I should think she would have told the doctors in the hospital when her child is in serious condition that, you know, I noticed all these terrible things happening. But there's no indication of that.

Tr. 6 at 1559.

Getting back to the central issue of proximate causation, Petitioners asked whether hypoxic ischemic encephalopathy was caused only by asphyxia, to which Dr. Rorke-Adams replied that, of course, there are other potential causes for a lack of air, such as when suffering from shock or other disorders, including pulmonary edema. Tr. 6 at 1563. Petitioners then asked if congestive heart

failure was somewhat analogous to pulmonary edema, which moved beyond the topic area of neuropathology. *Id*. Dr. Rorke-Adams stipulated that "pulmonary edema is oftentimes a consequence of congestive heart failure," which, if it were sufficiently severe, could lead to a hypoxic ischemic encephalopathy. *Id*. However, in this case, Dr. Rorke-Adams did not see any chronic abnormal findings, only acute ones. *Id*. And although the term acute can mean anything occurring within a one- or two-day span, in this case there was a very brief time interval–four hours or less, because if it had extended earlier, beyond the four-hour period in the early morning hours of 25 January 1999, Thomas would have manifested much more significant clinical symptoms. Tr. 6 at 1563, 1569-71. Also, Dr. Rorke-Adams stated that the pathologic process(es) she described could all transpire within a four hour interval. Tr. 6 at 1564.

Finally, on redirect examination, Dr. Rorke-Adams clarified that she did not believe that pulmonary edema precipitated the hypoxic ischemic injury as a first cause, but was a concurrent finding from a similar first cause of whatever mechanism (physical, not metabolic) caused hypoxia. Tr. 6 at 1564-65. She explained why:

> Well, because there was not isolated pulmonary edema in this case. There was evidence of pleural effusion, which refers to fluid in both sides of the chest cavity, which is a sign of, or it can be one of the signs of heart failure. There was severe congestion in the various organs, and a pulmonary edema was simply one aspect of the failing heart. So we had a combination of findings.

Tr. 6 at 1565. Likewise, when asked if she had seen any evidence of congestive heart failure in the microscopic slides, the autopsy's gross findings, or in the clinical records, she answered:

> [T]he bilateral pleural effusions are a characteristic, can be a characteristic feature of congestive heart failure. The heavy organs, the lungs, well, the normal weight that one would expect for a five-week-old is 68 grams; this baby's lungs were 180 grams, which is quite an increase. The liver was increased in weight. The spleen was almost double the size. All of these are signs of congestive heart failure [from a hypoxic injury].

Tr. 6 at 1565-66. This led the Court to press Dr. Rorke-Adams on sequence of causation and events:

> THE COURT: Can one say which came first? In other words, could you have had the congestive heart failure that led to the pulmonary edema, to the oxygen deprivation, and therefore to a hypoxic ischemic injury? Or would you have to say the hypoxic ischemic injury came first that brought about pulmonary edema and heart failure? Or can one say that?
>
> THE WITNESS: I think in this case, the hypoxia ischemia produced simultaneous damage to the heart and the brain because of the lack of oxygen. What happened was that the lack of oxygen produced the heart failure, which started to work on the brain. And also, as it continued, it led to the obvious manifestations of the pulmonary edema, the pleural effusions, and the enlargement, congestion of the visceral organs.
>
> THE COURT: And upon what can you base your conclusions that the hypoxic ischemic injury occurred first, bringing about the pulmonary edema and the cardiac

arrest; rather than the cardiac arrest bringing about the pulmonary edema, and then the hypoxic ischemic injury?

THE WITNESS: Well, we don't have any primary cardiac disease to explain why the heart isn't functioning properly. So that something had to produce some inadequacy of the heart to function as it should, to lead to generalized ischemia that would affect the brain, and then the continued functioning of the heart. And then the pulmonary edema and the effusions would be a consequence of the failing heart.

Tr. 6 at 1566-67.

Respondent asked Dr. Rorke-Adams to put this case's diagnosis in context of her experience:

Q How many autopsies have you done in which you diagnosed the cause of death as hypoxic ischemia?

A Oh, several thousand.

Q Okay. Is there anything about the Thomas Kolakowski case that distinguishes it diagnostically from those other cases?

A No.

Tr. 6 at 1567.

Lastly, the Court asked Dr. Rorke-Adams some clarifying questions regarding her offered opinion:

THE COURT: Okay ... What the Court wants is to be certain that it understands your perspectives in the delineation of what occurred. And are you saying that the congestive heart failure and the pulmonary ... edema, were precipitating causes to the hypoxic ischemic injury? Or that they were derivative from the hypoxic ischemic injury, and part of the agonal process? If you can say.

THE WITNESS: They were derivative to the hypoxia ischemia. And the changes that we saw occurred simultaneously affecting the heart first, and then the brain. And then the failure of the heart led to the pulmonary edema and the effusions, the pleural effusions, and the congestion in the visceral organs.

THE COURT: Okay. But am I to understand, then, that the hypoxic ischemic is what caused the other items? In other words, the congestive heart failure presumably caused the pulmonary collapse? Or not?

THE WITNESS: Yes. The stimulus for -- the whole process began because of the hypoxia.... The hypoxia then caused the cardiac insufficiency and the brain damage, and the cardiac insufficiency caused the pulmonary and visceral changes.

THE COURT: So from your perspective, it would be fair to say that the congestive heart failure and pulmonary issues are part of the agonal process?

THE WITNESS: Well, the terminal process.

THE COURT: Terminal process.

THE WITNESS: Yes.

THE COURT: And that they were triggered by the hypoxic ischemic injury.

THE WITNESS: That's correct.

Tr. 6 at 1572-73.

8. Additional Testimony of Dr. Shane, Dr. Rorke-Adams, and Dr. Gilbert-Barness

Putatively as rebuttal evidence, at the close of Dr. Rorke-Adams' testimony, Petitioners recalled Dr. Shane to the stand. Tr. 6 at 1574. This was solely for the purpose of offering and discussing a series of glossy, color photomicrographs Dr. Shane took of Thomas Kolakowski's autopsy slides, at varying magnification levels. *See*, *e.g.*, Tr. 6 at 1575-77.

Dr. Shane's purpose for this additional testimony was to dispute Respondent's experts on pathology in the interpretation of the slides, such as whether there were inflammatory cells in the heart or brain, or whether the cerebellar granular cells had "dropped out," or whether the Purkinje cells demonstrated an ongoing pathologic process. *See*, *e.g.*, Tr. 6 at 1580, 1587, 1595. Later, Respondent's pathology experts were recalled to argue that he was misreading the slides, or making much of very equivocal indicia that were more likely attributable to the process of setting the tissue to the slide. *See*, *e.g.*, Tr. 6 at 1642-43.

Ultimately, the Court does not base its decision on this testimony, because it is extraneous to the Court's task. It was helpful in that the pathology experts demonstrated more concretely what they used to formulate their opinion, but the content and rationale of their respective opinions remained precisely the same as when they had first testified. *See*, *e.g.*, Tr. 6 at 1662.

Most importantly, at no point during his rebuttal testimony did Dr. Shane postulate a mechanism for how two doses of thimerosal-containing vaccines caused the sudden death of Thomas Kolakowski, or indeed, how the amount of thimerosal in vaccines received by children in these United States could cause sudden death in any infants or children. That was primary reason for the testimony taken in this matter, and that remained patently absent, even into this exercise of slide interpretation. Without a framework of a theoretical mechanism, it is ultimately irrelevant whether a handful of inflammatory cells is sufficient to characterize an inflammatory response, a point upon which Dr. Shane seemed to be very focused.

To be clear, the Court does not exclude such testimony, but did not find it probative of the ultimate issues presented. It was of sufficient relevance to be admissible, but not sufficiently probative to be useful to the Court's decision-making.

D. Post-Hearing Submissions

At the conclusion of the hearing, the Court ordered briefing by the parties, whose arguments are summarized here.

1. Petitioners' argument is that their experts' testimony satisfies the elements sufficiently to prove causation, and their support for such argument is to repeatedly summarize the points made in the testimony: from the claim that Thomas received 30 times over what would have been a safe dose, to the purported analogy between the data from the Cinca and Dahhan studies and Thomas' clinical records and pathological findings. At one place in their closing brief, Petitioners argue that Thomas received, in each vaccination, thirty times "the EPA's safety limit," as if the reference dose were a "limit" beyond which harm would likely result, a dubious proposition and an unpersuasive argument.

2. Petitioners cited several cases where this Court has found vaccine-related injuries and death following Hepatitis B vaccine, but did not cite to any which found that the relevant injury or death was caused by thimerosal, which is the central issue in this matter.

3. Regarding the topic of pathology in particular, Petitioners argue that the medical examiner and the pathologists he consulted were dumbfounded about what caused Thomas' death, but that their "only suspected culprit" was the Hepatitis B vaccines administered to Thomas.

4. Petitioners' arguments are rendered confusing, if not incoherent, in that they argue that several conditions and circumstances obtain; however, in the absence of a proffered theoretical mechanism, these points cannot avoid seeming haphazard and irrelevant. Examples of this are pathological findings from Dr. Shane that support myocarditis, discussions about "QT intervals," or certain similarities between the subjects of the Cinca study and Thomas Kolakowski.

5. Petitioners argue that the inflammatory reaction mentioned by Dr. Connor and Dr. Shane was immunologic in nature and origin, but Petitioners offered no evidence, and certainly no expert testimony, on immunologic issues bearing on this matter.

6. Respondent's primary argument is that Petitioners failed to carry their burden of proof on both the can-it and did-it portions of proving causation–that "Petitioners have failed to offer reliable scientific evidence to support their claim that ethyl mercury, in the amounts contained in two Hepatitis B vaccinations, can cause mercury toxicity, adverse cardiac effects, immune dysfunction, or sudden death," and "have failed to offer reliable scientific evidence that the Hepatitis B vaccine caused or contributed to the death of Thomas Kolakowski."

7. Respondent argues that, assuming that Petitioners have failed to carry their burden of proof on causation, "Respondent, therefore, bears no burden in establishing an alternative cause of death," although, Respondent argues, the evidence does preponderate to a finding that "Thomas died as the result of accidental asphyxiation." Respondent (correctly) relies upon the Federal Circuit's

de Bazan v. Sec'y of HHS opinion[170] as support for using alternative causation evidence both as evidence of a "factor unrelated" under § 13(a)(1)(B), and as evidence to challenge Petitioners' proof during Petitioners' case in chief.

8. Respondent argues that Petitioners' reliance on the filed medical literature is too attenuated, either because of the form of mercury studied, or the dosages involved. Respondent argues that the only support for Petitioner's argument is the (scientifically unreliable) opinions of Petitioners' experts, which themselves are founded only in the *ipse dixit* of those same experts, or one another.

II. ULTIMATE FINDINGS OF FACT

The Court's task now is to analyze the differences between the opinions offered to determine whether Petitioner has established a plausible theory of medical causation and a logical sequence of cause and effect, having occurred in a medically appropriate time frame, which is biologically plausible to tie together the factual sequence and explain Petitioner's injury. *See Althen v. Sec'y of HHS*, 418 F. 3d 1274, 1278 (Fed. Cir. 2005); *Pafford v. Sec'y of HHS*, 451 F. 3d 1352, 1355 (Fed. Cir. 2006), *rehearing and rehearing en banc denied*, (Oct. 24, 2006), *cert. den.*, 168 L. Ed. 2d 242, 75 U.S.L.W. 3644 (2007); *Walther v. Sec'y of HHS*, 485 F. 3d 1146 (Fed. Cir. 2007); *de Bazan v. Sec'y of HHS*, 539 F. 3d 1347, 1352 (Fed. Cir. 2008).

Before the Court assesses the relative probative weight of each party's expert witnesses, it should first assess the weight assigned to the "tremors" described by Mrs. Kolakowski. This Court has always been reticent to supplant medical records with a mere testimonial affidavit, especially given the weight afforded medical records and the statutory command that a petitioner may not prevail based solely upon his own claims, without further support. Here the medical records are not only silent upon the matters averred to in the affidavits, the two are at odds. Whereas Mrs. Kolakowski reports that Thomas' startle reflex never properly developed, more that one medical record reports that it was patent and observed before Thomas was discharged from the hospital following birth. She reported the presence of tremors from the day of the first administration of Hepatitis B vaccine, Thomas' second day of life, but was spotty in her description of what she witnessed. When ordered by the Court to fill in the details of what she saw, what she saw was most likely not tremors at all, a point made most tellingly by Petitioners' own pediatric neurologist, who downgraded the significance of the affidavits' testimony about "tremors." Dr. Rorke-Adams, a neuropathologist, corroborated the pediatric neurologist's opinion. Most significantly, though, the affidavits' mention of tremors that began the very same day as the first Hepatitis B

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[170] 539 F. 3d 1347 (Fed. Cir. 2008). Respondent quoted the relevant section of that opinion:

> The government, like any defendant, is permitted to offer evidence to demonstrate the inadequacy of the petitioner's evidence on a requisite element of the petitioner's case-in-chief. [de] Bazan has identified no statute, regulation, or precedent that indicates that the special master is required to only consider the petitioner's evidence as to whether the vaccine was a cause-in-fact.

539 F. 3d at 1353.

vaccination–while Thomas was still in the hospital following birth–is belied by medical records that same day specifically stating that there were not tremors present or observable. Also, for symptoms so severe that Mrs. Kolakowski could not rouse Thomas from their effects, the medical records do not reflect complaints of such phenomena in their recorded history, and the histories taken were not perfunctory or cursory. The Court is left to conclude that whatever Mrs. Kolakowski referred to by her use of the term "tremors" was not medically relevant or intelligible for the Court's decision in this case.

As stated *supra* at 3-4, medical records in pursuit of treatment enjoy a weighty deference by the Court. Where medical records are silent or ambiguous, fact witness testimony may certainly be useful in filling in marginal details. Also, in the rare event that the medical records display patent errors, or are otherwise contradictory, fact witness testimony that is clear, cogent, and consistent may overcome discrepancies therein. None of those conditions obtain here, and the medical records as they are written are accepted as accurate, fact witness testimony notwithstanding.[171] Any reliance upon the so-called tremors is unpersuasive, and unsupported by the factual record.

Moving on to the medical issues presented, the Court will assess each medical area separately, then as a whole. The Court begins with the toxicology issues.

The issue presented by the toxicology evidence revolves around dosage, that the amount of ethyl mercury contained within the infant immunization regimen, and two Hepatitis B vaccines in particular, have enough mercury to cause adverse outcomes–and ultimately death in particular. Dr. Lucier's testimony was interesting, but its focus was on the regulatory decision-making that uses safety factors where scientific knowledge is spotty or absent. Dr. Lucier's testimony contained errors in basic scientific calculation, and he argued by analogy beyond the point of attenuation. His testimony was not based on the medical literature as a whole, but grasped onto isolated fragments as prooftexts to prove what no scientific study has: that the amount of mercury in thimerosal-containing vaccines is dangerous, let alone deadly.

A large part of the problem running as a current throughout all of Dr. Lucier's testimony is application of regulatory criteria to the realm of general and specific causation, the Court's focus herein. Of course, Dr. Lucier's orthogonal approach arises from the fact that he is a toxicologist, not a medical toxicologist. He is an expert on chemical properties and processes, not an expert on the effects of those chemicals in the human body, or the treatment therefor. Beyond the matter of his educational expertise, his professional experience has focused on measuring biologic effects in relation to the dose of chemical exposure. He has not really studied physiologic effects in humans, and in his experience overseeing the regulatory reference dose, he and his regulatory coterie assumed the relationship measured by the Faroe Island researchers in calculating a reference dose for methyl mercury. In that pursuit, where science did not provide an answer, the regulators (for perfectly good reasons!) assumed relationships and quantitative values beyond what any data provided. As Dr.

[171] Respondent's argument is well-taken that "The Vaccine Act does not permit a special master to make findings 'based on the claims of a petitioner alone, unsubstantiated by medical records or by medical opinion'" (citing 42 U.S.C. § 300aa-13(a)(1)).

Brent pointed out, however, what is good practice for regulators does not make a conclusion scientifically valid or evidence-based if the question is determining cause and effect relationships.

Another similar problem with Dr. Lucier's testimony, and one that is probably a result of his not being a medical toxicologist, is that he never makes any toxicologic associations regarding a mechanism whereby ethyl mercury causes harm by physiologic processes. His discussion amounted to a roundtable discussion of mercury's properties and forms as a general question, which possessed only tangential relevance to the questions presented by the case at hand. At best, Petitioners are left with extrapolating medical conclusions from his testimony, because none of his testimony that was based on substantive proofs was relevant to the question of causation, and what statements he did make on the question of causation were conclusory and unsupported by the evidence. A rather damning example of this problem was that Dr. Lucier never even undertook the analysis of whether, from a toxicological standpoint, the ethyl mercury contained in two Hepatitis B vaccines actually *did* cause the injury to and death of Thomas Kolakowski. Even if Petitioners were to provide a nuanced argument to connect Dr. Lucier's meandering musings to cardiologic or pathologic evidence, so as to provide a toxicologic basis for general ("can it") causation, there is still entirely missing from the record any opinion from the area of toxicology that addresses specific ("did it") causation in *this* case.

A large point of confusion, that apparently confused Petitioners in their brief, concerns the safety reference dose. This dose is an amount of the substance which, if no more than that amount is consumed daily (on average), adverse outcomes are all but certain to be avoided. In every instance, it assumes a value from the most sensitive data points recorded, and adds orders of magnitude whenever there is an ambiguity. Suffice it to say that it is a logical fallacy ("affirmative conclusion from a negative premise") to use the following statement: "IF the reference dose of methyl mercury is not exceeded in average daily consumption, THEN no adverse outcomes will result," in order to conclude through reformulation that "IF an amount of ethyl mercury over the reference dose of methyl mercury is consumed on two separate days of a series, but not every day or in very large amounts, THEN adverse outcomes will result." Aside from this obvious fallacy, there is also present in that formulation the "quantifier-shift fallacy" of attributing to ethyl mercury the precise characteristics of methyl mercury, mercury chloride, or elemental mercury: Even if ethyl mercury is a form of mercury (which in some dosage is toxic or lethal, depending on its form), and shares characteristics with all forms or mercury (most of all methyl mercury as another organomercurial), it does not logically follow, as a deductive exercise, that ethyl mercury shares every characteristic of the others. If it does not, then part of proving that ethyl mercury damages infants would include an inductive course of determining exactly what ethyl mercury is capable of, and its precise characteristics as a substance. It is improper even to begin from the same reference dose as methyl mercury, for that matter. Dr. Lucier subjectively weighed attributes that are qualitatively, not just quantitatively, distinct, in order to arrive at his judgment that ethyl mercury is just as toxic as methyl mercury. The Court does not accept such a premise as proven, without some objective standard to assess his balancing of toxicity factors. Moreover, that opinion is not corroborated by the medical literature filed in this case. Dr. Lucier admitted as much when he said that regulatory agencies, in the interest of efficiency, will study chemical groups instead of individual chemicals, and their practice in doing so is to pick the most toxic of the group to study, and assign a reference dose. Noticeably, not one of the regulatory agencies discussed herein ever thought to

study ethyl mercury for this purpose instead of methyl mercury. Despite this, Dr. Lucier's opinion concerning the toxicity of ethyl mercury in thimerosal was based throughout on the supposition either that ethyl mercury was the same as methyl mercury, or that it was even more toxic.

To be clear, the medical literature provided herein establishes that the organic ethyl group that gives ethyl mercury its name is easier to strip from the mercury atom than is the methyl group that is methyl mercury's namesake. Petitioners' main argument for the premise that ethyl mercury proves to be more toxic is that, since it is deethylated easier, and therefore more readily converted to leave inorganic mercury (mercury chloride, the inorganic mercury salt) as an end-product, it has the potential for more damage because inorganic mercury is difficult or impossible to eradicate from the body, and the brain in particular. That argument would be most persuasive if the facts involved a prolonged exposure, because if blood levels were kept regular or constant at a high level, the amount of ethyl mercury available to deethylate would be sufficient to populate the body (and ultimately, we are to assume, the brain) with inorganic mercury, above the conversion rate of methyl mercury. However, that is surely not the case. The Burbacher data make abundantly clear that ethyl mercury is eliminated from the bloodstream very quickly, especially when compared to methyl mercury. If ethyl mercury is consumed in light to moderate (non-lethal) doses, the medical literature preponderates to a conclusion that the body could eliminate it before it could be metabolized and deethylated into inorganic mercury. In contrast, methyl mercury is much more persistent in the blood. This is another reason why it is improper to use the reference dose of methyl mercury–a substance that long persists in the bloodstream–as the reference dose for ethyl mercury, which is quite different in that regard.[172]

Of course, all of this discussion is quite extraneous to the fact that Dr. Lucier never postulated a toxicologic theory by which the dosage of ethyl mercury in vaccines could cause toxicity or sub-toxic effects. That was the primary reason for calling him to the stand, and on that point, his testimony was abjectly wanting.

In contrast, Dr. Brent was a medical toxicologist, and his expertise not only permitted him to discuss the effects of toxic chemicals, it allowed him to state what effects would correspond with a given dosage of a particular chemical. He pointed out the scientific invalidity of using the reference dose of methyl mercury to assess the threat of harm from ethyl mercury in thimerosal-containing vaccines. Dr. Brent was insistent that the differences between ethyl and methyl mercury were stark and qualitative, such that the qualities of one could not be assumed for the other. He pointed out that the reference dose is based on a daily average, not a maximum daily amount for a single dose. He also pointed out the possible distortions that might result from using data from only the most adversely-affected children who had been prenatally exposed to methyl mercury as a basis for setting a safe level for children and adults, especially when the primary point of distinction between that population and a population without any measurable adverse effects (despite a similarly high consumption of methyl mercury) was a confounding factor of PCBs in the former group's

[172] Based in part on this rationale, the Burbacher study concluded that the reference dose for methyl mercury was inapplicable to ethyl mercury.

diet.[173] Dr. Brent also quashed Dr. Lucier's claim that the cardiac system was the most sensitive aspect of physiology to mercury toxicity, as cardiac problems were not even considered as a measurable endpoint for studying mercury toxicity. Indeed, the Court's question when reviewing Dr. Lucier's testimony about the relative sensitivity of the cardiac system *vis-à-vis* the central nervous system was why cardiac symptoms were never observed or systematically measured in any study of subjects who received a sub-toxic dose. All of the studies relied upon by Petitioners to implicate the cardiac system were in the context of mercury poisoning, hundreds if not thousands of times the dosage of a thimerosal-containing vaccine. However, neurological effects were the ones studied in all of the Faroe and Seychelles Island studies as the most sensitive point for study.

Far worse than Dr. Lucier's foray into medical toxicology was the attempt of Dr. Shane to opine in that area. Dr. Shane demonstrated his lack of knowledge in this area by his complete denial of a dose relationship. Dr. Shane said at one point that dose was irrelevant to his analysis, and all he needed to know was that a certain chemical had been administered, and that the patient had died. His analysis on this point assumes a hyper-cataclysmic result for every possibility: Some doses of ethyl mercury can be toxic, therefore it was toxic for this patient. If that potentiality is highly unlikely, then it must be assumed that this patient was just really "different" and hypersensitive to that chemical. If the manner of toxicity is that it can cause arrhythmias at high doses, then here it not only caused arrhythmias, but myocarditis, congestive heart failure, and death from spontaneous cardiac arrest. Dr. Shane's testimony was counter-inductive, which is not to say deductive: He assumed a conclusion, and used all manner of Procrustean rationale to arrive at that conclusion. Such an approach is not scientific, and it is not helpful or persuasive to the Court. The Court heard from Dr. Shane based upon his expertise and experience in pathology, not for him to try his hand at medical toxicology.

The Court is only slightly more forgiving of Dr. Connor. At least Dr. Connor admitted on the stand that he had no basis for the broad toxicologic claims he had made in his expert report. At some points, he conceded that he could not provide citation for his conclusions on the spot, and promised to supplement his opinion with them (as should have been present as part of his expert report); he never kept his word on this point. The Court is left to presume that he had no basis in medical literature upon which to state his claims regarding toxicology. At one point, concerning the Magos paper, Dr. Connor demonstrated that he misunderstood the contents and conclusion of the article, and yet relied upon that faulty understanding when he actually cited two (unrelated) papers. At least, to his credit, Dr. Connor acknowledged the verity of the dose relationship as a foundation of medical toxicology.

On the topic of medical literature, it is quite telling that no medical literature directly supported Petitioners' central contention. Many of the studies on mercury toxicity in general, to which Petitioners' experts attempted to analogize, involved dosages that were three to five orders of magnitude distant from the infinitesimal amounts of ethyl mercury administered in thimerosal-

[173] Interestingly enough, the testimony of Petitioners' own toxicologist, Dr. Lucier, acknowledged that "co-exposure to other neurotoxicants" was a source of uncertainty identified by the National Academy of Sciences' monograph, *The Toxicological Effects of Methyl Mercury*, and could explain the difference in outcomes between the Faroe and Seychelle Island studies. Tr. 1 at 64-66.

containing vaccines.[174] To make matters worse, Petitioners' experts seized upon terminal conditions observed in mercury poisoning cases in an attempt to find similar phenomena in the instant matter. Most of those findings were generalized and nonspecific to toxicity, some of them a side-effect of the pathological process. Those that were somewhat more specific indicia for mercury toxicity (*e.g.*, "torpedo"-shaped Purkinje cells) were unidentified or absent from the facts at hand. And those *in vivo* studies that analyzed the potential effects of thimerosal in vaccines, using dosages that approximated what might be in vaccines, redounded against Petitioners' conclusion, most notably Pichichero.

Dr. Brent also dispelled the notion of a group of the human population that is hyper-susceptible to mercury toxicity, such that a infinitesimal dose of thimerosal would be sufficient to cause death. Petitioners raised a host of factors that could add a person into such a hypothetical grouping, such as age, sex, and genetic predisposition. Considering that it forms one of several linchpins for Petitioners' theory of causation, the record is remarkably bare of any evidence in support of this notion. Dr. Connor, having claimed that such a susceptibility existed, when pressed to describe the basis for his claim, admitted that it was mere speculation, and that he was unaware of any proof on this point. Also, even if medical literature had identified a genetic predisposition for enhanced sensitivity to mercury toxicity, no genetic analysis was done in this case to test Thomas Kolakowski for such a gene expression. Petitioners and their experts merely assume that Thomas was among such a hypothetical grouping, and ask the Court to do the same. The Court declines.

The crux of this issue is the dose-response curve, a "bell" curve that represents how a population will respond to a given dose of ethyl mercury. Although there is a normal distribution among the population, even some outliers, those outliers remain within finite bounds. In that sense, there are no "hypersensitive" individuals who are just so "different" that a virtually non-toxic dose will have toxic effects upon them. Dr. Brent explained that, with some chemicals, there are individuals who are hypersensitive, and only require a very small portion (compared to the general population) to express an adverse effect, but noted that this has never been observed in the case of mercury.

The Court now turns to consider the cardiologic evidence. The implosion of Dr. Connor's testimony on the stand caused the Court to view the substance of his opinion askew, and to downgrade the persuasive weight of his testimony. He claimed to rely on medical literature for several statements that traversed far beyond the area of cardiology, and when he was called to account for these sources, he either was at a loss concerning them, or admitted he was actually relying on other articles for the conclusions stated, or was proven incorrect in his understanding of such medical literature. He opined far and wide in comparing the EKG patterns of terminally-ill patients suffering mercury toxicity with agonal rhythm strips from Thomas Kolakowski's EKG readings, before admitting that both sets of data were irrelevant for determining how Thomas Kolakowski died. Dr. Connor also placed a great deal of weight on the clinical observation (found to be insignificant by the Court, *supra*) of "tremors" as noted in Mrs. Kolakowski's affidavit, despite

[174] Respondent's Brief summarized that subjects of the Dahhan study possessed blood levels of 15,000 micrograms, compared to 1,000 to 5,000 micrograms in the Cinca subjects; on the other hand, the mean peak mercury blood level reported by the Stajich and Pichichero studies was 2.2 micrograms following Hepatitis B vaccination.

the fact that Dr. Connor is not a neurologist. In fact, for all the testimony Dr. Connor offered on the topics of toxicology and neurology, he offered no opinion as to a mechanism of injury as to how the amount of mercury in a thimerosal-containing vaccine could cause death, at least the cardiologic aspects of that process. Furthermore, it gave every appearance of "grasping at straws" for Dr. Connor to opine, finally, that the mechanism could have been or was, an immunologic response to mercury, a curious, if not dubious, proposition. This idea was never raised in his expert report, but came about after a withering cross-examination that led Dr. Connor to distance himself largely from his previously stated opinion. Of course, Dr. Connor is not an immunologist, and Petitioners offered no immunologic expert witness evidence. The Court finds it telling, nonetheless, that Petitioners' offered cardiologist was willing to offer an opinion on causation from a standpoint of toxicology, neurology, and immunology, but can offer no opinion for a theoretical mechanism as it relates to cardiology. Even in the offer of a mere description of whether Thomas Kolakowski demonstrated myocarditis, either clinically or pathologically, Dr. Connor was at a loss, and deferred entirely to the pathologists. Amazingly, Dr. Connor's only explanation, when pressed to suggest a mechanism of injury, was to retreat to the redoubt of the *post hoc ergo propter hoc* logical fallacy, that, because the vaccinations were the only clinically relevant antecedent to Thomas' death (in Dr. Connor's eyes, at least), therefore the vaccines must have caused Thomas' death. As repeated *ad infinitum* in Vaccine Program cases, this line of reasoning is fatally defective.

Indeed, the only things approaching cardiologic evidence in this case were references to medical literature where adverse cardiac findings were associated with individuals who had received poisonous doses of mercury. Interestingly enough, even those findings were inconsistent. If mercury had a tell-tale, specific effect on the heart as a sensitive organ-system, one would expect to see a specific syndrome of symptoms. Instead, many of the victims of poisoning had no cardiac symptoms observed at all. The ones that did show cardiac symptoms demonstrated a gamut of different, nonspecific cardiac symptoms that were in no way traced aetiologically to the level of mercury toxicity by the researchers, and demonstrated no specific clinical or pathological course.

In contrast, Dr. Ringel was firm that Thomas Kolakowski showed no signs of interstitial myocarditis, from his standpoint of a pediatric cardiologist. Similarly, Dr. Ringel opined that there was no evidence of congestive heart failure or pulseless electrical activity as Dr. Shane had insisted. Dr. Ringel was adamant that there was no medical literature or evidence in the record that would suggest, let alone support, a relationship between the amount of ethyl mercury in thimerosal-containing vaccines and cardiac dysfunction or death from cardiac causes. He reiterated what Dr. Connor had said, that the agonal EKG rhythm strips taken following the discovery of Thomas as unresponsive were irrelevant and "of no value" in assessing a cause of Thomas' death, and were useless in forging a comparison with the Cinca study's data.

Finally, on the topic of pathology, Petitioners' expert, Dr. Shane, undermined the weight of his own testimony repeatedly. Petitioners never officially proffered Dr. Shane as an expert in a particular discipline, and his testimony ranged far and wide across several in which he has no specific expertise; the Court admitted his testimony as a general pathologist with many years of experience. However, in playing the expert in such far afield specialties as cardiology, immunology, gastroenterology (*inter alia*), Dr. Shane overreached, making his testimony less persuasive in the Court's weighing of expert testimony on the whole. By contrast, both Dr. Gilbert-Barness and Dr.

Rorke-Adams were careful to testify only within their areas of knowledge, even when pressed to offer an opinion on another area by Petitioners' cross-examination. They were also careful to delineate those conclusions that were based on experience within the field of pathology which were not yet fully understood or explained within the profession. Even within the general realm of pathology, Respondent's expert pathologists remained reticent to offer an opinion outside of their niche subspecialty, such as Dr. Gilbert-Barness abstaining from neuropathology, or Dr. Rorke-Adams abstaining from forensic pathology.

Another fact that undermines Dr. Shane's ability to persuade the Court was his Procrustean approach to describing the cardiologic findings. In his first expert report filed with the Court, Dr. Shane described everything besides the brain tissue as normal, or at least unremarkable. In his second report, he withdrew his blanket appraisal, finding myocarditis and other findings in the heart and other organs that he believed corroborated a cardiac injury from mercury toxicity. When questioned about this shift at the hearing, Dr. Shane explained that the cardiac findings and other late-noticed organ findings were so subtle, they required a closer, second look to perceive. And yet, when he and Respondent's experts were battling over the interpretation of the enlarged photomicrographs on rebuttal and surrebuttal, Dr. Shane argued that evidence of interstitial edema was "more than slight; it's at least moderate, and one might even consider it severe." Tr. 6 at 1616. As Procrustes to his houseguests, Dr. Shane chopped and stretched the evidence to fit his designs. Moreover, in the same rebuttal/surrebuttal disputation, Dr. Shane focused on any (inflammatory) white blood cell on the heart section slides as evidence of an inflammatory process, even if it were rather isolated, well below the concentration of an actual inflammatory reaction, averring that one cell is all he would need to see to declare an inflammatory response.

Also, Dr. Shane made so many claims that proved to be unsubstantiated, it made the Court much more cautious in relying on any of his testimony. In one hyperbolic example, Dr. Shane presented, as part of his rebuttal testimony, a photomicrograph which he described as liver cells from Thomas Kolakowski's pathology slides, which he then used to advance his arguments on the existence of an ongoing pathological process. Pet. Ex. 6 at 1600-01. He was rather insistent that the slide included a distinctive feature of the liver, "the space of Disse." *Id*. However, when viewed by Dr. Gilbert-Barness on surrebuttal, she pointed out that the cells in the picture were actually heart (muscle) cells, and not liver cells at all, a point conceded by a chagrined Dr. Shane. Tr. 7 at 23-24; 66-67. Of course, for a pathologist to confuse heart cells for liver cells gives an amateurish impression, especially when Petitioners argue that the Court should follow Dr. Shane's interpretation that there was pathological evidence of interstitial myocarditis and congestive heart failure over Dr. Gilbert-Barness' interpretation that there was not. She did not see the "mixed inflammatory cell infiltrate" or other signs of cardiac degeneration that he testified to perceiving. In fact, she witnessed no evidence of chronic, or even subacute processes, only the acute trauma of hypoxic ischemia, and some cellular damage that was the resulting artifact of slide preparation.

In any event, Dr. Shane's insistent interpretation of myocarditis is ultimately irrelevant in the absence of a theoretical framework of a causation theory. As it is, Dr. Shane's argument about

myocarditis is an exercise in the colloquially-termed "Texas sharpshooter fallacy."[175] As Dr. Brent explained, myocarditis is not associated with mercury toxicity in any fashion, except that myocarditis can form part of the clinical profile of any very ill patient, such as those suffering from acute, severe mercury poisoning.

Dr. Shane committed a similar logical fallacy when he latched onto the findings reported in the Cinca article, and attempted to portray Thomas Kolakowski's pathological findings as similar. Although the authors of the study did not attribute certain findings to the mercury toxicity specifically, but merely described the profile of symptoms that manifested, Dr. Shane did attribute certain symptoms and pathological findings in that study to the mercury toxicity suffered by its subjects, and then strained to find similar findings in Thomas Kolakowski's records, in order to diagnose mercury toxicity. This is the "*cum hoc ergo propter hoc*" or "with this therefore because of this" fallacy,[176] which is another variant of the "*non causa pro causa*" fallacy. Once again, without a rubric within which to understand causation, this strained reasoning is unpersuasive, to the extent it is not downright irrelevant.

One prime example of Dr. Shane's flawed approach is when he grasped to find similarities to findings in poisoning studies, when they were inconsistent with actual findings associated with mercury toxicity. In one instance, he testified that Purkinje cell damage was evident in Thomas Kolakowski's cerebellar slides, and related that to a finding in an article of medical literature. It was only when Dr. Rorke-Adams testified that it was revealed, with citation to a reputable source, that the hallmark change of such cells in cases of mercury toxicity was the "torpedo" shape transformation, which was not apparent from Thomas' slides, and would be impossible to identify if it had been present, due to the necessity for a special kind of staining procedure to observe them. Similar errors regarding the microglia and oligodendroglia demonstrated that Dr. Shane was outmatched by the expertise and knowledge of Dr. Rorke-Adams in the realm of neuropathology. At one point, Dr. Rorke-Adams explained that what Dr. Shane thought was granular cell "drop out" was actually just a stage of development in the infant cerebellum. As per usual, the Court was impressed and heavily persuaded by the neuropatholgic expertise of Dr. Rorke-Adams, and her ability to explain her conclusions cogently.

Dr. Shane was also observed to stretch reality to fit his construct when considering the clinical findings upon which he relied in his pathological diagnosis. He viewed a rash as possible evidence of hypersensitivity, when the medical records are specific that the "rash" was actually

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[175] This category of fallacy receives its name from the concept of a gunslinger who fires a gun randomly at the side of a barn, and then draws a bullseye around the biggest cluster of bullet holes. It denotes a post hoc attempt to attribute association or causation between two or more unassociated events that happen by chance in conjunction with one another. It is a subspecies of the "*non causa pro causa*" family of fallacies.

[176] What follows is but one example:

> I talk about it being mercury toxicity. Again, in terms of the interstitial myocarditis, I don't know what the mechanism for that was. It is a finding. It's a finding that is reported in ethyl mercury toxicity deaths. I see it here. One and one equals two.

Tr. 3 at 736.

infantile acne in reaction to over-the-counter soap. He described ongoing gastrointestinal problems when the medical records present an adequately-nourished, healthy-eating child, with the exception of the night before his death. Risibly, Dr. Shane viewed as evidence of myocarditis Thomas' one-day history of gas. Of course, he relied on the tremors as neurologically significant, even though Petitioners' expert pediatric neurologist had already dispelled that notion months before the hearing.

Noticeably absent were the very distinctive symptoms of mercury toxicity one could expect to observe in Thomas were he to have suffered mercury toxicity. From the symptom of pinkish skin to the photophobia described by the experts in this matter,[177] Thomas appeared by all lights to be a healthy infant, even until he went to sleep in the wee hours of 25 January 1999. As "the dog that didn't bark," the absence of any such tell-tale clinical evidence of mercury toxicity militates against a finding of lethal mercury toxicity.

In contrast, Respondent's postulate of hypoxic ischemia, ostensibly related to some manner of partial (inadvertent) smothering, was corroborated by the evidence, and served as both a competing, alternative theory of causation undercutting Petitioners' case in chief, and as a so-called "factor unrelated" affirmative defense for Respondent. The most striking clinical signs of such a possibility were the condition of co-sleeping stipulated to by Petitioners and the anterior (frontal) lividity by which Thomas was described. Also corroborative in the clinical records was the slight head cold or upper respiratory infection noted to have been affecting Thomas the day prior to his death.

Corroborative evidence of hypoxic ischemia was much stronger in the pathologic findings. The consolidation of the lungs, the petechial hemorrhaging, the oral and nasal bleeding, and the cerebral edema all were consistent with, if not diagnostic of, hypoxic ischemic injury leading to death.

One point of contention regarding the pathologic record was the conclusion of the medical examiner, which concluded that Thomas' death was unexplained infant death, likely a formulation of SIDS, although it did not rule out a potential association with the Hepatitis B vaccination(s). Petitioners argue that this equivocation about the potential for vaccine causation is tantamount to a diagnosis for vaccine causation, and is therefore entitled to the purported shortcut around proving specific causation ostensibly provided by the Federal Circuit's *Capizzano v. Sec'y of HHS* opinion.[178]

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[177] *See supra* at 135. The Dahhan study listed clinical symptoms of toxicity including "sore mouth, metallic tastes, blue lines, vomiting, diarrhea, fever, insomnia, tremors, dysarthria, ataxia, hyperreflexion, hyporreflexion, [and] atrophy." *Supra* at 62.

[178] There the Federal Circuit stated:

> In our view, the chief special master erred in not considering the opinions of the treating physicians who concluded that the vaccine was the cause of [the] injury. The fact that these physicians' diagnoses may have relied in part on the temporal proximity of [the] injuries to the administration of the vaccine is not disqualifying… In other words, if close temporal proximity, combined with the finding that [the vaccine at issue] can cause [the injury alleged], then medical opinions to this effect are quite probative.

440 F. 3d 1317, 1326 (Fed. Cir. 2006).

Of course, the medical examiner's conclusion ascribes no such relationship, and his only conclusion is a quasi-Socratic conclusion not to conclude on a true diagnosis at all.[179] This leaves no true diagnosis for the Court to afford the deference due to a treating diagnosis, unless one reads his notation of sudden death as a shorthand for SIDS, which, as Dr. Gilbert-Barness testified, can be a sloppy code-word for positional asphyxia in many cases.[180]

Most damning for Petitioners' claim, because it affects a global requirement for their claim which remains foundational to recovery in an actual causation paradigm, was the absence of evidence in support of a theory of causation. This is the concept of general causation, which answers the question "can it?"–*i.e.*, can the agent implicated cause the injury alleged in a logical or plausibly hypothetical construct of medical science? This element is represented in the Federal Circuit's *Althen* opinion by the requirement of a "a medical theory causally connecting the vaccination and the injury." 418 F. 3d at 1278. The dearth of evidence on this crucial point is enough to defeat Petitioners' claim for compensation.

Also, because none of Petitioners' experts postulated an explanation of a theoretical mechanism by which injury or death could occur due to the amount of ethyl mercury within thimerosal-containing vaccines, there is no way to assess the medical records or the sequence and timing of events, to see if they correspond to such a theoretical construct. Without proffering a theory of causation, it is impossible for the Court to assess the other aspects of the *Althen* formulation of causation, namely a discernable chain of causation in the medical record and adherence to a medically-appropriate time frame.

To the extent that Petitioners argument is that ethyl mercury in thimerosal-containing vaccines is present in ample enough amounts to cause acute mercury toxicity, leading to death, not only are the evidence of clinical indicia absent, but the timing interval presented does not jibe with the latency of adverse effects of toxicity, a point that was made repeatedly. Dr. Lucier stated that mercury poisoning "is not manifested immediately after exposure," but "can be delayed for a considerable period of time." Tr. 1 at 66, 68. Not only did this latency period undercut his criticism of the Pichichero article (as pointed out by Dr. Brent), but it also undermines a postulate of acute mercury toxicity in this case. The only exception to this latency point was Dr. Shane, who believed that, regardless of dose administered, a person could die within a day of any exposure to mercury, but this was quite unpersuasive and gave the impression of overreaching beyond his area of professional expertise. Even in the poisoning cases ostensibly relied upon by Petitioners' experts, mercury toxicity followed a latency, followed by the onset of symptoms, and only then, if at all, by

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The problem for Petitioners in relying thereupon is that this verbiage does not relieve them of proving general ("can it") causation. In fact, the Circuit's wording indicates that the "can it" question must have already been answered in the affirmative through sufficient proof in order for the treaters' conclusions to weigh heavily on the "did it" question. As noted repeatedly *supra*, Petitioners have failed outright in postulating a theory of causation.

[179] ``Εν οἶδα ὅτι οὐδὲν οἶδα. "The one thing I know is that I know nothing." Plato, *The Apology*.

[180] Respondent also argues that a medical examiner cannot be considered a "treating physician" because he does not treat disease processes contemporaneously, but only analyzes disease processes posthumously. This question is not necessary to answer to resolve the issue raised, and the Court thus leaves it unanswered.

death. Thus, even an argument of comparison and analogy to the poisoning cases must fail, because the timing in the instant case does not follow what the evidence propounds to be a "medically appropriate time frame" for onset and injury.

All of these points build on one another, to arrive at the factual finding that the Court was persuaded neither that thimerosal-containing vaccines generally, nor that two Hepatitis B vaccines in particular, could cause the death of an infant like Thomas Kolakowski, and the Court remains unpersuaded that Thomas Kolakowski's death was actually caused by his two Hepatitis B vaccines. The Court finds that Thomas Kolakowski's death was unrelated to his vaccinations, based on the evidence presented, and that his death would have occurred notwithstanding whether those vaccines had been administered, or not. Furthermore, the Court finds that, in the case of Thomas Kolakowski, his death was the result of hypoxic ischemia, which may have been caused by external, physical asphyxiation, but which, in any event, was unrelated to the vaccine.

III. CONCLUSIONS OF LAW

A Court's authority to grant relief, just as with the Court's authority to hear a particular subject matter, is granted by the Legislature. With the exception of the Supreme Court of the United States, federal courts are, one and all, creatures of statute;[181] therefore they each exercise limited subject matter jurisdiction which is itself granted by statute.[182] Therefore, they have been granted

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[181] United States Constitution, Article III, Section 1; *Kline v. Burke Constr. Co.*, 260 U.S. 226, 234 (1922). The Supreme Court, in a landmark case on federal jurisdiction, held:

> Jurisdiction of the lower federal courts is further limited to those subjects encompassed within a statutory grant of jurisdiction. Again, this reflects the constitutional source of federal judicial power: Apart from this Court, that power only exists "in such inferior Courts as the Congress may from time to time ordain and establish."

Insurance Corp. of Ireland v. Compagnie des Bauxites de Guinee, 456 U.S. 694, 701-702 (1982).

[182] This restriction is based upon the appropriate deference due to state governments and laws contemplated by our federal system:

> The power reserved to the states, under the Constitution, to provide for the determination of controversies in their courts, may be restricted only by the action of Congress in conformity to the judiciary sections of the Constitution. Due regard for the rightful independence of state governments, which should actuate federal courts, requires that they scrupulously confine their own jurisdiction to the precise limits which a federal statute has defined.

Victory Carriers, Inc. v. Law, 404 U.S. 202 (1971), quoting *Healy v. Ratta*, 292 U.S. 263, 270 (1934) (internal marks omitted).

the authority and power to hear cases (i.e., jurisdiction[183]) where specific subject matter is at issue.[184] Jurisdiction, simply stated, is proper authority and exercise of (judicial) power.

In the context of federal courts, within the federal experiment of these United States, the focus rests upon subject matter jurisdiction. The previously independent, *sovereign* Colonies were each granted independence from King George III to become States,[185] and at first only confederated for mutual benefit under the Articles of Confederation. After some time, the people of these several States decided it was best to divest certain aspects of sovereignty (but only those aspects) from each of the States, and to reinvest that sovereignty in the national government (now commonly referred to as the "federal" government). As has been repeatedly shown, only those aspects of authority and sovereignty which were vested explicitly in the national government, through the ratification of the United States Constitution, can be exercised by any part of the federal government. United States Constitution, Amendment X. Therefore, in a narrowing cone of authority: the Constitution only grants to the national (federal) government the authority given it by the people of the several States; the national government as a whole may only exercise the authority contained in the body of the Constitution; Congress may only legislate national laws according to the grants and limitations of authority contained in Article I, including their legislation granting judicial authority to the federal courts; and federal courts may only exercise jurisdiction over the subject matter of the legislation passed by Congress. As a result, federal courts do indeed wield great power, but they wield that power within a circumscribed area of authority, beyond which they shall not trespass. This is the basis for the distinction between federal courts, which are of "limited" jurisdiction, and courts of the several State governments, which retain general jurisdiction over all subject matter not explicitly and exclusively assigned to the national (i.e. federal) courts.

This brings us back to the Vaccine Program. Congress ostensibly possessed constitutional prerogative to create the Program under the authority of the Taxing and Spending Clause of the Constitution, found at Article I, Section 8, Clause 1. In creating the Program, Congress vested the United States Court of Federal Claims (originally the Claims Court), acting through the Office of Special Masters, with "jurisdiction over proceedings to determine if a petitioner under [Section 11 of the Act] is entitled to compensation under the Program and the amount of such compensation." Section 12 (a). Therein Congress also vested the Court with the power to "issue and enforce such orders as the court deems necessary to assure the prompt payment of any compensation awarded."

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[183] The authority and power to hear cases relating to a particular subject matter of substantive law is what is properly referred to as "subject matter jurisdiction." *See generally Da Silva v. Kinsho Intern. Corp.*, 229 F.3d 358, 361 (2d Cir. 2000) (noting with chagrin that "Court decisions often obscure the issue by stating that the court is dismissing 'for lack of jurisdiction' when some threshold fact has not been established, without explicitly considering whether the dismissal should be for lack of subject matter jurisdiction or for failure to state a claim").

[184] *See*, *e.g.*, United States Constitution, Article III, Section 2: "The judicial Power shall extend to all Cases...arising under...the Laws of the United States..."; Vaccine Act, Section 12(a) ("The United States Court of Federal Claims and the United States Court of Federal Claims special masters shall, in accordance with this section, have jurisdiction over proceedings to determine if a petitioner under [section 11] is entitled to compensation under the Program and the amount of such compensation. The United States Court of Federal Claims may issue and enforce such orders as the court deems necessary to assure the prompt payment of any compensation awarded.")

[185] Treaty of Paris, Article I (1783).

Id. The subject matter jurisdiction of the Court then, by which authority the Court rules here, is then the authority to decide if petitioners are entitled to compensation on their claims of vaccine-related injury, and if so, the appropriate amount of that compensation.

Moreover, the Court certainly possesses personal jurisdiction over the parties. Indubitably, a petitioner's election to file a petition in the Program subjects him or her to the Court's control. Likewise, the express language of Congress in the Vaccine Act subjects the available amounts in the Vaccine Compensation Fund to the Court's power, and even grants explicit power to the Court to enforce its orders of payment from the Fund.[186] Section 12 (a).

As aforementioned, the Court is authorized to award compensation for claims where the medical records or medical opinion have demonstrated by preponderant evidence that either a cognizable Table Injury occurred within the prescribed period or that an injury was actually caused by the vaccination in question. § 13(a)(1). If Petitioners had claimed that Thomas had suffered a "Table" injury, to them would §13(a)(1)(A) have assigned the burden of proving such by a preponderance of the evidence. In this case, however, Petitioners do not claim a presumption of causation afforded by the Vaccine Injury Table, and thus the Petition may prevail only if it can be demonstrated to a preponderant standard of evidence that the vaccination in question, more likely than not, actually caused the injury and death alleged. *See* § 11(c)(1)(C)(ii)(I) & (II); *Grant v. Sec'y of HHS*, 956 F. 2d 1144 (Fed. Cir. 1992); *Strother v. Sec'y of HHS*, 21 Cl. Ct. 365, 369-70 (1990), *aff'd*, 950 F. 2d 731 (Fed. Cir. 1991). The Federal Circuit has indicated that, to prevail, every petitioner must:

> show a medical theory causally connecting the vaccination and the injury. Causation in fact requires proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury. A reputable medical or scientific explanation must support this logical sequence of cause and effect.

Grant, 956 F. 2d at 1148 (citations omitted); *see also Strother*, 21 Cl. Ct. at 370.

Furthermore, the Federal Circuit later summarized this analysis into a three part test:

> [Petitioner's] burden is to show by preponderant evidence that the vaccination brought about [the] injury by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury.

Althen v. Sec'y of HHS, 418 F. 3d 1274, 1278 (Fed. Cir. 2005).

As part of that analysis, the Federal Circuit recently explained:

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[186] In so doing, Congress made the conscious choice to vest adjudicatory authority in the Court of Federal Claims, an "Article I court" whose judges lack life tenure. *Ex parte Bakelite Corporation*, 279 U.S. 438 (1929) ("Legislative courts also may be created as special tribunals to examine and determine various matters, arising between the government and others, which from their nature do not require judicial determination and yet are susceptible of it.").

> [T]he proximate temporal relationship prong requires preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's aetiology, it is medically acceptable to infer causation-in-fact.

de Bazan v. Sec'y of HHS, 539 F. 3d 1347, 1352 (Fed. Cir. 2008).

Under this analysis, while a petitioner is not required to propose or prove definitively that a specific biological mechanism can and did cause the injury, he must still proffer a plausible medical theory that causally connects the vaccine with the injury alleged. *Knudsen v. Sec'y of HHS*, 35 F. 3d 543, 549 (1994).

As a matter of elucidation, the Undersigned takes note of the following two-part test, which has been vindicated and viewed with approval by the Federal Circuit,[187] and which guides the Court's practical approach to analyzing evidence in light of the *Althen* elements:

> The Undersigned has often bifurcated the issue of actual causation into the "can it" prong and the "did it" prong: (1) whether there is a scientifically plausible theory which explains that such injury could follow directly from vaccination; and (2) whether that theory's process was at work in the instant case, based on the factual evidentiary record extant.

Weeks v. Sec'y of HHS, No. 05-0295V, 2007 WL 1263957, 2007 U.S. Claims LEXIS 127, slip op. at 25, n. 15 (Fed. Cl. Spec. Mstr. Apr. 13, 2007).

Of importance in this case, it is part of Petitioners' burden in proving actual causation to "prove by preponderant evidence both that [the] vaccinations were a substantial factor in causing the illness, disability, injury or condition and that the harm would not have occurred in the absence of the vaccination." *Pafford v. Sec'y of HHS*, 451 F. 3d 1352, 1355 (Fed. Cir. 2006), *rehearing and rehearing en banc denied*, (Oct. 24, 2006), *cert. den.*, 168 L. Ed. 2d 242, 75 U.S.L.W. 3644 (2007), citing *Shyface v. Sec'y of HHS*, 165 F. 3d 1344, 1352 (Fed. Cir.1999). This threshhold is the litmus test of the cause-in-fact (a.k.a. but-for causation) rule: that petitioner would not have sustained the damages complained of, *but for* the effect of the vaccine. *See generally Shyface, supra*. "[T]he relevant inquiry ...[is]... 'has the petitioner proven ... that her injury was in fact caused by the ... vaccine, rather than by some other *superseding*[,] *intervening* cause?' ...[The petitioner need not] rule out every possible explanation ...[but]... must simply show ... that her injury was caused by a vaccine." *Johnson v. Sec'y of HHS*, 33 Fed. Cl. 712, 721 (1995), *aff'd* 99 F. 3d 1160 (Fed. Cir. 1996) (emphasis added).

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[187] *See Pafford v. Sec'y of HHS*, No. 01-0165V, 2004 WL 1717359, 2004 U.S. Claims LEXIS 179, *16, slip op. at 7 (Fed. Cl. Spec. Mstr. Jul. 16, 2004), *aff'd*, 64 Fed. Cl. 19 (2005), *aff'd* 451 F. 3d 1352, 1356 (2006) ("this court perceives no significant difference between the Special Master's test and that established by this court in *Althen* and *Shyface*"), *rehearing and rehearing en banc denied*, (Oct. 24, 2006), *cert. den.*, 168 L. Ed. 2d 242, 75 U.S.L.W. 3644 (2007).

"To prove causation, a petitioner in a Vaccine Act case must show that the vaccine was 'not only a but-for cause of the injury but also a substantial factor in bringing about the injury.'" *Moberly v. Sec'y of HHS*, 592 F.3d 1315, 1321 (Fed. Cir. 2010) quoting *Shyface v. Sec'y of HHS*, 165 F.3d 1344, 1352-53 (Fed. Cir. 1999); *see also Id*. citing *Walther v. Sec'y of HHS.*, 485 F.3d 1146, 1151 (Fed. Cir. 2007) (for causation analysis in off-Table cases, the Restatement (Second) of Torts applies and 'the petitioner is treated as the equivalent of the tort plaintiff'). In the watershed case of *Shyface v. Sec'y of HHS*, 165 F. 3d at 1352, the Federal Circuit "adopt[ed] the Restatement [(2d) of Torts] rule for purposes of determining vaccine injury, that an action is the 'legal cause' of harm if that action is a 'substantial factor' in bringing about the harm, and that the harm would not have occurred but for the action," and that rule continues to guide the Court today in the instant matter.[188] *Cf. Hargrove v. Sec'y of HHS*, No. 05-0694V, 2009 WL 1220986 * 39-40 (Fed. Cl. Spec. Mstr. Apr. 14, 2009).

Here, Petitioners never offered "a medical theory causally connecting the vaccination and the injury," in order to show how the ethyl mercury in thimerosal-containing vaccines could cause the death of an infant vaccinee. That alone is a fatal defect in an actual causation case, because if general causation is not proved, it is logically impossible for specific causation to be proved. In this specific case, Petitioners also failed to prove "a logical sequence of cause and effect showing that the vaccination was the reason for the injury," to show that the ethyl mercury in two thimerosal-containing Hepatitis B vaccines did cause the death of Thomas Kolakowski. As such, Petitioners have not proven that death would not have occurred *but for* the administration of the vaccines, and the inclusion of thimerosal within those vaccines in particular. This also means that Petitioners have not proven that thimerosal-containing vaccines are a proximate cause of death. In actual causation cases such as this, these defects of proof are fatal, and this Court has no alternative but to dismiss this claim. Hence, the Court **RULES** that Petitioners are not entitled to compensation.

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[188] The mandate of the Federal Circuit in *Shyface* to follow the RESTATEMENT (2D) OF TORTS on the application of actual causation did not indicate how this Court should approach the tectonic shift of the common law into the later Restatement(s). The short answer to this question is that the Federal Circuit incorporated the RESTATEMENT (2D) OF TORTS, and until the Circuit does otherwise to change that gloss, that is the mandatory precedent binding on this Court. By way of more detailed analysis, given the Circuit's reasoning in *Shyface* for incorporating the Restatement, *i.e.* that Congress contemplated the common law (in its then contemporaneous understanding) within the Vaccine Act draftsmanship, thus presuming the common law as a background legislative intent, it would appear that only the Second Restatement is binding on this Court in matters touching on actual causation, because that is the version in use at the time of the Act's drafting and passage. Likewise, when the Federal Circuit decided *Shyface* in 1999, the RESTATEMENT (3D) OF TORTS: PRODUCTS LIABILITY had already become available in published form, and yet the Circuit did not choose to incorporate or even reference that Restatement's provisions at all, notwithstanding the potential corollary to the Program's focus on causation in the absence of a fault element. Had it done so, a contrary argument could have been made that the Circuit's reading of congressional intent was a progressing correspondence to whatever Restatement provisions were most current. However, this would seem to correspond to the more dubious "statutory purpose" canon of interpretation. The Court's reading of *Shyface* leads to a result that the Third Restatement should be viewed at most as persuasive, but not mandatory authority, and is not to be followed where it conflicts with the Second Restatement.

IV. CONCLUSION

In light of the foregoing, the Court **RULES** against entitlement in this matter. Therefore, no alternative remains for this Court but to **DISMISS** this petition with prejudice. In the absence of the filing of a motion for review, filed pursuant to Vaccine Rule 23 within 30 days of this date, the clerk shall forthwith enter judgment in accordance herewith.

IT IS SO ORDERED.

s/ Richard B. Abell
Richard B. Abell
Special Master