In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 05-1261V

Filed: 4 March 2008

FRANCES PEREZ, Natural Mother and Legal Representative of the Estate of MARIO PEREZ, Deceased, Petitioner, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	**PUBLISHED** DTaP, Pertussis Encephalopathy, Death, Sudden Infant Death (Syndrome) (SID(S)), Positional Asphyxia, Table, Actual Cause, Vasogenic and Cytotoxic Cerebral Edema

Thomas P. Gallagher, Esq., Gallagher & Gallagher, Somers Point, New Jersey, for Petitioner; *Traci R. Patton, Esq.*, United States Department of Justice, Washington, D.C., for Respondent.

ENTITLEMENT RULING[1]

ABELL, Special Master:

On 5 December 2005, the Petitioner filed a petition for compensation under the National Childhood Vaccine Injury Act of 1986 (Vaccine Act or Act)[2] alleging that, as a result of the DTaP[3]

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[1] Petitioner is 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] 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.A. §300aa.

[3] At the same office visit, Mario also received the IPV, Hib, Hep B, and pneumococcal vaccines as well. Petition at 1. At first there was some ambiguity regarding whether Mario received the DTP vaccine or the acellular Pertussis vaccine (DTaP), but that inconsonance appears to have been resolved satisfactorily.

vaccination received on 17 December 2003, her child, Mario, suffered an acute Table Encephalopathy or, alternatively, an acute off-Table Encephalopathy or other injury actually caused by said vaccine. Petition at 1.

This petition was assigned to my chambers on 5 December 2005. Eventually, a live evidentiary hearing on the ultimate issue of entitlement for compensation was held in the Court's Hearing Room in Washington D.C. on 26 January 2007. Hearing Transcript ("Tr.") at 1. Whereupon, the Court heard from the Petitioner, mother of the decedent, and medical expert witnesses for both parties: Dr. John J. Shane and Dr. Marcel Kinsbourne for the Petitioner, and Dr. Enid Gilbert-Barness and Dr. Michael Kohrman for the Respondent. Subsequent to that hearing, the parties filed closing briefs with the Court, and the case is now ripe for a ruling.

As a preliminary matter, the Court notes that Petitioner has satisfied the pleading requisites found in § 300aa-11(b) and (c) of the statute, by showing that: (1) she is a valid legal representative of the injured party, Mario Perez; (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 § 300aa-16(a) requirement that the petition be timely filed has been met. On these matters, Respondent tenders no dispute.

The Vaccine Act authorizes the Office of Special Masters to make rulings and decisions on petitions, which 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 listed. § 11(c)(1)(c).

I. FACTUAL RECORD

Despite their accord on certain factual predicates contained in Mario's medical records, there is, unsurprisingly, a pronounced conflict between the parties as to the following issues: whether Mario's autopsy results were consistent with "significant" vasogenic cerebral edema, whether those results bore evidence of asphyxiation, whether the clinical indicia and/or microscopic analysis of autopsy slides indicated an encephalopathy, and, ultimately, whether an encephalopathy led to Mario's untimely death. 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 the Petitioners bear 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. 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).

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. Secretary 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. Secretary 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. Secretary of HHS*, No. 90-45V, slip op. at 3 (Cl. Ct. Spec. Mstr. March 28, 1991).

A. Medical Records *et al*.

The Court turns first to the recorded facts drawn from the medical records engendered and maintained by those responding to, and treating, Mario's health concerns:

Mario Perez was born 16 October 2003 by Caesarian section. In the discharge summary of Mario and his mother, following his birth, the reason given for the Caesarian section was "persistent nonreassuring fetal heart tones." Petitioner's Exhibit ("Pet. Ex.") A at 5. Other than a couple of hospital visits at eight and thirteen days for occasional vomiting (Pet. Ex. B at 68-69), Mario's first two months passed unremarkably for the purposes of this Petition.

On 17 December 2003, Mario was seen for his two-month-old well-child medical examination, at which he was observed to be developing normally. Pet. Ex. B at 69. At that time, Mario was 21.25 inches (53.975 cm) long, he weighed 10.1 lbs. (4.6 kg), and his head circumference

was 39 cm (15.35 inches). *Id*. At that visit, the DTaP, IPV, Comvax, and Prevnar vaccines were administered to Mario. *Id*.

In her affidavit, Petitioner described Mario before the vaccination at issue as normal, interactive, vocal, observant, even smiling. Affidavit of Frances Perez. She indicated that once she arrived home with Mario, "his behavior had changed," such that he slept in intervals of two hours, had a lessened appetite, and whined often, a situation that persisted all that night. *Id*. The day after the vaccination, 18 December 2003, Mario whined less, but became less interactive and responsive. *Id*. In the late morning, Mario did not cry as usual when placed into a bath, but simply lay staring at the ceiling, unresponsive to Petitioner's voice or physical presence. *Id*. Even when removed from the bath, Mario did not react, even when exposed to the cooler air or toweled dry. *Id*. That day proceeded much as the preceding one had: "he wouldn't drink more that two ounces of formula, he slept for [maybe] two hours, he whined constantly, and he had a glazed-over stare." *Id*.

The next medical records are from 19 December 2003, when Mario arrived at Lourdes Medical Center "unresponsive" (Pet. Ex. D at 81) and "cold to the touch" (*Id*. at 88), giving rise to the diagnostic impression of "DOA" (*Id*. at 87).

The physical examination performed by the police of the scene of Mario's death described the house as "extremely well[-]kept and very clean," appearing "orderly and nothing out of place." Pet. Ex. E at 93. A responding officer reported that he thought Mario "may have just suffocated himself in the bassinet while sleeping," as "[t]here was no pillow in the bassinet but it was not firm either." *Id*. at 96.

The Police also examined the witnesses who were at home when Mario was found to be unresponsive, prompting the call to emergency services. *See* Pet Ex. E. The police account reflects the tragedy and loss that beset the Perez family on the morning of 19 December 2003. Regarding Mario's health on the evening of 18 December 2003, the Police reported that Petitioner's adult son, George Perez, Jr., recounted to them that he was unaware of any manifest health problems affecting Mario, but that he had thought Mario occasionally sounded congested, but that a doctor had categorized that as normal. *Id*. at 94.

The Police report records that Petitioner, Mario's Mother, told them that Mario had not evidenced "any complications from the shots," but that "he just cried a little more just following," adding that Mario slept all night long on the night of 17 December 2008. *Id*. Her husband, George Perez, Sr., told the police that, upon waking on the morning of 19 December, he mentioned to her that Mario had slept all through that night, an unusual event at the time. *Id*. at 96. When Petitioner went to check on Mario, she found that he did not respond to her touch, but was instead cold and stiff. *Id*. at 94. She ran with him, handing him to George Sr. so as to call 911, and George Sr. felt immediately that he was stiff, "cool to the touch and his lips were blue." *Id*. at 96. When the police arrived, they took Mario to Our Lady of Lourdes Hospital Emergency Room, administering CPR to him along the way. *Id*. at 97.

The autopsy report, as amended, states that Mario died of natural causes from sudden infant death syndrome ("SIDS").[4] Pet. Ex. C at 73-74. The autopsy examination report states regarding external examination that Mario was a "well[-]developed, well[-]nourished, Hispanic male," with no evidence of any traumatic injury. *Id*. at 76. Similarly, there was no traumatic injury evident in Mario's internal examination either. *Id*. at 76-79. With the exception of some fluid buildup in a few areas, examination of the serous cavities, the gastrointestinal tract, and the abdominal organs revealed nothing abnormal. *Id*. The lymph nodes were not swollen. *Id*. at 78. The heart weighed 28 grams and both left and right chambers of the heart were congested; otherwise it was in all appreciable aspects normal. *Id*. at 77. The larynx and trachea were normal. *Id*. Both the right and left lungs showed congestion in the serosa surface, from which surfaced bloody fluid when cut; they weighed 66 grams and 47 grams, respectively. *Id*. On the thymus gland, "small petechiae [were] noted bilaterally." *Id*.

Mario's head was normocephalic. *Id*. at 79. His brain weighed 549 grams at examination. There was no evidence of hemorrhage, trauma or tumor in the brain or spinal cord, although the report notes some congestion "along the posterior durameter of the vertebra column." *Id*.

B. Testimony at the Entitlement Hearing

1. Petitioner Frances Perez

Petitioner testified live at the entitlement hearing in Washington, D.C. Her testimony was consistent with the contents of her written affidavit, filed previously, although her testimony was focused significantly on resolving conflicts between her affidavit and potential inconsistencies in the police records.

She began her direct examination, understandably, discussing the emotional turmoil that surrounded the event of Mario's death. Tr. at 9. Mario's death came after a day of Christmas shopping, while Petitioner's older son was visiting on medical leave from the Fort Bragg base of the United States Army, having lost a substantial portion of his left leg in the war in Iraq. Tr. at 10, 24; Pet. Ex. E.

In response to a question from the Court, Petitioner indicated that it was only four or five hours after Mario's death that she spoke with the Police, having only "just come from the hospital, from holding [Mario] for probably about an hour and a half." Tr. at 9. She explained her statements to the Police, saying that Mario did indeed evidence the behavior described in her affidavit, but that when she spoke with the police, she meant that Mario's behavior seemed "normal under the circumstances of having received the shots," but that "it wasn't the same as what he was like prior to receiving them." Tr. at 11. She continued, explaining why she had not thought to mention the specific behaviors discussed in her affidavit: "When the police officer was asking me, I wasn't sure what he was looking for, and I was in a rather weird state since I had just lost my son. But looking

[4] Sudden Infant Death Syndrome 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 Illustrated Medical Dictionary (30th ed. 2003) (Saunders) at 1833.

back and analyzing his behavior from the time he got [the vaccination], I did remember a lot of things." Tr. at 12. She elaborated on this point further in response to direct examination: "It wasn't something that I thought of at the time. When he interviewed me, I thought he was looking for criminal activity and he just wanted to rule that out." Tr. at 13.

After the conclusion of Petitioner's direct testimony, the Court queried her whether she had consulted with medical personnel in preparation of her affidavit regarding the behaviors she witnessed and/or described, "what the significance may or might not be in regard to those symptoms," and further, whether she had talked about those potential symptoms with anyone outside of her family. Tr. at 14. She replied that she had not, and that she did not receive any "assistance" from her attorney either. Tr. at 14-15.

On cross-examination, Petitioner disclosed that, very shortly after Mario had died, a family friend and pediatrician asked whether Mario had died within 48 hours of vaccination, urging Petitioner to order a "packet" from the NVICP, and that this friendly notification was the first circumstance which led Petitioner to believe that the vaccination "may have played a role" in Mario's untimely death. Tr. at 15-16. Even at that time, Petitioner's concept of the Program was for "getting information to help prevent that in the future, any children, any infants died as a result of vaccinations and that type of thing," such that she then participated "if it was going to help with something." Tr. at 16.

Also on cross-examination, Petitioner contrasted what behaviors had been normal for Mario before the vaccination with his behavior after the vaccinations, such as consuming four to five ounces of formula and sleeping four to five hours at a time instead of one to two ounces of formula and only two-hour sleep intervals. Tr. at 16. Petitioner admitted that her interaction with Mario between the vaccination and his death did not raise her suspicion because she assumed such behavior was within normal range for the period immediately following vaccination. Tr. at 17-18. She also discussed Mario's preference for sleeping on his stomach instead of on his back, and how he slept in a bassinet. Tr. at 19.

Given ample observation of her presentment, comportment, and demeanor, the Court found Petitioner's testimony to be clear and cogent. The Court was persuaded in her credibility and her veracity, and accepts her testimony as reliable, regarding the period between vaccination and Mario's death.

2. John J. Shane, MD

Petitioner filed multiple medical expert reports from Dr. Shane. In addition to the written medical records discussed *supra*, Dr. Shane also evaluated 20 original glass pathology slides via microscopic examination. *See* First Expert Report of Dr. Shane, dated 26 June 2006.

He comments in his first expert report that the heart, lungs, gastrointestinal tract, kidneys, liver, spleen, larynx, adrenal glands, pancreas, and thymus were each unremarkable, in some cases indicating to him that SIDS death is less likely because of the absence of typical indicators in the

autopsy findings. *Id*. That is, because the heart did not show right ventricular hypertrophy,[5] the lungs did not show medial hypertrophy of the small pulmonary vessels, the liver did not show extramedullary hematopoiesis,[6] and the adrenal glands did not manifest brown fat, all of which can be indicative of SIDS, Dr. Shane believes it less likely that Mario died a SIDS death. *Id*.

On the other hand, Dr. Shane found the brain significant because of what he identified as "substantial" or "profound" cerebral edema,[7] which he related to encephalopathy. *Id*. He noted that microscopic examination showed the medium- and small-sized blood vessels, the neurons, and the glial cells were all surrounded by large clear spaces, and identified "scattered gliosis[8] in the cerebral hemispheres." *Id*. He goes on to note the absence of inflammatory cells, and that "brain stem changes of SIDS is [*sic*] not seen." *Id*. Dr. Shane defends his conclusion of cerebral edema, which he links directly to the alleged encephalopathy, by reference to this microscopic examination of autopsy slides and to what he understands to be "significantly increased brain weight." *Id*.

Dr. Shane also places reliance upon the clinical behaviors exhibited by Mario, as reported by his Mother, within the period between his vaccination and untimely death. He focuses on Mario's poor feeding, poor sleeping and wakefulness on a bihoral basis, whining, fussiness, and abnormal reactions (or lack of reactions) to stimuli (e.g., uninterrupted staring, nonresponse to mother's presence) as "unequivocally encephalopathic symptoms." *Id*. By way of exclusion, he also adds:

> There are absolutely none of the expected findings pathologically in sudden infant death. There are none of the petechial[9] findings of sclera,[10] conjunctiva,[11] pleura,[12]

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[5] Hypertrophy is "the enlargement or overgrowth of an organ or part due to an increase in size of its constituent cells." DORLAND'S, *supra*, at 890.

[6] Extramedullary hematapoiesis is "the formation and development of blood cells outside the bone marrow, as in the spleen, liver, and lymph nodes." DORLAND'S, *supra*, at 826.

[7] 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.

[8] Gliosis is "an excess of astroglia in damaged areas of the central nervous system," and diffuse gliosis in particular is "gliosis affecting the whole of the cerebral tissue, or widely scattered through it." DORLAND'S, *supra*, at 778.

[9] A petechia is "a pinpoint, nonraised, perfectly round, purplish red spot caused by intradermal or submucous hemorrhage." DORLAND'S, *supra*, at 1411.

[10] The sclera is "the tough white outer coat of the eyeball, covering approximately the posterior five-sixths of its surface, and continuous anteriorly with the cornea and posteriorly with the external sheath of the optic nerve. DORLAND'S, *supra*, at 1667.

[11] The conjunctiva is the delicate membrane that lines the eyelids and covers the exposed surface of the sclera. DORLAND'S, *supra*, at 409.

[12] The pleura is "the serous membrane investing the lungs and lining the thoracic cavity, completely enclosing a potential space known as the pleural cavity." DORLAND'S, *supra*, at 1451.

> or thoracic organs seen with asphyxial death. In the absence of these findings, this is not sudden infant death or asphyxia.[13]

Id.

Just prior to the entitlement hearing in this case, Petitioner filed another expert report from Dr. Shane, after he had opportunity to review the expert reports filed by Respondent. See Second Expert Report of Dr. Shane, dated 12 January 2007. He cites Dr. Gilbert-Barness' textbook, which incorporates the same data sources for a table compiled within that Dr. Shane also relies upon in making his finding of cerebral edema.[14] *Id*. Indeed, he notes, Dr. Gilbert-Barness' expert report also cedes that Mario's autopsy evidenced cerebral edema, although she concludes that it is essentially mild in nature. *Id*. Also, her report relies upon an abstract of medical literature which itself found cerebral edema uncommon for SIDS cases.[15] *Id*. Therefore, Dr. Shane summates, "her own textbook establishes a somewhat heavy brain and she identifies cerebral edema which by her own reference is inconsistent with a diagnosis of SIDS." *Id*. In the same report, Dr. Shane also critiques Dr. Kohrman's expert opinion because it "uses the police report to negate the clinical findings of the parents which implicate encephalopathy," and because it relies upon Dr. Kohrman's belief that Dr. Gilbert-Barness found "no apparent changes" in the brain, which is inconsistent with her finding of mild cerebral edema. *Id*.

At the entitlement hearing, the parties agreed that Dr. Shane is credible as an expert in the area of pathology, but that, even though "he has performed autopsies on many children," he is not board-certified as a pediatric pathologist. Tr. at 29. Moreover, he is not board-certified in neuropathology, although he has undergone special training in that area, and has practiced in that area throughout his career, adding "by the time it became time to take the board exam, which I would certainly qualify for at the inception, I had already been practicing for 15 years and didn't take the exam." Tr. at 59.

Dr. Shane began by reviewing Mario's autopsy results, noting the importance of examining "the small pulmonary vessels for the medial hypertrophy of those vessels," which is a finding closely associated with SIDS, and pointed out that the lungs were unremarkable in this respect. Tr. at 31. He continued to establish that other hallmarks of SIDS, signs of extraaxillary hematopoiesis in the liver and periadrenal brown fat, were likewise absent. Tr. at 31-32.

In contrast, Dr. Shane found the findings in the brain to be of great significance:

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[13] Asphyxia is the "pathological changes caused by lack of oxygen in respired air, resulting in hypoxia and hypercapnia." DORLAND'S, *supra*, at 165.

[14] That table from Dr. Gilbert-Barness' textbook, attached to Dr. Shane's report, shows the normal brain weight in children of 2-3 months of age to be 516g, the heart at 23g, the right lung at 23g, and the left lung at 35g.

[15] That article abstract, however, did find a correlation between higher brain weight and SIDS cases, although such higher weight was apparently not attributable to cerebral edema. See attachment 2 to Second Expert Report of Dr. Shane, dated 12 January 2007.

> The brain showed the classic perivascular,[16] perineal,[17] [*sic*] and perineuronal[18] clear spaces -- you can have some of this clear space present as the result of the shrinkage of the -- but when it goes beyond a certain degree and when it's associated with some surrounding brain fungiosis [*sic*] both in the gray and white matter, it is significant as kind of the so-called vasogenic[19] edema, that their intestine [*sic*] was present in this case....Vasogenic edema such as we found is commonly seen in the encephalopathy. It sometimes can be seen in trauma, can be seen in infarcts, and can be seen in toxic brain injury. It is not the cytotoxic[20] edema that we see in asphyxia or the cytotoxic changes that we see in ischemia.[21] So this was classic vasogenic edema.
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> There was scattered gliosis. It was not a consistent finding in all of the sections, but there were foci or pockets of gliosis, which is a proliferation of the glial cells of the brain. The glial cells of the brain are the inflammatory cells of the brain, and this indicates that there was an inflammatory process evolving in this brain. And I believe the brain findings were the most significant in terms of possibility. The brain weight of 549 grams, particularly in the case of a child who was only five pounds, two ounces at birth and who at eight weeks was 10 pounds, one ounce, this is not a large baby, so the weight of 549 grams is an increase in brain weight, and that certainly corresponds to the vasogenic edema. Cytotoxic edema while pregnant does not cause any increase in brain weight.
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> ...[W]hat I was seeing under the microscope was toxic cerebral edema. In my practice in neuropathology, I've seen thousands of brains with cerebral edema, and it certainly was notable cerebral edema.

Tr. at 32-34.

Dr. Shane explained further that the brain weight at issue was above the upper end of the normal range, especially when one considers that Mario was "a rather small baby according to today's norms." Tr. at 35. Dr. Shane then referred these clinically-observable traits with the microscopic evidence from the autopsy slides:

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[16] Perivascular means "near or around a vessel." DORLAND'S, *supra*, at 1408.

[17] Perineal means "pertaining to the perineum." DORLAND'S, *supra*, at 1403.

[18] Perineuronal means "surrounding a nerve or nerves." DORLAND'S, *supra*, at 1403.

[19] 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.

[20] 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.

[21] 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.

> But most important, you have to take into consideration what you're seeing under the microscope. If you're not seeing the profound vasogenic edema under the microscope, then you would want to say well, maybe it's -- common and normal. I think it was over normal based on what I see and given the size of this baby.

Id.

Next, Dr. Shane went on to explain why he does not believe Mario's death was in any way causally related to asphyxiation:

> What I was seeing on there is petechial hemorrhages[22] into the [...] organs. That was not present. And another important factor in excluding asphyxia is the fact that asphyxial deaths occur over a short period of time. You know, asphyxia for five minutes will certainly result in brain damage; asphyxia for 10 minutes, unless of course it's a drowning asphyxia full of water, results in death. And looking at these slides under the microscope, in five to 10 minutes, you're not going to get cerebral edema. That brain is not going to swell at all in that very short period of time. It takes a while for the cerebral edema to develop. Cerebral edema develops by gaps forming in the endothelial[23] cells lining the capillaries of the brain, and these little gaps become the site through which fluid from the intravascular compartment seeps into the extravascular compartment. ...[I]t would take longer than the five to 10 minutes that is what is expected in an asphyxial death. So I don't have the gross statement, as I mentioned, and I certainly have -- findings that would not occur in an asphyxia based on the timing of that event.

Tr. at 35-36. Dr. Shane noted a few moments later that, because an infant like Mario is more resistant to hypoxia[24] than someone older, and because the time it would take for a disruption of the blood-brain barrier junction, cerebral edema such as was evident in this case would take 20-30 minutes or more to develop. Tr. at 37-38.

Dr. Shane also discussed what he believed to be the pathologic cause of Mario's death:

> Based on the findings in the brain, an evolving multifocal gliosis and the vasogenic edema and the background of the symptomatology clinically that you had in this child of the whining, [...] the abnormal sleeping pattern, the blank stare, the poor eye contact, the clinical and the pathologic findings that I mentioned certainly are mutually supportive, and they are supportive of a diagnosis of an encephalopathy.

Tr. at 37.

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[22] 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.

[23] Endothelial cells are those made up of endothelium, "the layer of epithelial cells that lines the cavities of the heart, the lumina of blood and lymph vessels, and the serous cavities of the body." DORLAND'S, *supra*, at 616.

[24] 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.

In contrast to the decisive findings supporting cerebral edema and therefore encephalopathy Dr. Shane drew from the microscopic slides, he found none supporting the contention of SIDS: Mario did not have a history of upper respiratory health problems, and did not manifest any of the correlative indicia typically associated with SIDS. Tr. at 38-39. In explaining his disagreement with the autopsy report of the County Coroner, Dr. Manion, Dr. Shane stated that Dr. Manion does not share "the flow volume that [Dr. Shane's] department serves," and that "chances are he didn't have this detailed clinical history at the time when he made his conclusion." Tr. at 38. He added that "unless you've been doing a lot of neuropathology, [these] subtle findings [] sometimes escape the customary medical examiner system." Tr. at 39. Moreover, Dr. Shane continued to elaborate that cerebral edema is not a typical corollary to SIDS, and that even Respondent's expert, Dr. Gilbert-Barness, has noted a finding of cerebral edema in Mario's case. Tr. at 40-41. Even though she does not agree with Dr. Shane's ultimate opinion, and disputes the severity of the cerebral edema, Dr. Shane pointed out, questions of degree relating to cerebral edema are of the subjective realm; what is important is that she has stipulated that the edema was present. Tr. at 42-43. For his part, Dr. Shane later rated his opinion of the cerebral edema as "moderate," or "even borderline severe." Tr. at 65. Additionally, Dr. Shane drew attention to the fact that the "gross findings" one would associate with asphyxiation were absent in Mario's case. Tr. at 44-45.

At the conclusion of Dr. Shane's direct testimony, the Court asked whether his conclusion in support of the Petition was contingent upon the clinical history, as related by Petitioner's testimony. Tr. at 45-46. Dr. Shane explained that, assuming the Petitioner's account to be true, he would conclude in favor of a positive encephalopathy diagnosis; however, assuming that he could not rely on Petitioner's testimony, Dr. Shane would render a diagnosis merely *consistent with* encephalopathy. Tr. at 46-47.

On cross-examination, Dr. Shane defended his reliance on Petitioner's testimony vis-à-vis the police report that seems at odds thereto, stating that the police were not in a skilled position to elicit a detailed medical history from Petitioner:

> You speak to a policeman differently than you speak to your doctor, number one. And number two, your doctor knows the right followup questions to ask to detail a medical history. And good doctors are better at this than not-so-good doctors because they know what followup questions they ask, they have to ask. They can mold a clinical history very, very precisely with a lot of detail. Police don't have the background or training to detail and ask the right followup questions and obtain a history like a medical person would. So I think based on the previous position of the person who was answering the questions -- in this case, a distraught mother -- and the lack of experience and lack of training on the part of police in medical-history taking and the circumstance under which it is taken are certainly terrible, terrible bases for obtaining an accurate, clear, precise, detailed medical history.

Tr. at 51. Dr. Shane added another reason, one more specific to the facts of this case, as to why the statement recorded by the police may prove ultimately inferior to Petitioner's sworn testimony regarding Mario's clinical history: "There are many, many emotional factors that go into the horrible timing of the police taking a medical history, the psyche of the mother, the lack of experience on the part of the police." Tr. at 52.

Next, Dr. Shane agreed with Respondent's assertion that Dr. Manion's autopsy report "didn't make any significant findings in this case," but disagreed with the position that a pathologist would not have diagnosed encephalopathy, if it was evident. Tr. at 57-58. Dr. Shane added that "in this case, [Dr. Manion] did not report any findings at all and speculates that this was a - syndrome, and we wouldn't do that [in Dr. Shane's practice]. Tr. at 58.

Respondent questioned whether the small petechiae found on the thymus gland in the thoracic cavity and the higher weight of the lungs were more indicative of asphyxia than of encephalopathy, to which Dr. Shane responded that no, such signs are typical, and indicative of the agonal process:

> We all become a little bit hypoxic at the time of death, and certainly we're not breathing. And that is sufficient to trigger some kind of petechiae. And you will see findings of petechiae very, very commonly as part of the agonal process.
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> ...
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> In asphyxia, you'd see ...pulmonary edema ...[which] forms very, very quickly and very, very deeply in the lungs when the left ventricle fails....
>
> ...
>
> [The County Coroner noted pulmonary] congestion and focal-chronic inflammation. He is not even identifying pulmonary edema. And congestion is agonal. Your heart doesn't stop beating instantaneously. Your heart reduces output in the last minute or so of death or 30 seconds and you get congestion into those lungs. And it's not edema. It's congestion. And that's an agonal change.

Tr. at 61-63. Dr. Shane also dismissed as unimpressive the putative evidence of focal chronic inflammation in Mario's lungs. Tr. at 63.

Respondent later queried Dr. Shane as to the precise pathological "process" to which Mario succumbed. Tr. at 70. Dr. Shane explained that the rise of edema swells the brain within the confines of the cranium, which creates pressure on the brain; the whole process "compromises the blood supply," can distort the structure of the brain itself, and "interferes with the function of the central nervous system," such that autonomic process of circulation and respiration are negatively affected or ceased. Tr. at 71. Even a slight increase in brain size can mean increased intracranial pressure on the brain, and therefore swelling need not be extreme for death to occur. Tr. at 72-73. Consequently, even mild cerebral edema can cause death, a circumstance Dr. Shane stated he saw "all the time." Tr. at 73. Dr. Shane believes that this process affected the brain first, and that cardiorespiratory death was "a result of the abnormal brain function." Tr. at 73.

Responding to another question posed by Respondent, Dr. Shane stated that, even though cerebral edema may be caused by a number of stimuli, most of those causes are excluded by the facts in this case, leaving only encephalopathy and concurrent vasogenic cerebral edema. Tr. at 74-76.

3. Marcel Kinsbourne, MD

Petitioner submitted an expert report from Dr. Kinsbourne prior to the entitlement hearing. *See* Expert Report of Dr. Kinsbourne, dated 18 July 2006. In stating Mario's history prior to vaccination, Dr. Kinsbourne notes that "he was essentially a well baby, able to kick, vocalize, follow past the midline and smile responsively." *Id*. In discussing the autopsy findings, Dr. Kinsbourne noted (i) the reported microscopic finding of lung congestion and focal chronic inflammation, and (ii) that the brain was described as absent of inflammation, but otherwise undescribed. *Id*. Dr. Kinsbourne contrasts that latter finding with Dr. Shane's finding, based upon microscopic examination, of cerebral edema. *Id*. These data lead Dr. Kinsbourne to conclude the following:

> Cerebral edema is not a feature of SIDS. It is definitive evidence of an encephalopathy. The pertussis vaccine is known rarely to cause encephalopathy. The medical records did not reflect any alternative explanation for the cerebral edema, or any evidence of an alternative cause of death.

Id.

At the hearing, Dr. Kinsbourne was welcomed by the Court as undeniably qualified in expertise to testify as a pediatric neurologist, and Respondent stipulated by accepting him as a pediatric neurologist without cavil or question. Tr. at 81. He summated his expert medical opinion, "to a reasonable degree of medical probability," stating, "My opinion is that Mario Perez's death was the consequence of an encephalopathy and that that encephalopathy was most likely due to the pertussis vaccine." Tr. at 82. From there, Dr. Kinsbourne explained what he observed to be the mechanism by which such causation could occur:

> I have three pillars on which my opinion rests. One is the presentation of the DPT vaccine. The second is the time interval of less than 48 hours between the [vaccination] and the child's death. And third is the evidence for encephalopathy -- which in this circumstance are the -- and if I may, I would like to tell you about what kind of effect it had. Although Mrs. Perez's testimony that you had this morning is consistent with my opinion that an encephalopathy occurred, it is not necessary for that opinion.

Tr. at 82-83. Later, Dr. Kinsbourne added that the testimony that Mario became "apathetic" and "staring" was consistent with the occurrence of an encephalopathy. Tr. at 88. He continued in explaining his opinion as follows:

> The other thing is the -- the weight of the brain. On the one hand, the weight is at the upper end of the range given for children of about that age. On the other hand, this child is a lot lighter than the average child at that age. So one could either take the -- of the table or make a subjective adjustment for the lower weight. The issue, of course, is that there are no tables for children of Mario's weight. In my point of view, the evidence for cerebral edema comes directly from the operation of cerebral edema by those pathologists. And whether the brain weighs more than average but is within a certain range or is outside a range is immaterial.

Tr. at 83-84. Dr. Kinsbourne referenced the cerebral edema found by the pathologist experts in this case as evidence of encephalopathy, edema which, he said, "is not something that a neurologist would suspect after five or eight minutes," but is instead "indicative of a more enduring process, which is indeed compatible with a pertussis vaccine encephalopathy." Tr. at 84.

In continuing to discuss the mechanism of injury, Dr. Kinsbourne relied upon the known "neurotoxic effect of pertussis vaccine" component within the DTaP vaccine. In fact, Dr. Kinsbourne went on to state that "edema is only a part of how the pertussis vaccine in my opinion killed this child," adding that death would be also due to "impairment of function of the neurons by the toxin [the pertussis vaccine]." Tr. at 84-85.

Regarding the putative diagnosis of SIDS, Dr. Kinsbourne elaborated on the ambiguity of the label, expressing that asphyxiation is only one potential causal factor that could explain SIDS cases, along with infection, or even cerebral edema from encephalopathy. Tr. at 86. Later, he explained that, as he understands things, SIDS is becoming subdivided into discernable causal factors, as they are increasingly understood, and thereby taken out of the "unknown" rubric of SIDS. Tr. at 89. Dr. Kinsbourne firmly stated that a diagnosis of SIDS is insufficient as a etiological explanation to establish a physiologic mechanism. Tr. at 89-90.

On cross-examination, Dr. Kinsbourne reiterated and clarified that the three primary bases of his opinion in support of causation were: (i) the fact that Mario was vaccinated with the DTaP vaccine on 17 December 2003, (ii) the time interval between vaccination and Mario's death, which itself occurred by the morning hours of 19 December 2003, and (iii) the autopsy findings of the various pathologists who examined Mario and his autopsy slides, and, more particularly, those findings enunciated by Dr. Shane. Tr. at 91-92, 97. Dr. Kinsbourne noted of the autopsy report prepared by the County Coroner that "it simply doesn't mention any kind of detail that would help me believe that he looked at it carefully and [with] expertise." Tr. at 92-93. Therefore, he went on to say in response to a question from Respondent, without the expert opinion offered by Dr. Shane and/or Dr. Gilbert-Barness expressing a finding of cerebral edema, he would not be in a position to opine that it was more likely Mario had suffered an encephalopathy than not. Tr. at 93.

Dr. Kinsbourne admitted that cerebral edema can result from asphyxiation, but noted that "once cerebral edema is observed, there is a differential diagnosis for that," and that "[a]fter having done a differential diagnosis, [he] didn't find any other diagnosis other than encephalopathy." Tr. at 94. Dr. Kinsbourne also rebuffed the idea of asphyxiation as an explanation for causation in this case because "five minutes are not enough to cause cerebral edema," as the development of cerebral edema would have taken longer to develop. Tr. at 94.

Regarding the question of Mario's "relatively heavy" brain weight, and whether or not Mario's brain was within the range of "normal", Dr. Kinsbourne maintained that, given the fact "that this child's brain is at the end of the normal range or outside it...is consistent with an encephalopathy," adding that his reliance on Dr. Shane's conclusion limits his need "to rely upon something as superficial as the weight of the brain." Tr. at 96.

On redirect examination, Dr. Kinsbourne elaborated more fully the nexus between the three primary bases upon which he rests his expert opinion. Having restated the two day time period between vaccination and death, he said the first question to answer was whether known medical science "could provide a biologically plausible mechanism that would connect" any of the vaccines administered with Mario's death. Tr. at 98. He continued, "And it was generally acknowledged that the cytotoxin, which is to be found in the [DTaP] vaccine, is neurotoxic and is a biologically plausible cause of encephalopathy." *Id*. This led him to conclude that the Pertussis toxin's known neurotoxic effects in rare cases could be culpable for Mario's death via encephalopathy, because such effects would take their toll within a few days. *Id*.

4. Enid Gilbert-Barness, MD

Respondent filed one expert report from Dr. Gilbert-Barness, filed prior to the entitlement hearing. *See* Respondent's Exhibit (Resp. Ex.) D, Expert Report of Dr. Gilbert-Barness, dated 25 October 2006. In her report, she summarizes her findings from the autopsy slides thusly:

> All organs were intensely congested. There was subpleural[25] hemorrhage in the lungs [citing particular slide]. The brain showed a mild degree of edema. The brain weight of 549 grams was not significantly heavier than normal (normal brain weight for 2-months-old was 489-560 grams), and there was no apparent changes in the brain.

Id.

Dr. Gilbert-Barness also based her opinion upon the presumption that "Mrs. Perez laid Mario on his stomach when she put him in the basinet [*sic*]," such that "[a] few moments later Mr. Perez heard his wife screaming," and that Mario was already stiff at that point, and was "cool to the touch and his lips were blue." *Id*. She seems to assign weight to the responding policeman's statement, that, "It appears from the outset that the deceased may have just suffocated himself in the bassinet while sleeping. There was no pillow in the bassinet but it was not firm either." *Id*. Based upon these presupposed facts, Dr. Gilbert-Barness concludes that "this infant died as a result of sudden infant death due to asphyxia in the prone position. I see no evidence of a vaccine[-]induced encephalopathy." *Id*.

In her report Dr. Gilbert-Barness took issue with the expert opinion report of Dr. Shane, filed by Petitioner, on the grounds that "[t]he changes in the organs in SIDS cases are not consistent," noting that she did not find "evidence of clear spaces around neurons or gliosis," and that the cerebral edema was "only slight–entirely consistent with sudden infant death." *Id*. She concludes her report by opining that the weight of Mario's brain "was within normal limits." *Id*.

At the hearing, Respondent offered evidence that Dr. Gilbert-Barness is a pediatric pathologist, a professor of pathology and molecular medicine, pediatrics, and obstetrics and gynecology, and an emeritus professor, with "extensive experience in sudden infant death syndrome," evidenced by her having served on a National Institute of Health committee devoted to

[25] A subpleural hemorrhage is "the escape of blood from the vessels" below the serous membrane which lines the thoracic cavity and within which the lungs are situated. DORLAND'S, *supra*, at 834, 1451, 1780.

that topic and having penned copious writings on the topic. Tr. at 101. She has been an examiner in pediatric pathology for a period of several years and president of the Society for Pediatric Pathology and the International Pediatric Pathology Association. *Id*. She is board-certified in clinical pathology, anatomic pathology, and pediatric pathology, and has performed "well over 10,000" autopsies, almost all of them on infants. Tr. at 101-02. She agreed with Respondent's statement that it is "important in reviewing reports and in performing autopsies that if there are going to be diagnoses made for a child that [the diagnosing] pathologist be trained in pediatrics and perhaps specialize in pediatric pathology" as "an important distinction." Tr. at 106.

Dr. Gilbert-Barness refuses the contention that "Mario suffered an encephalopathy which was the cause of his death on December 19, 2003." Tr. at 102-03. She set forth the reasoning for this refusal as follows:

> First of all, I think the symptoms that his mother described after the death of this child and following the vaccination were those that you ordinarily see in any infant who receives vaccinations. I've seen it many, many of the other cases. I've had five children of my own, I've got 15 grandchildren, and I've seen it in virtually all of them. So I don't think that that was particularly abnormal. That's as far as the critical issue is concerned.
>
> I think that the diagnosis of encephalopathy is a critical diagnosis, perhaps supported by the neuropathological changes. First of all, the cerebral edema that was present in this child was minimal to mild at most, and I believe that this child died from what we call sudden infant death. Many of those cases are sudden infant death syndrome.
>
> And those cases I think now have been very well defined as probably asphyxial death. [W]e believe now that many of those deaths that were called SIDS, that are still called SIDS right now, are actually others.

Tr. at 103-04. Dr. Gilbert-Barness agreed that she believes that many SIDS deaths are probably caused by asphyxia. Tr. at 104.

Dr. Gilbert-Barness disagrees with Petitioner's offered testimony that Mario's brain weight of 549 grams is a substantially increased brain weight for his age grouping at two months. Tr. at 106. She contrasted the table included in her book which was mentioned by Dr. Shane, Appendix 29, to another in her book, Appendix 37, which she averred was "more accurate." Tr. at 106-07. Due to what she observed as only mild edema, she stated that she did not "put a great deal of emphasis in this case on brain edema." Tr. at 107. Similarly, her recollection of the extant medical literature is that there is a greater incidence of increased brain weight in SIDS cases, and therefore, "this is entirely within the range of all the normal brains but also in SIDS brains." Tr. at 107-08. Based upon this discussion, Dr. Gilbert-Barness concluded that, even if Mario's brain was "on the high end of average," the involvement of encephalopathy in Mario's death "would be highly unlikely." Tr. at 108.

Dr. Gilbert-Barness next described what pathologic evidence she would expect to find if there existed in Mario's case a fatal encephalopathy:

> I would expect to see considerable cerebral edema. In a two-month-old, there may not be herniation,[26] but there may be coating of the brain stem, because the systems are open at that period of life and there may not be a herniation, but certainly there would be compression of the brain, and I don't think this child demonstrated that. Certainly from the -- point of view, neither Dr. Shane nor I saw the gross specimen ourselves.
>
> And I might add that I certainly do respect Dr. Shane's opinion. I think he gave a very eloquent description of types of edema in the brain. I do note, however, that we really disagree.

Tr. at 108-109. Dr. Gilbert Barness also stated that gross evidence of edema should also have been apparent just by observing the structure of the brain at autopsy, observable as massive swelling by any reasonable pathologist. Tr. at 109-10. She noted her disagreement with Dr. Shane, who, she says, "described a much more severe degree of damage which [she] certainly did not see." Tr. at 110.

As to whether the more limited degree of cerebral edema she noted in her report was sufficient to prove fatal, she stated that, "It's all part of sudden infant death -- sudden infant death syndrome that we described, and as I mentioned -- some degree of edema is almost always present in sudden infant death," because "many of these cases are -- now clearly appear to be an asphyxial death [in which] there is obstruction of the respiratory efforts, and these babies do develop a little bit of edema." Tr. at 110-11. Prompted by this testimony, the Court questioned Dr. Gilbert-Barness whether she believes that the "mild" edema she observed was more appropriately understood as arising from the agonal process, to which she responded, "Well, it occurs in asphyxia and it occurs in what we call sudden infant death syndrome. Yes, it's probably correct." Tr. at 111.

Respondent followed up on this statement by asking whether Dr. Gilbert-Barness "would expect to see the death was the result of asphyxia," to which Dr. Gilbert-Barness responded, "Or sudden infant death." Tr. at 113. Questioned whether she used the terms asphyxiation and sudden infant death interchangeably, Dr. Gilbert-Barness explained that "Well, most pathologists still call it sudden infant death syndrome, but I prefer to say sudden infant death and leave it at that or say consistent with an asphyxial death or positional asphyxia or whatever it is." Tr. at 113-14.

In response to another question on direct examination, Dr. Gilbert-Barness explained the evidence from the autopsy that were consistent with death by asphyxiation:

> Well, in asphyxia or sudden infant death syndrome, you would sometimes find petechial hemorrhages, which were described in the thymus of this infant by the medical examiner. ... They are tiny little hemorrhages in the thymus and also in other areas. In microscopic examination, I certainly saw some fluid -- hemorrhage in the lung. Again -- very frequently -- in a SIDS death or an asphyxial death. And this is a very minimal or mild degree of cerebral edema that was present. So I think that all

[26] Herniation is "the abnormal protrusion of an organ or other body structure through a defect or natural opening in a covering, membrane, muscle, or bone." DORLAND'S, *supra*, at 844.

> of these features are certainly most consistent with an asphyxia or sudden infant death syndrome.

Tr. at 114. She continued, discussing the autopsy report's reference to chronic pulmonary inflammation, which Dr. Shane contested:

> There was chronic inflammation in the lungs, and in 75 percent of so-called SIDS cases, are in some degree minor, but some degree of inflammation, which this baby had. So I think all of the patients are so typical of a SIDS or an asphyxial death.

Tr. at 114-15. Dr. Gilbert-Barnes likewise differed from Dr. Shane's finding of gliosis in the brain, but stated that, if there was indeed such gliosis, such gliosis "would certainly be consistent with a SIDS death." Tr. at 115.

Regarding the higher than normal weight of the lungs recorded in the autopsy report, Dr. Gilbert-Barness reflected that "certainly one can conjecture that some of it was due to the chronic inflammation of the lungs, but that again is consistent with SIDS or asphyxia." Tr. at 115.

In contrast, in describing what evidence would be necessary to support a conclusion that an encephalopathy had killed Mario, as reflected by examination and brain weight, Dr. Gilbert-Barness stated that "the brain would show significant edema, which this did not. And I think pathologically, there would be certainly evidence of much more severe edema." Tr. at 116. In response to Respondent's concluding question on direct examination, Dr. Gilbert-Barness agreed that "there is no objective evidence in the record either from the report done by the medical examiner or [her] view of the slides that would show objective evidence of an encephalopathy," and that "had the edema been severe enough to cause death, that's something that the medical examiner would have or should have seen upon gross examination." Tr. at 116.

When the Court asked Dr. Gilbert-Barness to assume as true the account proffered by Petitioner's fact witness testimony, she responded that her opinion would remain unaffected, because the symptoms described there were "universal" and reflected "a pretty much normal sort of response following a vaccination." Tr. at 119. "In this case," she continued, "it might have been worse than usual, but this would not affect [her] testimony in [her] opinion of the cause of death." Tr. at 119. On cross-examination, however, when asked whether the account of blank staring in the fact-witness testimony indicated a "significantly decreased level of consciousness," Dr. Gilbert-Barness replied that she could not really render comment on that question, but, when pressed to answer, replied that she would not consider the blank staring to signify a symptom of an encephalopathy. Tr. at 121-22.

When asked on cross-examination to identify the authoritative sources for her contention that asphyxia can produce cerebral edema, she referenced "any textbook" and her experience, but "couldn't tell you anything more than that." Tr. at 124-25. More specifically, she stated that she was relying "mostly" on her experience, but noted that she has authored 16 textbooks. Tr. at 125.

When asked by the Court to state her professional opinion of what actually caused Mario's death, Dr. Gilbert-Barness replied:

I believe that it's what most people would call sudden infant death syndrome. I think that many of those are asphyxial. And this baby was just lying in the prone position on a soft mattress, and that certainly is a high-risk factor for sudden infant death syndrome.

Tr. at 125-26. At this, the Court asked to what degree of medical probability she held this opinion, and whether it surmounted a preponderant quantum, to which she interlocuted:

Well, you see, as I've tried to define sudden infant death syndrome, now almost certainly a large percentage of them are actually asphyxial deaths, positional asphyxia, lying in a prone position on a soft mattress and all the other features that can contribute to it. So I think that this infant died from what most people and -- call sudden infant death syndrome. I think it was the positional asphyxial form of sudden infant death.

Tr. at 126-27. This explanation led to the following interchange:

THE COURT: -- Doctor, what I'm trying ascertain, because I understand your testimony, but what I'm trying to ascertain is by your analysis, is the preponderance of the evidence that what caused this child's death asphyxia? Not sudden infant death syndrome, because that includes a lot of things.

THE WITNESS: Right.

THE COURT: But in this case, asphyxia.

THE WITNESS: Yes, I would agree with that.

THE COURT: Okay. So you can say that from your perspective?

THE WITNESS: Right.

THE COURT: Okay.

BY MR. GALLAGHER:

Q And do you characterize that as sudden infant death syndrome?

A Well, I myself would describe it as sudden infant death and modify it by saying a positional asphyxia.

THE COURT: Okay. Mr. Gallagher, the Court understands her testimony as being she isn't saying it is SIDS, but it is SID, that is, there's a sudden infant death.

THE WITNESS: Right.

THE COURT: And that her explanation for this death is asphyxia. And if the Court understood what she said a moment ago, more likely than not by a preponderance of the evidence, but it doesn't sound like it's a massive preponderance, but it's a preponderance from her perspective.

MR. GALLAGHER: Okay. Just one question.

BY MR. GALLAGHER:

Q So the mechanism of death is putting the baby face down in the crib? Is that the mechanism that we're talking about?

A Yes. It's obstruction of the airway when the baby is lying in a prone position on a soft mattress. If you obstruct the nose of a baby, it cannot breathe. They are obligate nasal breathers. So if you squash the nose, and the nasal passage in a baby is very soft, you can squash the nose and obstruct the breathing process, and therefore, that becomes an asphyxial-type death, positional asphyxia.

Tr. at 127-129.

Cross examination continued on potential alternative *causata*:

Q ...Do you believe that this child died from gastroesophageal reflux?

A No, I do not.

Q Okay. How about chronic focal lung inflammation?

A Right.

Q You do agree with that?

A Yes.

Tr. at 130.

5. Michael Kohrman, MD

Respondent likewise filed an expert medical opinion report from Dr. Kohrman. *See* Resp. Ex. A, Expert Report of Dr. Kohrman, dated 5 November 2006. In his report, Dr. Kohrman pointed out in the autopsy report findings that "congestion was noted along the posterior dura of the vertabral column," but that the anatomy of the brain parenchyma[27] was normal, "without evidence of hemorrhage, trauma, or tumor," and "no significant inflammation" of the brain was noted. *Id*. In critiquing Dr. Shane's expert report, Dr. Kohrman noted that Dr. Shane "found no evidence of inflammation," adding that the influx of fluid in the brain is "certainly consistent with post[-]mortem changes we often see after SIDS." *Id*.

Dr. Kohrman's reading of Dr. Gilbert-Barness' expert report is that Mario's brain weight was "not heavier than normal," and that, consistent with the autopsy report, Mario died of SIDS. Dr. Kohrman bases his opinion upon this reading: "Given the normal brain weight and no apparent changes in the brain she concluded that there was no evidence of vaccine[-]induced encephalopathy." *Id*. Dr. Kohrman also states as a basis for his expert opinion that he presumes the factual account of the police report is alone correct, and not Petitioner's sworn affidavit to the contrary. *Id*.

Dr. Kohrman cited to medical literature that "found a mean of approximately 520 grams and interval of 360-640 grams." *Id*. Unfortunately, he did not explain that statement or that finding, nor did he relate it to the present case, nor is the attached graph clear enough to read or self-explanatory.

[27] Parenchyma is "the essential elements of an organ." DORLAND'S, *supra*, at 1371.

See Resp. Ex. B. As such, the Court is unable to assign meaningful weight to this reference in Dr. Kohrman's report.

Lastly, Dr. Kohrman's expert report states that he takes as a "given" a "history of gastroesophageal reflux and chronic focal lung inflammation." *Id*. Based upon this presupposition, and those others listed *supra*, Dr. Kohrman concludes as follows:

> [I]t is much more likely that this child died of an event related to gastroesophageal reflux than from vaccine injury, as there is no evidence of an acute encephalopathy clinically, based upon the reports from the police of parent statements at the time of the child's death that were independently recorded. In addition[,] independent pathology review demonstrates normal brain weight and no changes in the brain. As such, there is no evidence of encephalopathy in this case, and no evidence of causation in fact.

Id.

At the hearing, Respondent offered Dr. Kohrman as "board-certified in pediatrics, in child neurology with special competency in child neurology," with "a subboard [certification] from the American Board of Neurology and Psychiatry in clinical neurophysiology," as well as independent board certifications in clinical neurophysiology and sleep medicine. Tr. at 136. In his practice, Dr. Kohrman says he treats children suffering from encephalopathy on a daily basis. Tr. at 137. At SUNY Buffalo, he directed a "SIDS center" such that he is now "very comfortable with the diagnosis of SIDS and the care and treatment of children with SIDS." *Id*.

On direct examination, Dr. Kohrman first considered the facts recounted in Petitioner's proffered fact witness testimony to counter their use as manifestations of encephalopathy. Tr. at 138. He noted that, according to his understanding of Petitioner's testimony,[28] even though the amounts consumed at feedings were halved (from four to five oz. to two oz.), feedings occurred more often (every two hours, instead of every four or five hours), such that "feedings really didn't change very much." Tr. at 138. Likewise, said Dr. Kohrman, Mario's irritability and crying, as reported in Petitioner's testimony, "is very typical postvaccination." *Id*. Moreover, Dr. Kohrman did not assign significance to the accounts of nonresponsive, glazed-over staring at bathtime, but speculated that Mario "was very soothed during the bath, and that doesn't necessarily imply an encephalopathy per se." *Id*. A moment later, however, Dr. Kohrman stated the following:

> [I]f I take Mrs. Perez's testimony word for word, then yes, I would come back and say this is clinical evidence of an encephalopathy as defined by the vaccine table. If I take what she said and place that in the context of other children with a vaccination, there is not clear evidence of encephalopathy present.

Tr. at 139.

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[28] The Court notes that Dr. Kohrman assumed that Mario was feeding every two hours, a fact which does not seem to be reflected in the affidavit filed or the testimony presented at the hearing; Petitioner's testimony was simply that Mario woke every two hours in the period following vaccination, and that when he fed, he consumed only one to two oz. of formula, instead of his usual four to five ounces. *Cf.* Affidavit of Frances Perez; Tr. at 16.

Dr. Kohrman stated his agreement with Dr. Shane that a contemporaneous medical record is more reliable than a contemporaneous police statement for the purpose of developing a clinical history; nonetheless, Dr. Kohrman noted the absence of a clinical history recorded by medical personnel as justification for relying on the testimony reflected in the police reports in the formation of an opinion of diagnosis. Tr. at 140. His rationale for focusing on the police reports instead of Petitioner's proffered fact testimony is that "*typically*, when someone is distraught, they are going to go for anything they can possibly put their hands on as a cause for the child's problems," because parents are prone to "overreact to what happened and pick up every minutiae [*sic*] of what happened, because during that time, they're so afraid of things, that they did something wrong," and thus skilled questioning to elicit full testimony is not required. Tr. at 141 (emphasis added).

Turning to the evidence presented by the autopsy report, Dr. Kohrman elucidated the "objective evidence" he would require in order to diagnose encephalopathy:

> I would expect to have seen severe swelling of the brain. I would expect to have seen some inflammation of the brain. I would expect to have seen some pathologic changes described by that pathologist, either gross or microscopic, none of which were seen by the medical examiner.

Tr. at 142. Dr. Kohrman added that "most medical examiners would not miss these types of pathology." *Id*. Moreover, in order to diagnose encephalopathy severe enough to be fatal, Dr. Kohrman stated that he would expect to find "significant cerebral edema" and "coning of the brain or brain stem." *Id*.

Regarding the contentions of greater brain weight by Petitioner's expert witnesses, Dr. Kohrman countered that even when a child's growth is otherwise stunted, brain growth remains near constant, such that "we will often see the head circumferences preserved in the absence of height and length." Tr. at 143. This led him to the following discussion:

> He was 55 centimeters in length according to the medical examiner's office, which is exactly right there as the average weight, [*sic*] which would suggest that his brain would be not small, but if anything, it would be the same size. It would be average at the least.

Tr. at 144. Respondent followed up on this point by asking Dr. Kohrman's opinion whether, based solely upon the police reports, medical records, and autopsy report, he thought there was any objective evidence of encephalopathy: he did not. Tr. at 145. Even adding to that grouping the findings of Dr. Gilbert-Barness (including her finding of mild cerebral edema), his conclusion was the same. *Id*.

Regarding Petitioner's experts' focus on cerebral edema, Dr. Kohrman opined *per contra*:

> Cerebral edema can come from any cause of death. Once the child dies, there is going to be brain swelling. And depending on how long it takes from the time the child dies to the time that the child's brain is refrigerated, then cut, there will be varying degrees of swelling of the brain.

> Vasogenic edema tends to be a little different than the cytotoxic edema, which tends to be more related to the time course. But vasogenic edema of a very mild nature would be seen with an asphyxial event, as Dr. Barness has said; it could be from toxic, it could be from metabolic causes, it could be from a myriad of different causes in this child.

Tr. at 144. Indeed, Dr. Kohrman would expect to find a mild amount of cerebral edema in the postmortem results of any infant. Tr. at 145.

Dr. Kohrman stressed that "encephalopathy is a clinical diagnosis and not a pathologic diagnosis," and therefore a conclusion based on the autopsy slides would be incomplete, because "[w]hat you see are a number of different changes that are consistent with encephalopathy, but in no way do those changes mean an encephalopathy has occurred." Tr. at 145-46. He added that the changes evidenced by microscopic analysis of the autopsy slides "may be related to encephalopathy, [] may be related to asphyxia, [] may be related to a multiple carboxylates [*sic*] deficiency[29]... [or] may be related to X, Y, or Z disease." Tr. at 146. Thus, he concluded: "So the pathologic changes that Dr. Shane reports, that Dr. Barness reports, do not in and of themselves in any way diagnose an encephalopathy," but only "document pathologic change." *Id*.

Next, Dr. Kohrman's testimony turned to what, in his opinion, was the probable cause of Mario's death. Dr. Kohrman thought that "the preponderance of evidence that we have based on the child's sleep position, based on the lung findings on autopsy, would make asphyxia more than 50 percent likely the cause of this child's death." Tr. at 146. The Court then questioned Dr. Kohrman why asphyxia was not diagnosed in the autopsy report, in lieu of the generic categorical SIDS, to which Dr. Kohrman responded that, due to the transitional state of medical science in this area, emerging studies have only recently concluded that "SIDS really is a positional effect." *Id*. This begs the question "what causes death in these positional changes," and Dr. Kohrman says the answer is that "asphyxia is the most likely cause." Tr. at 147. Dr. Kohrman supported his answer with reference to epidemiological evidence from changes in how a European government has instructed its citizens how to place infants into bed, and the changed incidence of SIDS in that country.[30] *Id*.

The next subject addressed by Dr. Kohrman on direct examination was the concern he raised of gastroesophageal reflux, which he believed "contributes to a concern about lung disease." Tr. at 148. He explained:

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[29] A multiple carboxylase deficiency is "an inherited aminoacidopathy correctable by biotin therapy and due to deficiency of either holocarboxylase synthetase or biotinidase, which causes deficiency of activity of the biotin-containing carboxylases...; it is characterized by metabolic ketoacidosis, organic aciduria, hyperammonemia, and variable manifestation of breathing difficulties, hypotonia, seizures, ataxia, alopecia, skin rash, and developmental delay." DORLAND'S, *supra*, at 291.

[30] The Court pauses to note that, although epidemiological evidence may bear upon the question of "can it?" (i.e., can a vaccine actually cause the injury alleged), its utility is minified on the question of "did it?" (i.e., did the vaccine actually cause the injury alleged in the instant case). *Cf. Augustynski v. Secretary of HHS*, Case No. 99-0611V, 2007 WL 3033614 (Fed. Cl. Spec. Mstr. Sep. 28, 2007) citing *Capizzano v. Secretary of HHS*, 440 F.3d 1274, 1325 (Fed. Cir.2006).

> Certainly his gastroesophageal reflux could explain the chronic multiple foci of inflammation. If he's having some aspiration from the reflux, this certainly would lead to problems. It could also decrease his ability to oxygenate at night given these areas of chronic inflammation and as such put him at greater risk when he's lying on his stomach to stop breathing.

Tr. at 148-49. However, Dr. Kohrman agreed that, despite his opinion that gastroesophageal reflux could pose greater risk to Mario, he was not opining that such condition was the preponderant cause of Mario's death. Tr. at 149.

Dr. Kohrman concluded his direct testimony by clarifying the statement in his written report that "independent pathology review demonstrates normal brain weight and no changes," by adding the modifier "significant" to that statement. Tr. at 149.

On cross-examination, Dr. Kohrman clarified that he is a pediatric neurologist and not a pathologist, and that he relied upon the autopsy report, Dr. Gilbert-Barness' opinion, and the supposition that there was no significant change in brain weight to form his opinion that "there is not significant cerebral edema to cause an encephalopathy and death in this child." Tr. at 150. Dr. Kohrman conceded that pertussis "clearly" is capable of causing encephalopathy, as evidenced in medical literature, but reasoned that such did not occur here because "there is no significant edema here to produce death." Tr. at 152. When asked the level of edema that would need to be present to result in death, Dr. Kohrman responded, "There have to be moderate to severe changes in the brain seen by the pathologist." *Id*. Asked to elaborate on those changes, he explained:

> Those would be the changes that Dr. Shane described in terms of what we would see in terms of vasogenic and cytotoxic edema. We would expect to see some physical changes in the brain consistent with significant cerebral edema, and we would expect to see significant changes in brain weight, significant increases in brain weight.

Id.

6. John J. Shane, MD

Dr. Shane was recalled at Petitioner's option. Tr. at 156-57. Regarding the contention of Respondent's experts that cerebral edema is typical for asphyxial deaths, Dr. Shane responded that the edema present in that case is cytotoxic edema, and not vasogenic edema, a distinction that, in his experience, a neuropathologist could be expected to identify, but one which a general forensic pathologist might not. Tr. at 157-58. He further dissected this distinction thusly: "So I would disagree that, number one, a forensicologist would identify this cerebral edema..., and number two, that a generalist in pathology or a forensic pathologist ... would necessarily separate out vasogenic edema from cytotoxic edema, which we as neuropathologists do." Tr. at 158.

Next, Dr. Shane spoke in rebuttal of Dr. Gilbert-Barness' focus on the weight of Mario's lungs, stating that, based on the standard measures used in his office, Mario's lung weight was only 13 grams more than the standard. Tr. at 159. He also addressed the absence of other indicators of asphyxial death. *Id*.

> So we don't have the massive increase in lung weight. We have congestion. Congestion is agonal. What we didn't have in those lungs was the petechial hemorrhage and asphyxia. We didn't have it in the pericardium. We didn't have petechial hemorrhages in the sclera, in the conjunctiva. Now these are issues that forensic pathologists do identify, and in this case, they were not reported.

Tr. at 159.

Regarding Dr. Gilbert-Barness' testimony that the cerebral edema she did find in Mario's autopsy results was too mild to be fatal, Dr. Shane challenged that idea as lacking support in standard medical literature. Tr. at 159-60.

> To the contrary, the textbook statements are that cerebral edema is dangerous, and it's dangerous mild, moderate, or severe. It gets more dangerous if it's severe, but nonetheless there's no textbook support that I'm aware of that would say cerebral edema has to be severe to cause death. That is something I would take issue with.

Tr. at 160. Dr. Shane also questioned Dr. Gilbert-Barness' statement that the symptoms which Petitioner alleges Mario exhibited were 'universal', contending that the "loss of focus, staring at the ceiling, not making eye contact" and other described symptoms seemed a vaccine-related symptomatology. *Id*. Based upon that symptomatology, and the evidence culled from the autopsy, Dr. Shane explained his reasoning:

> I think that we as neuropathologists separate out the cerebral edema. We do make a difference, and that difference does establish causation. And again, whatever you find in that cerebral edema you've got to put together with the rest. You've got to put it together with well, was there scleral -- petechiae. There are customary findings in asphyxial death. Was there pleural pericardial edema petechiae. Again, there are customary findings in asphyxial death, and they certainly were not present here.
>
> Dr. Gilbert-Barness did say that encephalopathy is a clinical diagnosis, but then she had the findings at autopsy that would suggest encephalopathic process, she would say consistent with encephalopathy. That's exactly why we were in agreement.
>
> And in this case, what we have in the clinical data, the presentation of this child, was the symptoms that I talked about before, that you go beyond the consistent with and you make a clinical and pathologic diagnosis of encephalopathy. And I believe that's where we are here.

Tr. at 160-61.

Respondent challenged Dr. Shane's self-identification as a neuropathologist on cross-examination, given the fact that Dr. Shane is not board-certified in that sub-specialty. Tr. at 162. Dr. Shane responded that he received special training in neuropathology during his residency, during a time when neuropathology was subsumed within general pathology, and was not specialized distinctly therefrom, as it is now. Tr. at 162-63. Even though he has never taken a Board exam, he has been practicing neuropathology his entire career, a 40-year practice in that specialty. *Id*. When pressed by cross-examination, he conceded that he was not board-certified as a neuropathologist, but added that neither was Dr. Gilbert-Barness. Tr. at 164.

Regarding the degree of cerebral edema which would be sufficient to cause death, Dr. Shane explained that even mild cerebral edema can "absolutely" be fatal:

> Mild cerebral edema is dangerous. In review from learned textbooks like the textbook by Ruben and Barber, that edema of the brain is dangerous.... There is little room for any expansion of the brain inside that cranial wall, and any mild cerebral edema still causes increased intracranial pressure. And what happens is the growth structure of the brain under increased pressure lays the groundwork for functional abnormalities. And then functional abnormalities can be brain-stem abnormalities, and they do and can cause death.

Tr. at 164-65. Dr. Shane also stated the general premise, that "a brain with significant edema is going to be significantly heavier." Tr. at 164.

Following these statements made by Dr. Shane, Respondent's Counsel asked, if cerebral edema had caused death, what evidence would be likely found postmortem, whether by gross or microscopic examination. Tr. at 165. To this, Dr. Shane responded:

> The evidence microscopically of the edema, of course. And the microscopic findings we talked about before are present. There is microscopic vasogenic edema categorically in this case.

Tr. at 165-66. Asked further why his opinion should differ from the examining coroner and Dr. Gilbert-Barness, and why the coroner did not note the existence of cerebral edema, Dr. Shane replied:

> Well, first of all, the medical examiner registered a brain weight....I examined the brain because I am more likely to find these subtle findings that [medical examiners] don't find. And with all due respect, Dr. Gilbert-Barness is a good pediatric pathologist; she's not a pediatric neuropathologist.

Tr. at 166-67. He continued, stating that, because of his experience in brain examination, he found gliosis and vasogenic edema, and that even the examining coroner reported (what Dr. Shane considers) the greater than normal brain weight. Tr. at 167.

At that point, Respondent returned to question Dr. Shane on the process by which cerebral edema can cause death, to which he responded:

> [C]erebral edema causes the brain to have a larger size.... And with any degree of cerebral edema, the limited space in the cerebral wall becomes compromised. There is increased pressure, and increased pressure phenomena can cause death, whether it's mild, moderate. Severe cerebral edema, that universally causes death. The risk of death with cerebral edema increases with increased degree of severity of cerebral edema. But nonetheless, any degree of cerebral edema can functionally cause death.

Tr. at 169. Respondent questioned further on how death is caused by this pressure, and Dr. Shane read from page 299 of a text by Ruben and Barber:

> "Edema of the brain is dangerous because the confined space of the cranium allows little room for expansion. Increased intracranial pressure from edema compromises

> the blood supply, distorts the structure of the brain, and interferes with the function of the central nervous system."

Tr. at 170.

Dr. Shane's rebuttal testimony concluded on the topic of gliosis, and Respondent queried how long it takes for gliosis to develop, to which Dr. Shane responded:

> The type of isomorphic[31] gliosis we see here certainly will be evident in 24 to 48 hours. And as was evident here, it wasn't global; it was focal. I believe it was evolving. As I said before, I believe that if this child had lived on another 48, 72 hours, the gliosis would have been much more prominent and much more global in its occurrence.

Tr. at 170-71.

7. Marcel Kinsbourne, MD

Dr. Kinsbourne was also recalled for additional testimony. Tr. at 171. On direct examination, Dr. Kinsbourne raised three specific points. First, he disputed comments made by Respondent's experts that a lowered level of consciousness was a common side-effect of vaccination, and pointed out that studies of adverse effects following the first two days of pertussis did not list decreased consciousness among the phenomena typically associated. Tr. at 172. His point was that, if the Court accepted the fact witness testimony offered by Petitioner, the phenomena of decreased consciousness attributed to Mario postvaccination cannot be dismissed as merely a normal response to the vaccination. *Id*.

Secondly, Dr. Kinsbourne drew attention to the fact that Mario was a significantly smaller baby than was normal for his age, and urged the Court to take that fact seriously in considering whether Mario's brain weight was heavier than it would have been normally. Tr. at 173-4. Thirdly, regarding whether a conclusion of encephalopathy is justified on the basis of pathological findings alone, or must necessarily be corroborated by clinical history, Dr. Kinsbourne pointed out that Mario was sleeping unobserved for the hours leading up to his death, and would not necessarily have visibly manifested all potential symptoms of encephalopathy to his family, if the course of that encephalopathy was relatively precipitous. Tr. at 173.

Fourthly and lastly, Dr. Kinsbourne drew attention to the absence of explanation and/or literature filed from Respondent's experts to support the idea that cerebral edema "could terminate in five minutes when a child asphyxiates." Tr. at 174. Until that adequate support was rendered, Dr. Kinsbourne reiterated that "the asphyxial part of SIDS is too great to explain the cerebral edema in Mario Perez's case." *Id*. That is to say, the "asphyxial type of positional death" described by Respondent's experts occurs "too fast to explain cerebral edema of any degree found in Mario." Tr. at 175.

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[31] Isomorphic gliosis is "gliosis in which there is a regular and parallel arrangement of glial fibers." DORLAND'S, *supra*, at 778.

8. Michael Kohrman, MD

Lastly, Dr. Kohrman returned for additional testimony as well. Tr. at 175-76. In response to the additional testimony rendered by Dr. Shane and Dr. Kinsbourne, Dr. Kohrman made several comments in rebuttal.

The first idea Dr. Kohrman sought to challenge was that "mild cerebral edema can produce a significant compromise in blood flow to cause herniation in a two-month-old," which he found "unlikely" because an infant of that age "has two means in which to dissipate increased intracranial pressure and does so very effectively." Tr. at 176. The first method would be that, because "the sutures are not closed," "in a period of time, the sutures will split," which "would allow for some increase in head size." *Id*. The second would be that the still wide-open fontanelle would act as an acute relief valve, and "would distend, allowing room for the brain to swell slightly as well." Tr. at 176-77. For these reasons, said Dr. Kohrman, "mild edema is very, very unlikely to cause the kinds of pressure effects or changes in blood vessels that would lead to death that Dr. Shane has hypothesized." Tr. at 177.

Dr. Kohrman also addressed the question of whether Mario suffered an asphyxial death, which, he emphasized, "is not necessarily an all-or-none phenomena." Tr. at 177. He explained that emphasis more completely as follows:

> [T]here may be chronic hypoxia that goes on for a period of hours before the child has its agonal event and dies so that this edema that develops has a period of time in which it can develop based on just the hypoxia that goes on for those periods of hours.... There is reason to believe that this is a slow process of decreased flow, and then the hypoxia is enough to produce then just death at that point as well.

Id.

Focusing then on Dr. Kinsboune's statements, Dr. Kohrman countered:

> We see mild cerebral edema in most asphyxia. Any child who has a sudden asphyxia will have some cerebral edema. Most cerebral edema is not purely vasogenic. It's not purely cytotoxic. It usually has a mixture of both components. One may look more prominent than the other, but it's certainly not inconsistent with death from asphyxia.

Tr. at 178.

Beyond that, Dr. Kohrman wanted to state his opinion on how the Court should attach credibility to the various sources of factual testimony which detailed Mario's course leading up to death. He urged:

> [M]emory of events three years later in an affidavit is not a tape recorder. It isn't a video camera. We color our memories with what we feel happened, and our feelings often cloud what we actually saw. And reports to individuals at the time of the event

> are usually much more reliable, and we know this from witness testimony in many, many legal proceedings. It's very, very important that we pay credence to contemporaneous reports that are corroborated by two different police officers and are congruent amongst four witnesses.

Tr. at 177-78.

C. Post-Hearing Submissions

At the conclusion of the hearing, Petitioner was adjured to file the transcripts of the recorded police interviews of the Perez family present at Mario's death on 19 December 2003. These were filed on 19 March 2007. The transcript of Petitioner's interview with the police does not contain significant information relating to Mario's clinical history following vaccination, save for some portions from the interview with Petitioner herself:

> Q. Following those shots Wednesday evening at home, did Mario have any signs of any discomfort from the shots?
>
> A. He was just crying more th[a]n usual, I mean there was really way that, he wasn't doing anything else aside from crying a little bit more, he didn't have a fever, I had given him the [Tylenol;] he still had an appetite.
>
> Q. He still ate normal throughout the night?
>
> A. Right and he was still [taking] his naps.
>
> Q. And that night when you put him, Wednesday night into Thursday, the 17th, into the 18th, when you gave him his bottle for the night, did he sleep his normal routine?
>
> A. He did, he slept his regular six hours.
>
> Q. So he was asleep from when you put him in to approximately 5 or 6 in the morning?
>
> A. Exactly.
>
> Q. Okay, and that was clock work?
>
> A. [No audible answer.]

Transcript of Police Interview with Frances Perez at 5-6.

Also, Respondent filed for the sake of clarification and correction Appendix 36 from the textbook written by Dr. Gilbert-Barness, which was referenced at the hearing. Resp. Ex. F. The table, entitled "Brain Weight as a function of Age in Children and Adolescents," correlates a brain weight of 609 grams for a two-month-old male infant, at 58 cm of body height. *Id*. For a body height closer to Mario's, the chart correlates a normal brain weight of 523 grams and a body height of 54 cm for a one-month-old male infant. *Id*.

II. ULTIMATE FINDINGS OF FACT

A. THE PARTIES' ARGUMENTS

Regarding the Court's findings of historical facts in this case, Petitioner argues that her interview with the police, and the police-written report based upon that interview, are not reliable to discover Mario's clinical history, because the focus of that interview was on investigating criminal activity that might have caused Mario's death, rather than rendering a medical diagnosis. Petitioner's Posthearing Brief at 5. Petitioner likewise points out the severe mental and emotional anguish that troubled her at the time of the interview, and argues that "the sudden loss of a child" affected her recollection. *Id*. Petitioner points out that the assessment within the police report of Petitioner's account, that Mario followed a "normal routine," actually originated with Detective Kelley, and was not quite what Petitioner actually said during her questioning. *Id*. at 4. Based on the facts of this case, Petitioner argues, the police reports should not be afforded the same evidential gravity as would be granted for contemporaneous, written medical records taken by a licensed medical professional. *Id*. at 5.

To argue that the police were principally seeking after evidence of trauma (both violent and accidental), Petitioner points to portions of the police interview transcripts where the detectives ask questions about whether Mario fell and struck himself against his environment, or whether she did "anything wrong." *Id*. at 6, quoting Transcript of Police Interview with Frances Perez at 6-7. They also questioned George Jr., Petitioner's adult son, whether Petitioner "is a good mother and whether there [was] any fighting or arguing in the house." Petitioner's Posthearing Brief at 6.

Respondent, on the other hand, argues regarding the Court's findings of historical fact that Mario did not have clinical symptoms of encephalopathy, referencing the police reports based on interviews with Petitioner and her family members. Respondent's Posthearing Brief at 7. Respondent argues that "it is the Special Master's onus to weigh these contemporaneous factual records against [P]etitioner's affidavit, written some twenty-seven months after Mario's death as a contribution to a legal proceeding," as part of his determination of witness veracity, credibility and persuasive weight. *Id*. at 10-11, citing *Burns v. Secretary of HHS*, 3 F.3d 415, 417 (Fed. Cir. 1993) and *Richardson v. Secretary of HHS*, 23 Cl. Ct. 674, 678 (1991). Respondent reminds the Court that "the Special Master is not bound by such testimony that he deems inconsistent, inconclusive, inherently unreliable, built on an incorrect premise, illogical, or simply not credible." *Id*. at 11, citing *Estate of Arrowood v. Secretary of HHS*, 28 Fed. Cl. 453, 459 (1993).

Regarding the medical findings of fact, Petitioner stressed Dr. Shane's statement to the effect that "the most significant pathologic finding was the brain," and his finding of vasogenic edema, the type of edema present in cases of encephalopathy, rather than cytotoxic edema, common in cases of asphyxia. Petitioner's Posthearing Brief at 7, 26. Petitioner urges the point made by both Dr. Shane and Dr. Kinsbourne that the vasogenic cerebral edema present indicated that "an inflammatory process [was] taking place in the brain," which "takes a while," in contrast to the five to ten minutes typical of asphyxial death. *Id*. at 26. Petitioner's argument also draws a line in the shifting sands that represent medical understanding of SIDS, by citing to Dr. Kinsbourne's statements that "there's more than one cause of sudden unexpected infant death," a grouping which certainly includes

positional asphyxial suffocation, but which probably also includes infection and other causes as well, adding that evidence is clear that "children who lie on their back can still die of sudden unexpected infant death," such that "to say that something is or isn't a hallmark of SIDS is [beside] the point." *Id*. at 12.

Petitioner assails Dr. Gilbert-Barness' testimony that cerebral edema is a typical finding in SIDS deaths by reference to an article of medical literature held up as authoritative by Dr. Gilbert-Barness, which found cerebral edema to be present in less than fifteen percent of the SIDS cases studied. *Id*. Petitioner likewise attacked Dr. Kohrman's statement that cerebral edema resulting from hypoxic asphyxiation would continue for hours by pointing out that the edema that would appear in that instance would be cytotoxic edema, and not the vasogenic edema identified by both Dr. Shane and Dr. Gilbert-Barness. *Id*. at 19.

In essence, Petitioner argues that the Court should find that a fatal Table Encephalopathy occurred within the time period of 72 hours stated in the Table, or that, in the alternative, that the acellular pertussis component within the DTaP actually caused an encephalopathy which led to Mario's death. *Id*. at 22, 26. Regarding the latter of those two theories of recovery, Petitioner cites Dr. Kinsbourne's opinion that "the mechanism of injury is the neurotoxic effect of the pertussis," as the vasogenic cerebral edema is "indicative of a more enduring process, which is indeed compatible with a pertussis vaccine encephalopathy," where the toxin damages the brain by destroying barrier tissues in the brain or by toxically impairing the neurons themselves. *Id*., quoting Tr. at 84-85.

Respondent encourages the Court to make findings of medical fact based on the premise that there was "no objective evidence of encephalopathy in this case." Respondent's Posthearing Brief at 13. Respondent argues that there should be "considerable or severe cerebral edema and inflammation, coning of the brain or brain stem, and other pathological cerebral changes observable under gross examination" evident in cases of fatal encephalopathy. *Id*. at 7. Respondent also notes that "Clinical symptoms of encephalopathy evidence significantly decreased levels of consciousness, and manifest as abnormal sleeping, feeding, and interpersonal communication patterns. *Id*. In particular, Dr. Gilbert-Barness believes that she would expect to find "significant" cerebral edema, certainly more severe than she found in her examination of Mario's autopsy slides, if Mario had indeed suffered a fatal encephalopathy. *Id*. at 16. Respondent states that Mario's lungs were almost double their expected weight, a finding that Respondent's experts believe is more compatible with asphyxia as a probable cause of death. *Id*. at 13. Respondent also posits that chronic focal inflammation of the lungs and petechial hemorrhages of the thymus are also proof of SIDS by "positional asphyxiation." *Id*. at 18. Moreover, Respondent argues against Petitioner's theory of causation by encephalopathy was composed "more than three years after Mario's death [and] contradicts the diagnosis[32] made by the medical examiner performing the autopsy upon Mario's death." *Id*. at 15. Respondent combines these arguments to state that, "Even if the Special Master

[32] The Court notes that, to its understanding, by stating a conclusion of SIDS, the County Coroner made a diagnosis of partially exclusive non-diagnosis. Therefore, to state an affirmative diagnosis based upon the same tissue samples available to the Coroner, when the Coroner's conclusion is SIDS, does not actually "contradict" any contemporaneous medical record in this case.

accepts [Petitioner's] testimony *in toto*,...no objective evidence from the autopsy supports a finding that Mario's death was the result of an encephalopathy." *Id*. at 17.

Lastly, on legal grounds, Petitioner reminds the Court that the determination of how much persuasive weight to assign to oral testimony and/or contemporaneous medical records "is uniquely within the purview of the special master." Petitioner's Posthearing Brief at 20, quoting *Burns v. Secretary of HHS*, 3 F.3d 415, 417 (Fed. Cir. 1993). Petitioner states that compelling oral testimony may be more persuasive under the right facts and circumstances of the particular case. *Id*.

Respondent's legal argument is basically that Petitioner has not proffered a preponderance of proof that Mario suffered an encephalopathy.[33] Respondent focuses on the Table definition of encephalopathy in support of this argument. To that end, Respondent quotes the language of the Table and the Table's "Qualifications and Aids to Interpretation" (QAI) to say, "an acute encephalopathy is indicated by a significantly decreased level of consciousness lasting for at least 24 hours," and to add that "increased intracranial pressure may be a clinical feature of acute encephalopathy in any age group." Respondent's Posthearing Brief at 6, quoting 42 C.F.R. § 100.3 (b)(i)(A) and (C). Respondent also references the indicative signs of a 'decreased level of consciousness' listed in the Table's QAI as (1) Decreased or absent response to environment (responds, if at all, only to loud voice or painful stimuli); (2) Decreased or absent eye contact (does not fix gaze upon family members or other individuals); or (3) Inconsistent or absent responses to external stimuli (does not recognize familiar people or things)." *Id*. at 6-7, quoting 42 C.F.R. § 100.3 (b)(i)(D).

B. The Court's Conclusions

In sorting out the disputed issues presented above, the Court first notes certain matters that appear not to be in dispute. Both parties agree that Mario's autopsy slides show objective evidence of cerebral edema, although how severe that edema was is the subject of some dispute. Both apparently agree that, if Petitioner's fact witness testimony is assumed to be true, Mario's clinical history is consistent with encephalopathy. Despite their basic agreement on these matters, the Court must evaluate the evidence presented in this case to find which facts are true to a preponderance.

The primordial issue to be decided is what persuasive weight to assign to Petitioner's fact witness testimony, and to judge whether that testimony is vitiated by the police reports composed in the hours following Mario's death. The Court begins by stating the obvious: there are no contemporaneous records composed by medical personnel upon which to rely principally. In that absence, the Court must decide between Petitioner's memory of the events, composed some time after their occurrence, or the notations from an interview prepared in the hours following Mario's death, pursuant to criminal investigation.

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[33] Respondent correctly cites the test of causation followed by the Undersigned Special Master: "In order to prevail on entitlement, petitioner is obliged to prove not only that the vaccine *could have caused* the injury, but also that it *did cause* the injury in this particular case." Respondent's Posthearing Brief at 19, citing *Hines v. Secretary of HHS*, 940 F.2d 15518, 1527 (Fed. Cir. 1991) (emphasis in original).

Even if there were contemporaneous medical records which were inconsistent with the facts as Petitioner presented them, the persuasive weight of those medical records could still be overcome by "clear, cogent and consistent testimony" offered by the fact witnesses in explanation of the discrepancy. *Stevens v. Secretary of HHS*, No. 90-221V, 1990 WL 608693, at *3. (Cl. Ct. Spec. Mstr. Dec. 21, 1990). *A fortiori*, in the absence of contrary medical records, where only police interview reports are predicated as countervailing proof, Petitioner's testimony may be found prevailingly persuasive if it is "clear, cogent and consistent."

At the entitlement hearing, the Court was able to observe Petitioner (and her husband and young daughter) when she testified. The Court took note of her presentment, comportment and demeanor while she was present, especially when testifying, and interlocuted with her on more than one occasion therein. The Court was impressed with her credibility and assured of her veracity. Based upon these observations, the Court finds that her testimony is clear and cogent.

Moreover, the Court finds that her testimony was consistent. Even in her police statement, she noted that Mario was "crying a little bit more;" she did not say in any way that he was unsymptomatic. Also, despite her comment to the police about his sleep patterns in the overnight hours between 17 and 18 December 2003, her husband noted in his statement to the police that the fact that Mario had not awoken the night between 18 and 19 December 2003 (the hours leading up to and including his death) was noteworthily unusual. He did not likewise discuss the preceding night in the same terms. Her affidavit and testimony at the hearing were also internally consistent in relating the events between the vaccination and her discovery of Mario's death on the morning of 19 December 2003.

Petitioner was careful to clarify at the hearing what she meant by her statements to the police describing Mario's behavior as basically normal. She noted foremost that she did not understand the focus or the thrust of the police questioning, and from what she did know, it seemed that they were primarily concerned with investigating malicious causes for Mario's death. Her uncertainty is understandable, as perusal of the interview transcripts manifest many questions that are, at best, general, and, at worst, ambiguous or even ambiguously accusatorial (*e.g.*, the last question, "And you didn't do anything wrong[?]"). Even if the police interrogators were also pursuing potential causes of death from accidental or health-related sources, there is no doubt that their questioning also targeted the potential for malign intentions and actions by the family members (as well they should). There is nothing untoward about police investigating the death of a child by looking for potential criminal activity; the problem arises when a Court lays the summarized report of that interrogation as its foundation in the highly technical context of deciphering and reconstructing a clinical history for the departed child.

Furthermore, contrary to Dr. Kohrman's discussions of human psychology in the context of police questioning, an area of expertise not listed in his *curriculum vitae*, it is not manifestly true that every person responds identically to police questioning; indeed, the same person might not respond identically to individually separate experiences of police questioning. To assume that all parents everywhere will grope for potential explanations and open them up for discussion with an investigating police detective, just as if they were in a confessional booth or a psychiatrist's couch (or a medical examining room), is more than slightly facile. It is entirely plausible that Petitioner

might not have rendered as complete and detailed an account of Mario's (certainly somewhat medically subtle) clinical history in response to the generalized interrogation of the questioning police officer, as compared to what she might have expressed in response to skilled medical inquiry by Mario's treating pediatrician.

In her testimony given at the hearing, Petitioner also put her comments in their subjective testimonial context of when they were rendered: she thought then that the signs and symptoms of Mario's condition were within the normal range for a child who had just recently been immunized with four vaccinations by injection. In the Undersigned's experience in the Vaccine Program, it has quite often been the case that parents are aware that children will suffer some adverse symptoms following a vaccination, even if they are uncertain of how exactly those symptoms might surface or what those symptoms might be. To state it succinctly, parents are aware that their children will be distressed following a vaccination, even if they do not understand to differentiate between those symptoms that are typical and unremarkable, and those that are especially adverse and the cause for some alarm.

In sum, the Court accepts the fact witness testimony offered by Petitioner in this case as true. The question remains, however, as to which of the scientific theories offered by the parties is most consistent with these factual postulates. The questions presented in this case appear to be: (1) did Mario suffer an encephalopathy, and, if so, (2a) was it an encephalopathy that qualifies as a Table Encephalopathy, as defined in the Vaccine Injury Table, or, if it does not rise to that standard, (2b) was the encephalopathy (or whatever fatal condition) nonetheless actually caused by the DTaP vaccine?

All four experts were personally and professionally credible; that premise is beyond a cavil of doubt in the Court's mind. However, the Court must analyze the differences between the opinions offered to determine whether Petitioner has established a logical sequence of cause and effect that is biologically plausible to tie together the factual sequence and explain Petitioner's injury. *See Walther v. Secretary of HHS*, 485 F.3d 1146 (Fed. Cir. 2007); *Althen v. Secretary of HHS*, 418 F.3d 1274, 1278 (Fed. Cir. 2005).

There seems to be little dispute that the pertussis vaccine component *can* cause an acute encephalopathy. Tr. at 121, 151; *see also* Institute of Medicine, *Adverse Effects of Pertussis and Rubella Vaccines*, 118 (1991). The question in this case is really one of *did it cause* an acute encephalopathy which then led to Mario's death. As far as the evidence set forth has indicated, the Court would be justified in finding that an encephalopathy had occurred if such finding is supported by the pathological findings from Mario's autopsy examination and is corroborated by the clinical history filling the time between the vaccination on 17 December 2003 and Mario's death on 19 December 2003.

There was some dispute regarding whether encephalopathy was more properly diagnosed in Mario's case by pathological evidence or clinical history. From Petitioner's perspective, Dr. Shane found the pathological findings of the brain most significant, but saw the clinical history provided by Petitioner as supportive (Tr. at 32-34, 37), and Dr. Kinsbourne put predominant weight upon the pathological findings, viewing the cerebral edema noted as "definitive evidence" that Mario suffered

an encephalopathy (Dr. Kinsbourne's expert report). Dr. Kinsbourne added that the clinical history was less informative, because Mario was not observed during the overnight hours leading up to his death. Tr. at 173; *Cf. Kincaid v. Secretary of HHS*, Case No. 02-1766V, 2003 WL 23119834 *9 (Fed. Cl. Spec. Mstr. Nov. 26, 2003) (holding that even though decedent infant was unobserved in hours surrounding overnight death, "this does not prevent a holding in favor of petitioners because there is evidence that, had she been observed by appropriately knowledgeable observers, she would have been discovered to have a significantly decreased level of consciousness"). Respondent took the converse position, holding clinical history to be most important. Dr. Gilbert-Barness thought that primary reliance was properly placed on clinical symptomatology, with pathological findings serving for corroboration. Tr. at 103. Dr. Kohrman was even stronger, opining that "encephalopathy is a clinical diagnosis and not a pathologic diagnosis." Tr. at 145. The Court concludes that both are significant in analyzing this case, and defers the question of which is paramount, due to the unique factual circumstances of this case.

Having accepted Petitioner's fact witness testimony regarding Mario's clinical course following vaccination, it remains for the Court to adjudge the significance of that clinical history. The Court notes that, since 1977, medical science has contemplated "pertussis reaction syndrome" (first postulated by Stewart), to which is attributed the following indicia: "(1) persistent crying or screaming 4 to 48 hours after a pertussis immunization; (2) pallor and shock within 48 hours, usually 6 to 12 hours after immunization; (3) irritability and interrupted sleep; (4) refusal or vomiting feedings; (5) altered response to parents; (6) weakness or paralysis; (7) one or more convulsions, with or without fever." Institute of Medicine, *Adverse Effects of Pertussis and Rubella Vaccines*, 87 (1991). However, the IOM has noted that "[t]his specific clinical picture has not been confirmed by other investigators." *Id*.

The Vaccine Table itself defines the clinical indicia that can indicate the occurrence of an encephalopathy; if these indicia are met, the vaccine is legally presumed to have caused the vaccination. However, it is also possible for the Court to find that an encephalopathy occurred which does not meet the Table requirements, which would then leave Petitioner in the position of proving that such an off-Table encephalopathy was actually caused by the vaccine.

The Vaccine Table includes "Encephalopathy" as a listed Table Injury, corresponding to categories I, II, and III. 42 C.F.R. § 100.3(a). That means that, if the Court were to find that Mario actually suffered an encephalopathy (as defined in the Table's Qualifications and Aids to Interpretation) within 72 hours of receiving the vaccines listed in those categories, Petitioner would be entitled to a presumption that the vaccine caused that condition, requiring Respondent to prove that the condition was caused by a factor unrelated, lest Petitioner prevail on the issue of entitlement. §§11(c)(1)(C)(i) and 13(a)(1)(A)-(B). In finding facts to support or oppose a finding that Mario's condition fits the Table definition of encephalopathy, the Undersigned is directed by the Statute to consider "relevant medical and scientific evidence," to include "any diagnosis, conclusion, [or] medical judgment...contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, [or] condition," as well as "the results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." §13(b)(1)(A)-(B). Of course, these sources are not mechanistically determinative, and the Undersigned may

consider other portions of the record so as to view the record "as a whole" in arriving at particular factual findings. §13(b)(1)-(2).

The Vaccine Table's Qualifications and Aids to Interpretation (QAI) posits that an encephalopathy sufficient to be granted the statutory presumption in this case must manifest within 72 hours of vaccination as an "acute encephalopathy." 42 C.F.R. § 100.3(b)(2). In Mario's situation, "an acute encephalopathy is indicated by a significantly decreased level of consciousness lasting for at least 24 hours." 42 C.F.R. § 100.3(b)(2)(i)(A). The phrase "significantly decreased level of consciousness" is also a defined term in the QAI. Such state is "indicated by the presence of at least one of the following clinical signs lasting 24 hours or more: (1) Decreased or absent response to environment...(2) Decreased or absent eye contact...or (3) Inconsistent or absent responses to external stimuli. 42 C.F.R. § 100.3(b)(2)(i)(D).

In this case, Mario seems to have exhibited a "decreased level of consciousness," which lasted from 17 December 2003 until late into 18 December 2003, a period of over 24 hours, and which terminated, unfortunately, with Mario's death, which is clearly a state of decreased (i.e. nonexistent) level of consciousness. *See Jay v. Secretary of HHS*, 998 F.2d 979, 983 (Fed. Cir.1993); *Kincaid v. Secretary of HHS*, Case No. 02-1766V, 2003 WL 23119834 *9 (Fed. Cl. Spec. Mstr. Nov. 26, 2003). His responses to his environment were decreased, evidenced by his lack of normal response to bathing and toweling, as related by Petitioner. Whereas he would normally fuss while being bathed, instead his behavior was marked by torpor. He remained stupefied even when taken from the bath, when the shock of cooler air would normally have elicited a noticeable reaction. This certainly seems to correspond to a "decreased or absent response" of Mario to his "environment" at that time. It is likewise notable that Mario did not react to or interact with his mother at any point during that entire experience either, and his gaze did not meet his mother's when she bathed him. In contrast to his usually involved pattern of interaction, observation, vocalization, and emotional response (smiling), his behavior at that time does indicate a decrease or absence of eye contact or other response to external stimuli, as well as an "altered response" to his mother. Moreover, Petitioner's testimony (which the Court has accepted) reflects that Mario was consistently irritable throughout that period and that his sleeping pattern was significantly interrupted, as he did not sleep for more than two hours at a time, instead of his former pattern of four to five hours. Additionally, his feeding pattern was disrupted, such that he refused being fed after receiving a diminished amount of food. Petitioner's accepted testimony states that these events all took place between Mario's vaccination on 17 December 2003 and his death on 19 December 2003, which was a period of less than 72 hours. Also, Petitioner testified that these behaviors persisted from midday on 17 December 2003 until the late evening of 18 December 2003, when she put Mario to bed for the last time. Based upon these facts, a finding that the Table definition of Encephalopathy was met appears to be warranted.

Although Dr. Gilbert-Barness argued that a significantly lowered level of consciousness was "pretty much normal" or even "universal" response to vaccination (Tr. at 119), the Court found Dr. Kinsbourne more persuasive, as he made the point that lowered consciousness is not a normal response to vaccination as it is not viewed thus within the field of neurology (Tr. at 172). Dr. Kinsbourne's expertise in the clinical field of neurology gives his opinion greater weight on this point. Also, the Court is quick to mention, the Vaccine Program could hardly be expected to list on

the Vaccine Injury Table, as sufficient proof to award a presumption of causation, symptoms which were "pretty much normal" and/or "universal". Even Dr. Kohrman noted that if he accepted in full the clinical history testified to by Petitioner, he would be compelled to conclude that "this is clinical evidence of an encephalopathy as defined by the vaccine table." Tr. at 139.

Even if the Table criteria are not met in this case, they still manifest that, for the purposes of the Vaccine Program, the DTaP vaccine *can* cause encephalopathy.[34] And even if these clinical indicia are too ambivalent to surmount the requisite proof to qualify as a Table Injury, they are of corroborative utility towards a finding of causation, if the Court were to accept Petitioner's view of the pathological findings.

It is, of course, noteworthy that both pathologists in this case found cerebral edema present in Mario's brain, upon microscopic examination of the autopsy slides, especially considering that the County Coroner made no finding of edema in the brain or anywhere else, nor did he notice any inflammation of the brain. Furthermore, the County Coroner's focus in examining the brain appeared to seek after traumatic or critical events (*e.g.*, hemorrhage), not pathologic processes. That is not necessarily to say that he would not have noticed the evidence of a pathologic process where it was manifest, but the prose of the autopsy report reflects a focus on the punctuated and emergent. The Court is more inclined, then, to be persuaded by the expertise and experience of the expert pathologists testifying to the Court in this proceeding, that there was cerebral edema present in Mario's postmortem remains.

Once the Court accepts that cerebral edema was present in Mario's brain postmortem, it remains for the Court to determine the type and degree of edema present. Testifying for Petitioner, both Dr. Shane and Dr. Kinsbourne contrasted a finding of vasogenic cerebral edema, which is indicative of a limited number of phenomena (most of which, besides encephalopathy, are excluded in this case), from cytotoxic cerebral edema, which can more generally accompany death from other means not localized to the brain. Dr. Shane was emphatic that he specifically found vasogenic cerebral edema on the autopsy slides of Mario's brain. *See*, *e.g.*, Tr. at 165-66. The other pathology expert, Dr. Gilbert-Barness, noted her acceptance of the distinction between the two types, but did not specifically state which type she believed was more prevalent in Mario's brain. Tr. at 109. Dr. Kohrman, who is a neurologist, but not a pathologist, stated that "Vasogenic edema tends to be a little different than the cytotoxic edema, which tends to be more related to the time course," even though he went on to state that "vasogenic edema of a very mild nature" could present in deaths from asphyxial, toxic or metabolic causes. Tr. at 144. Although Dr. Kohrman challenges the dispositive quality Petitioner's experts attempted to attach to the presence of vasogenic edema, he does seem to concede that cytotoxic cerebral edema is "more related to the time course" of the agonal process. Therefore, in light of the expert testimony proffered in this case, the Court finds that vasogenic cerebral edema is significant as occurring prior to death and not a consequence of death. As a result, a finding of vasogenic cerebral edema militates against the conclusion that cerebral edema was simply an incident of death, and is persuasive evidence in favor of encephalopathy as a cause of

[34] Additionally, Respondent's expert witness, Dr. Kohrman conceded that pertussis "clearly" can cause encephalopathy, even if he disputed that occurrence in this case. Tr. at 152.

death. Dr. Kohrman said that, for him to be convinced that encephalopathy caused death, he would expect to see, *inter alia*, "the changes that Dr. Shane described in terms of what we would see in terms of vasogenic and cytotoxic edema." Tr. at 152.

Petitioner offered unchallenged evidence[35] that the edema found in Mario's brain was in fact vasogenic cerebral edema. Given Respondent's stipulated finding of cerebral edema, coupled with this unchallenged description of its vasogenic type, the Court finds that Mario's brain showed evidence of vasogenic cerebral edema. That finding, though, begs the next question: what degree of vasogenic cerebral edema was present?

Turning to the issue of heightened brain weight as an indicium of cerebral edema, and its degree (or as Respondent has emphasized, its "significance"), the Court gives credence to the evidentiary exhibits of medical literature that have been filed on this point. The first, more general table, culled from Dr. Gilbert-Barness' book and submitted by Petitioner for Dr. Shane, gives as the normal brain weight of a child of Mario's age as 516 grams. However, this table does not differentiate male from female children, a factor which could potentially skew the average lower, as female infants as a rule have smaller, and therefore lighter brains than their male peers. Greater probative weight seems to favor the second table, Resp. Ex. F, submitted after the hearing by Respondent for Dr. Gilbert-Barness, which differentiates by sex. If Mario's age of two months is used as the constant value to predict brain weight as the variable value, the normal brain weight stands at 609 grams. However, if Mario's length/height is used as the constant value, the corresponding normal brain weight is 523 grams.

Although Dr. Kohrman testified that Mario's brain growth was constant and standardized, not subject to fluctuation or deviation from the standard, there is reason to question that assertion. Dr. Kohrman believed that even if Mario's growth in height and weight were impeded, that his brain growth would have remained constant, and would have tracked the normal progression of his age group. Tr. at 143. Dr. Kohrman relied on the autopsy report stating that Mario was 55 centimeters in length, which Dr. Kohrman believed was "exactly" average for his age, indicating a head size at the normal size. As seen above, the normal length for a male two-month-old given in Resp. Ex. F, is 58 centimeters. Mario was actually significantly smaller than average, much closer to the size given for a male one-month old: 54 centimeters. For a boy of that size, 523 grams is the normal brain weight. Also instructive is the fact that the County Coroner recorded in the autopsy report his observation that Mario was normocephalic–not macrocephalic, as Dr. Kohrman's testimony suggests would be the case if Mario's cerebral and cranial growth outstripped his general growth rate for the rest of his body. Pet. Ex. C at 79; Tr. at 143. Inasmuch as 549 grams actual postmortem measured brain weight is significantly greater than the norm of 523 grams stated in the exhibit Respondent has proffered as authoritative, it appears that Mario's brain was significantly heavier than normal.

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[35] Dr. Gilbert-Barness expressed her respect for Dr. Shane's "eloquent description of types of edema in the brain." Tr. at 109. The only testimony approximating a challenge to Dr. Shane's finding of vasogenic, not cytotoxic edema was a generalized statement from Respondent's expert in neurology (and not pathology), Dr. Kohrman: "Most cerebral edema is not purely vasogenic. It's not purely cytotoxic. It usually has a mixture of both components." Tr. at 178.

As Dr. Shane elaborated, "a brain with significant edema is going to be significantly heavier." Tr. at 164. Dr. Kinsbourne agreed: "[T]hat this child's brain is at the end of the normal range or outside it -- is consistent with an encephalopathy." Tr. at 96. Dr. Kohrman stated that his opposition to a diagnosis of encephalopathy was premised on "the medical examiner's review and Dr. Barness's review and the fact that there is not significant brain weight changes," which led him to conclude "that there [was] not significant cerebral edema to cause an encephalopathy and death in this child." Tr. at 150. The Court reads all of these statements to conclude that brain weight serves as a correlative indicium of the degree of cerebral edema.

Dr. Gilbert-Barness referred to an article of medical literature filed by Petitioner (Hazim Kadhim *et al.*, "Incongruent Cerebral Growth in Sudden Infant Death Syndrome," *Journal of Child Neurology*, pp. 244-46 (March 2005)) which correlated heightened brain weights with SIDS deaths (Tr. at 108), to construe an above-normal brain weight as corroborative of SIDS, or just an ambiguous finding. In fact, Dr. Gilbert-Barness stated on direct examination that "some degree of edema is almost always present in sudden infant death." However, the abstract of that article clearly states that, according to the SIDS cases evaluated by their study, "Brain edema was not a major element, and there were no significant microscopic anamolies" among those brains studied. Kadhim at 244. As seems to be the case with much of the study of SIDS, "the precise cause of the increase in brain weight in sudden infant death syndrome remains elusive." *Id*. at 245. In contrast, the expert pathologists of both parties identified evidence of cerebral edema, which is not correlated with SIDS, according to the literature cited.

Given the fact that Mario's brain was notable for vasogenic cerebral edema, and given the fact that Mario's brain was significantly heavier than normal, and given the stipulated point that increased brain weight serves as a rough index of the extent of cerebral edema, it is logical to find that Mario suffered from cerebral edema that was itself significant. According to Dr. Kohrman, to justify a finding of encephalopathy by fatal cerebral edema, "[t]here have to be moderate to severe changes in the brain seen by the pathologist," such as "the changes that Dr. Shane described in terms of what we would see in terms of vasogenic and cytotoxic edema," and/or "some physical changes in the brain consistent with significant cerebral edema," and "significant increases in brain weight." Tr. at 152. Although the autopsy report does not indicate gross findings of brain swelling or coning, which Dr. Kohrman would have expected if the cerebral edema was "severe" (Tr. at 142), the presence of those facts found by the Court, and the expert testimony presented, persuades the Court to a preponderance that the vasogenic cerebral edema was significant, consistent with encephalopathy.

The only question remaining short of a finding that Mario suffered an encephalopathy and that it caused his death is whether some other theory of causation diminishes the likelihood or persuasive strength of Petitioner's theory of causation. Into this briar patch Respondent has thrown the tar baby of Sudden Infant Death Syndrome, or Sudden Infant Death, or Sudden Infant Death by Asphyxia, etc.[36]

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[36] Respondent also raised the specter of gastroesophageal reflux as a potential substantial cause of death. In his expert report, Dr. Kohrman thought that Mario's death was more likely caused by gastroesophageal reflux than by a vaccine-related encephalopathy; however, at the hearing, he clarified that he did not believe to a preponderance of the

Dr. Gilbert-Barness was highly confusing, or at least ambiguous, in her discussion of SIDS and its role in the instant case. She seemed to use the terms "Sudden Infant Death Syndrome (SIDS), Sudden Infant Death (SID), generalized asphyxia, and positional asphyxia as basically interchangeable, except that she also seemed to retain some differentiation between them at other points in her testimony. As the Court understands her testimony, she posited that many, if not most cases diagnosed as "SIDS" (or the label *au courant*, "SID") are actually caused by an asphyxiation process related to blockage of airways by surrounding objects in the infant's physical environment. Tr. at 103-104, 110-111. Distilled to its essence, Dr. Gilbert-Barness' theory of causation appears to collapse down into positional asphyxia as the cause of Mario's death (*i.e.*, that Mario suffocated as a result of being laid face-down in his bassinet), which the Court was only able to elicit from direct questioning of her position at the hearing. Tr. at 127-129. In response to the Court's query, Dr. Gilbert-Barness testified that she proffered this theory to a preponderance of the evidence. Tr. at 127-128. Dr. Kohrman agreed, opining that, to a preponderant probability, Mario died of asphyxiation. Tr. at 146.

In Respondent's brief, Petitioner's theory of causation was reproached on the basis that it appeared to conflict with "the diagnosis made by the medical examiner." Respondent's Posthearing Brief at 15. Aside from the point the Court has already made, that an affirmative diagnosis cannot be said to contradict a partially exclusive non-diagnosis, it appears that Respondent's proffered theory of causation–asphyxia–likewise falls prey to the same accusation Respondent has leveled against Petitioner. The County Coroner did not diagnose asphyxia, presumably because he did not notice evidence that such had occurred. In this case, SIDS became something akin to the proverbial Rorschach inkblot test: Respondent's experts concluded that the otherwise unexplained conclusion "SIDS" in the autopsy report really meant asphyxia, at least as they saw it. This was made manifest when the Court queried Dr. Kohrman as to why the County Coroner concluded with that generic diagnosis of exclusion, if Mario really died from the etiologically meaningful diagnosis of asphyxia: Dr. Kohrman answered, in essence, that the Coroner must not have been current with the "evolving state of our art right now." Tr. at 146. Even though Dr. Gilbert-Barness opined that a "large percentage [of SIDS cases] are actually asphyxial deaths" (Tr. at 126-27), that does not mean that all SIDS deaths are asphyxial, or that the two are equivalent for purposes of etiological explanation. As Dr. Kinsbourne aptly noted, asphyxiation is one explanatory constellation out of the (largely undiscovered) SIDS universe, which is why SIDS is of limited utility as an etiological explanation. Tr. at 86, 89-90. The diagnosis of SIDS–if indeed it truly fits that categorical term–is a diagnosis of exclusion. *See Corzine v. Secretary of HHS*, Case No. 01-0230V, 2004 WL 1047394 (Fed. Cl. Spec. Mstr. Apr. 23, 2004) (noting that "SIDS is not a disease, nor is it a cause of injury...[but] is a diagnosis of exclusion, a catchall definition used when the cause of death in a previously healthy infant cannot be identified because no cause is apparent...[such that] when the doctors, pathologists, and other experts are at a loss for possible causes of death, they rely on SIDS as a diagnosis"). In this case, it appears that the County Coroner was merely attempting to exclude traumatic or emergent

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evidence that it was the cause of Mario's death. Tr. at 149. Moreover, Dr. Gilbert-Barness flatly denied that gastroesophageal reflux was in any way causally related to Mario's death. Tr. at 130. Because of this uncertainty that this condition might even be a factor, and absent persuasive references from the medical records extant, the Court excludes gastroesophageal reflux from its consideration as patently unpersuasive in this case.

circumstances as potential causes, and once that question was satisfactorily answered in the negative, and Mario's death could be attributed to "natural causes" (*See* Pet. Ex. C at 73), the County Coroner concluded his inquiry there, finding that Mario died of unexplained natural causes.

To find that, firstly, SIDS is a diagnosis, and secondly, that it may be relied upon as more persuasive than Petitioner's theory of causation is an act of casuistry: it is to place faith certain in a diagnosis of uncertainty. It is also reversible error. *See Davis v. Secretary of HHS*, 54 Fed. Cl. 230 (2002) (reversing the special master's decision denying compensation that relied upon Respondent's expert Dr. Gilbert-Barness' opinion that evident cerebral edema was only mild, was consistent with SIDS deaths, and was inconsistent with a death caused by cerebral edema or encephalopathy; and remanding with instruction that "[w]hether SIDS is an alternative explanation for the evidence of cerebral edema and increased brain weight as Dr. Gilbert-Barness claims should be irrelevant to the special master's consideration of whether petitioner demonstrated ... an encephalopathy;" ordering the special master to "address whether the evidence of cerebral edema and increased brain weight, in combination with other evidence, is sufficient to demonstrate a prima facie case of a vaccine-related injury"). For this reason, the Court finds the explanation of SIDS (or SID) unpersuasive as a theory of causation. *Compare Kincaid v. Secretary of HHS*, Case No. 02-1766V, 2003 WL 23119834 (Fed. Cl. Spec. Mstr. Nov. 26, 2003) (finding, against the less credible SIDS theory, that a Table Encephalopathy had occurred within 72 hours of vaccination, due to the presence of, *inter alia*, moderate to severe brain edema, which, according to evidence offered by Respondent therein, is a typical finding in the "pathology of encephalopathy").

As the Gordian knot of Respondent's all-inclusive SIDS theory has been dissevered asunder, the Court is left with the critical task of weighing the persuasive probability of Respondent's more concrete postulate of positional asphyxia against Petitioner's theory of vaccine-related encephalopathy, discussed *supra*. Even though both of Respondent's experts stated their professional opinion (to a preponderant probability) that Mario died from positional asphyxia, Respondent did not officially proffer asphyxia as a "factor unrelated", pursuant to § 13(a)(1)(B) of the Vaccine Act. Nonetheless, the Court turns now to the factual issues bearing on the theory of asphyxial death exclusive of, or superseding, vaccine-related encephalopathy as a cause of Mario's death.

Just as Respondent has decried Petitioner's theory of encephalopathy because of what Respondents experts saw as a dearth of gross (that is, patent or plainly visible) evidence that such had occurred, just so Petitioner's experts derided asphyxia as an explanation for the same reason. *See*, for Respondent's criticism, Tr. at 109-10, 116; for Petitioner's, Tr. at 44-45. The Court understands this predicament as its own delicate task of delineation throughout this case, as the findings are more subtle than one might prefer in order to render a definitive diagnosis in either direction. The lack of obvious evidence in gross findings at autopsy is a sinking tide that lowers both Petitioner's and Respondent's ships, and is therefore of diminished utility in leading the Court to prefer one over the other. Consequently, the Court turns to more latent pathological evaluation and circumstantial evidence to distinguish between the two.

One point of circumstantial import is the time period within which the agonal process extended. Dr. Shane explained that asphyxiation on dry land is a relatively short process, and

altogether too brief for the brain to manifest the vasogenic cerebral edema found in this case (Tr. at 35-38), and Dr. Kinsbourne agreed that, from his perspective as a neurologist, the five minutes it would take an infant like Mario to suffocate is not long enough for vasogenic cerebral edema to develop (Tr. at 94). Dr. Kinsbourne later returned to this point, forcefully stating that the appearance of any such edema is inconsistent with suffocation for the same reason. Tr. at 175. Dr. Kohrman attempted to rebut this criticism by hypothesizing that Mario could have had his breathing partially obstructed or impeded, such that his breathing was decreased, but not entirely stifled: the theory being that Mario suffocated over a longer period, which would have enabled the edema to develop. Tr. at 177. The Court is reluctant to accept Dr. Kohrman's hypothesis, due to the lack of thoroughgoing explanation on how that could occur. That circumstance is not commonsensical to the Court's understanding, such that explanation is unnecessary. Especially in comparison to the sedulous repetition and unanimity of Petitioner's experts on their point, this question of timing is resolved against the occurrence of asphyxiation. The Court therefore finds that the circumstantial evidence surrounding the timing of Mario's death supports a finding of encephalopathy.

In his testimony at the Hearing, Dr. Shane was sure to point out that the typical pathological markers associated with asphyxiation were absent from the autopsy findings from Mario's examination. Tr. at 61-63. Dr. Shane insisted that the petechial hemorrhages were agonal, and that they commonly arise in connection the hypoxia that results from the agonal process. Tr. at 61. There seems to be some dispute between the parties as to what to make of the fluid found in Mario's lungs at the time of the autopsy. Dr. Shane pointed out that the autopsy report described the fluid as "congestion" and not pulmonary edema or (the Court would add) hemorrhage. Tr. at 62. Dr. Shane found the increase in lung weight to be minimal, consistent with congestion from the agonal process. Tr. at 159. Dr. Shane also pointed out that there was no petechial hemorrhage in the lungs themselves, as is normal in asphyxial death, or in the pericardium, the sclera, or the conjunctiva, indicia that an examining pathologist would be sure to look for and observe if present. *Id*.

In contrast, although Dr. Gilbert-Barness spoke generally in her expert report that "All organs were intensely congested," she added later that she found evidence of subpleural hemorrhage in the lungs. At the Hearing, she testified that she "certainly saw some fluid -- hemorrhage in the lung." Tr. at 114. However, the autopsy report, to which Respondent has urged the Court to closely adhere, made no mention of any hemorrhage anywhere in the lungs or thoracic cavity. Pet. Ex. C at 77. Instead, the autopsy report describes "a few cc's of serous fluid" in the pleural spaces, "a cc of straw colored fluid" in the pericardial cavity, and congestion in the lungs. *Id*. Neither fluid was described as blood or bloody, which could have indicated hemorrhaging. Additionally, the autopsy report did not report any fluid accumulation in the spleen. *Id*. at 76, 78. The Court finds that there was no hemorrhaging in these areas, and no pulmonary edema; the lungs, much like many of the other organs, were merely congested.

Based on this analysis, it seems unlikely that Mario's death was caused by asphyxia, although some hypoxia was likely to have occurred as a step in the agonal process. The Court therefore finds that Mario's death was not caused by asphyxia. *Cf. Frank v. Secretary of HHS*, 34 Fed. Cl. 29 (1995) (affirming special master's decision denying encephalopathy had occurred where autopsy reported "marked pulmonary congestion and edema, scant petechial hemorrhages over [the] thymus

gland and the epicardial[37] surface of [the] heart, and congestion of [the] spleen and the leptomeninges[38] of [the] brain," and where the autopsy identified cause of death as "viral respiratory illness," noting elsewhere that "inflammation was present throughout the respiratory tract"); *Hellenbrand-Sztaba v. Secretary of HHS*, Case No. 91-0572V, 1995 WL 650678, *4 (Fed. Cl. Spec. Mstr. Oct. 19, 1995) (finding more credible Respondent's proffered expert opinion explaining that "the presence of pulmonary edema is extremely common on autopsy," "usually is not indicative of the cause of death," and is often "simply a result of the process of dying, rather than the cause of death").

In sum, the Court finds that the Pertussis component of the DTaP vaccine can cause encephalopathy, that Mario did suffer an encephalopathy, and that such encephalopathic condition was a cause in fact and a substantial cause of Mario's death. Moreover, the Court finds that Mario's encephalopathy fit the Table definition of encephalopathy, and that, in the alternative, the DTaP vaccine did actually cause the encephalopathy, of which death was the sequela.

III. CONCLUSIONS OF LAW

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). The Petitioner has claimed to have suffered a "Table" injury, which §13(a)(1)(A) assigns the burden of proving such by a preponderance of the evidence. In the alternative, if Petitioner is not entitled to a presumption of causation afforded by the Vaccine Injury Table, the Petition may prevail if it could be demonstrated to a preponderant standard of evidence that the vaccination in question, more likely than not, actually caused the injury. *See* § 11(c)(1)(C)(ii)(I) & (II); *Grant v. Secretary of HHS*, 956 F.2d 1144 (Fed. Cir. 1992); *Strother v. Secretary 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.

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[37] The epicardia is "the lower portion of the esophagus, extending from the hiatus esophagi to the cardia." DORLAND'S, *supra*, at 625.

[38] The leptomeninges are "the pia mater and arachnoid considered together as one functional unit; the pia-arachnoid." DORLAND'S, *supra*, at 1016.

Furthermore, the Federal Circuit recently articulated an alternative three-part causation-in-fact analysis as follows:

> [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. Secretary of HHS, 418 F.3d 1274, 1278 (Fed. Cir. 2005).

Under this analysis, while 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. *See Knudsen v. Secretary of HHS*, 35 F.3d 543, 549 (1994).

Of importance in this case, it is part of Petitioner's 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. Secretary of HHS*, 451 F.3d 1352, 1355 (Fed. Cir. 2006)(emphasis added), *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. Secretary 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*.

As a matter of elucidation, the Undersigned takes note of the following two-part test, which has been viewed with approval by the Federal Circuit,[39] and which guides the Court's practical approach to analyzing 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. Secretary 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).

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[39] *See Pafford v. Secretary 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).

The Court found, *supra*, as a matter of fact, that encephalopathy can be caused by the Pertussis component of the DTaP vaccine which Mario received on 17 December 2003. The Court also found that encephalopathy did occur within 72 hours of administration of the vaccination. The Court based this finding on an analysis of clinical indicia and pathological markers. Reliance on the facts of Mario's reported clinical history in the finding of encephalopathy is warranted in this case, even though Mario died unobserved in the overnight hours between 18 and 19 December 2003. *See, e.g., Kincaid v. Secretary of HHS*, Case No. 02-1766V, 2003 WL 23119834 *12 (Fed. Cl. Spec. Mstr. Nov. 26, 2003) (holding that, "when someone dies in the middle of the night, and there is sufficient evidence to show that the person had an acute encephalopathy (e.g., credible medical interpretation of pathologic findings and the failure to move in order to breathe, showing an altered mental state), the mere fact that the contemporary records do not show clinical symptoms of a significantly decreased level of consciousness does not defeat petitioners' case"). It is also appropriate for the Court to find that Mario's death was a sequela of the encephalopathy. *Hess v. Secretary of HHS*, Case No. 90-0760V, 1991 WL 123577 (Cl. Ct. Spec. Mstr. Jun. 17, 1991).

Furthermore, the Court found that the preponderant weight of the pathological evidence supports Petitioner's allegation that the DTaP vaccine actually caused the encephalopathy and, ultimately, death. The Court made this finding vis-à-vis Respondent's counter-allegation of Sudden Infant Death Syndrome and/or Sudden Infant Death, as such an empty category was devoid of etiological explanation. The Court also found the alleged vaccine-related encephalopathy to be more persuasive than Respondent's counter-allegation of asphyxia, based upon the pathological evidence presented on the Record.

Taken together, the Court found that the DTaP vaccine did actually cause Mario's death via encephalopathy. As both the *can it* and *did it* prongs have been proved to the Court's satisfaction of a preponderance of the proof, the Court finds that Petitioner has proved that the DTaP vaccine caused a fatal encephalopathy in Mario.

These facts likewise satisfy the *Althen* test set forth above. There is no dispute that there exists a medical theory which establishes a causal connection between the DTaP vaccination and fatal encephalopathy; Petitioner's experts described the logical sequence by which the injury occurred; and the biologic process occurred within a time frame recognized by the Vaccine Injury Table as appropriate to presume causation.

The logical sequela of these findings of fact is that Petitioner has carried her burden of proof on the issue of vaccine-related causation. Inasmuch as the other elements of § 300aa–11 (b) and (c) have already been satisfied, the Court holds that Petitioner has met her burden on her case in chief, on the ultimate issue of entitlement to compensation.

III. CONCLUSION

Therefore, in light of the foregoing, the Court rules in favor of entitlement in this matter. Petitioner is awarded the statutorily-determined sum of $250,000.00.[40] § 15(a)(2). In the absence of the filing within 30 days of this date of a motion for review, filed pursuant to Vaccine Rule 23, the clerk shall forthwith enter judgment in accordance herewith.

IT IS SO ORDERED.

Richard B. Abell
Special Master

[40] If Petitioner believes that she is entitled to lifetime economic losses, she may move the Court for Reconsideration on the limited question of any additional, legally cognizable damages. *See Zatuchni v. Secretary of HHS*, __ F.3d __ , 2008 WL 360997 (Fed. Cir. 2008).