OFFICE OF SPECIAL MASTERS

No. 99-259V

(Filed: March 31, 2003)

TALA DAWN WOOLF, deceased, by her Mother and Personal Representative, KALENA WOOLF,	
Petitioner,	**TO BE PUBLISHED**
v.	
SECRETARY OF THE DEPARTMENT OF HEALTH AND HUMAN SERVICES,	
Respondent.	

Sylvia Chin-Kaplan, Esq., Boston, Massachusetts, for Petitioner.

Tami Parker, Esq., United States Department of Justice, Washington, D.C., for Respondent.

ENTITLEMENT DECISION

ABELL, Special Master:

I. Issue

The issue in this case is whether Tala Woolf's tragic death occurred as a result of her diphtheria-pertussis-tetanus (DPT) vaccination administered on 24 April 1997. Regrettably, this Court finds that Petitioner did not meet her burden by a preponderance of the evidence.

II. PROCEDURAL BACKGROUND

On 27 April 1999, Petitioner, Kalena Woolf, filed a claim under the National Childhood Vaccine Injury Compensation Act (Vaccine Act or Act)[1] for the alleged vaccine-related death of Tala

[1] The statutory provisions governing the Vaccine Act are found at 42 U.S.C. §§ 300aa-1 to 300aa-34 (1991 & Supp. 1997). Hereinafter, for ease of citation, all references will be to the relevant subsection of 42 U.S.C. §

Dawn Woolf (Tala). Petitioner claims that as a result of receiving a DPT vaccination on 24 April 1997, Tala suffered an "on-Table" encephalopathy resulting in the sequela of Tala's tragic death. *See* Petition (hereinafter "Pet.") at 1. Petitioner has satisfied the requirements for a *prima facie* case pursuant to 42 U.S.C. § 300aa-11(b) and (c) by showing that: (1) Petitioner Kalena Woolf is the legal representative of Tala Woolf, the person who allegedly sustained a vaccine related injury; (2) the vaccine at issue, DPT, is a vaccine set forth in the Vaccine Injury Table; (3) the DPT vaccination was administered to Tala in the United States; (4) no one has previously collected an award or settlement of a civil action for damages arising from the alleged vaccine 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 24 August 2000, Petitioner filed the medical expert report of John J. Shane, M.D.,[2] a certified pathologist. Petitioner's Exhibit 13.[3] In his report, Dr. Shane stated that Tala had a very substantial reaction to the 24 April 1997 DPT vaccination which "precipitated encephalopathy with cerebral edema and the resultant vomiting from the cerebral edema. As a result of her vomiting, [Tala] developed severe hyperkalemia which caused cardiac arrest." Pet. Ex. 13 at 2. Additionally. Dr. Shane stated that the *in situ* neuroblastoma, listed as the cause of death by the Medical Examiner who performed Tala's autopsy,[4] "did not have a role in [Tala's] death." *Id.*

On 26 October 2001, the Secretary of Health and Human Services ("HHS") filed a report in this matter contesting Petitioner's entitlement to compensation. Res. Rpt. HHS contended that neither the petition nor the medical records establish a *prima facie* case of presumptive causation, and Petitioner has not submitted a medical expert report sufficient to establish that Tala's [injuries and subsequent death] were in fact caused by her childhood immunizations." Res. Rpt. at 8.

Respondent filed an expert report from Virginia M. Anderson, M.D.,[5] a pediatric pathologist.

300aa.

[2] Dr. Shane is board certified in pathologic anatomy and clinical pathology (Clinical Pathology includes chemistry and toxicology). He is currently an adjunct professor of pathology at Allegheny University in Pennsylvania and he has served in academic appointments as a professor at Hahnemann Medical College, Thomas Jefferson University Hospital, and at Hershey Medical Center at Penn State University, in Pennsylvania. To his credit, he has authored 14 articles related to his field and has a number of professional society memberships.

[3] References to the Petitioner's Exhibits and Respondent's Exhibits, shall be hereinafter truncated to "Pet. Ex..", and "Res. Ex." respectively.

[4] Pet. Ex. 6 at 1. "An AUTOPSY was performed and the cause of death is : Neuroblastoma." *Id.*

[5] Dr. Anderson is one of a small group of board-certified pediatric pathologists in this country. She is currently an associate professor of pediatric pathology at the State University of New York in Brooklyn. She is also the pediatric pathologist-in-charge at the Institute of Pathology, Kings County Hospital Center, Brooklyn. She received her B.A. at Hunter College of the City University of New York in 1963 and her M.D. at the Medical University of South Carolina in 1968. Dr. Anderson is board certified in pediatrics, pathology, and pediatric pathology. She has numerous professional and administrative appointments. She has participated in writing some 90 plus publications, book chapters, reviews, and abstracts.

Res. Ex. A. Dr. Anderson stated that "[n]o evidence of encephalopathy is present in sections of the brain." Res. Ex. A at 1. Further, she went on to state that "[s]ignificant brain edema with resultant increased intracranial pressure sufficient to cause vomiting does not occur in very young infants who have an open anterior fontenelle. Neuroblastoma is the most probable cause of death in this infant." *Id*.

On 26 March 2002, the Court conducted an evidentiary hearing in this matter. The Court heard testimony from Petitioner's medical expert, John J. Shane, M.D., and Respondent's medical expert, Virginia M. Anderson, M.D. The hearing transcript was filed on 14 April 2002.[6]

On 15 May 2002, Petitioner filed a Supplemental Expert Medical Report authored by Dr. Shane. Pet. Ex. 15. In the supplemental report, Dr. Shane stated that Tala "had documented cerebral edema which was caused by the pertussis component of the DPT vaccine. The cerebral edema led to vomiting and the mild dehydration noted on autopsy. The prolonged vomiting leads to a metabolic alkalosis. To compensate, the child would increase the respiratory rate to lower the ph. A prolonged increased respiratory rate will lead to hypoxia and eventually, asphyxia and metabolic acidosis." Pet. Ex. 15 at 2. Dr. Shane concluded his supplemental report by stating that it was his "opinion that Tala Woolf's death occurred as a result of her DPT administration." *Id*.

Thereafter, the parties filed post-hearing briefs. On 17 September 2003, Petitioner filed her post-hearing brief. On 4 October 2003, Respondent filed a post-hearing brief. Petitioner filed no *sur-response.* Thus the record is complete and ripe for decision.

II. FACTS

Tala was born a healthy, normal, infant on 6 December 1996. Pet. Ex. 1 at 6. She had well-baby exams at two weeks, two months and four months of age which indicated that she was a well child. Pet. Ex. 1 at 2-4. At her two month well-baby exam on 13 February 1997, Tala received her two month vaccinations and was diagnosed as a "well child." Pet. Ex. 1 at 3. Approximately two weeks after her two month well-baby visit, on 24 February 1997, Tala was seen at her pediatrician's office and diagnosed with bilateral otitis and bronchiolitis. *Id*. Tala was not seen again by her pediatrician until 10 April 1997 for her four month well-baby exam. *Id.* at 2. During that visit Tala was diagnosed as a "well baby." *Id*. Her weight was 13 pounds, 8 ounces. *Id.*

Tala received her second administration of DPT on 24 April 1997. Pet. Ex. 4 at 2. Shortly after receiving the vaccination, Tala became fussy and irritable and developed a slight fever. Pet. Ex. 10 at 1. Tala's symptoms persisted through to the following day, 25 April 1997. Pet. Ex. 11 at 2. By the next day, 26 April 1997, according to Tala's mother and grandmother, Tala's fever had subsided but her irritability continued and she threw up at least three times. *Id*.; *see also* Pet. Ex. 10 at 1. That same day Tala's extremities had become red and slightly blue. Pet. Ex. 3 at 43.

On Sunday, 27 April 1997, Tala continued her vomiting . Pet. Ex. 10 at 2. Some time after noon, Tala's family called her pediatrician and he advised her to give her Tylenol and Motrin as well

[6] Citations to the 26 March 2002 hearing transcript will be referred to as "Tran."

as giving her Benadryl every four hours. Pet. Ex. 10 at 2. Tala's grandmother stated that by Sunday afternoon the redness had spread from her face and hands to her arms and legs and the rash was worsening. Pet. Ex. 11 at 2. By late afternoon, Tala was sweating profusely, she had no temperature, was only drinking water and was throwing up everything else. *Id*. Just prior to bringing Tala to the emergency room, the family noticed blueness in Tala's feet and around her mouth. *Id.* at 3.

On Sunday evening, 27 April 1997, at around 6:30 p.m., Tala was taken to the emergency room at Wadley Regional Medical Center. Pet. Ex. 3 at 1. The contemporaneous medical records state that Tala had exhibited a 24-hour history of irritability, vomiting and had a macular type rash on her cheeks and some redness and mild swelling of the extremities. Pet. Ex. 3 at 2. A physical examination revealed that the anterior fontanelle was "quite soft, and no evidence of any neurologic symptoms were present." *Id*. Tala "was otherwise alert, active, and appeared in no acute distress." *Id*. Vital signs revealed a "blood pressure of 75/43, pulse rate of 140-160 range, respirations in the 40's." *Id*. Tala's weight was measured at 5.86 kg (12lbs. and 15 ounces). *Id.* at 43. Tala was placed on Tylenol and Benadryl to combat her rash and noted fussiness. *Id* at 2. It was also reported that Tala did not have diarrhea and, that while she had been fussy and irritable, she had some periods of normal sleep and playfulness. *Id*. Tala was initially diagnosed with urticaria and possible reaction to pertussis. *Id.* at 9.

Upon admission to the hospital at approximately 9:20 p.m., the nurse's notes stated that Tala's skin was warm on the abdomen, her upper extremities were dark red in color and cool to the touch and her lower extremities were mottled and cool to the touch. *Id.* at 45. It was noted that Tala's cheeks had a red rash and her mucous membranes were moist and pink. *Id*.

At approximately 10:00 p.m., the doctor was advised that Tala continued to vomit and was not tolerating fluid. *Id*. The doctor prescribed new orders to abate the vomiting. *Id*. Tala's nurse twice attempted to insert a heparin lock[7] in her but was unsuccessful because Tala was too swollen. Pet. Ex. 3 at 45; Pet. Ex. 11 at 4. Soon thereafter, the nurse noted that Tala's upper and lower extremities were "edematous"[8] with delayed CFT [capillary filling time] in all extremities." Pet. Ex. 3 at 46.

At approximately 11:30 p.m., it was noted that Tala's fontanelle was soft and flat, *Id*.; her respiration was regular and un-labored. *Id*. She continued to vomit, the rash remained on her cheeks and extremities and Tala was intermittently irritable. *Id.*

Throughout the night Tala continued to vomit after any attempt to give her fluid. *Id*. However, her respiration was regular, even and unlabored. *Id.*

At approximately 7:00 a.m. on the morning of 28 April 1997, the rash on Tala's cheeks had

---

[7] A heparin lock or hep. lock is a cap attached to the end of an intravenous line that provides access for medical personnel to administer medications and also prevents bleeding out by the patient.

[8] "Pertaining to or affected by edema." DORLAND'S *supra* at 531. Edema: "The presence of abnormally large amounts of fluid in the intercellular tissue spaces of the body; usually applied to demonstrable accumulation of excessive fluid in the subcutaneous tissues."*Id*.

resolved, her skin color appeared quite good, there was less swelling and redness on her extremities and Tala was active and alert. *Id*. at 3. Tala had taken an ounce of formula which she sucked down eagerly with no vomiting. *Id*. A little later that morning Tala's family voiced concern that Tala's hands looked blue. *Id*. at 47. A nurse checked Tala's hands and found them cool to the touch. *Id*. Tala's pulse was taken and it was 160. *Id*. Some time during the morning, Tala was weighed and her weight was recorded at 13 pounds and 2 ounces (5.96 k.g.). Pet. Ex. 3 at 36.

At approximately 11:30 to 12:00, Tala appeared mottled and was described as lethargic. *Id.* The doctor ordered that Tala be given intravenous Ampicillin and Claforan and that she be administered a third of a Phenergan suppository for nausea and vomiting on an as needed basis. *Id.* at 3-4. After a lunch break, Tala's mother returned to Tala's hospital room and found Tala to be unresponsive. Pet. Ex. 10 at 3. The nurse's notes state that Tala's color was dusky, her lips were pale and her body mottled. Pet. Ex. 3 at 47. Her abdomen was cyanotic.[9] *Id*. At that time Tala's respiration was 44, her pulse was 160, her temperature 102.6 and her blood pressure was 96/80. *Id*. at 48.

At around 2:00 p.m., multiple attempts to start an IV were unsuccessful. *Id*. at 3. Tala was then taken to the medical intensive care unit for in depth evaluation and monitoring. *Id*. A lumbar puncture revealed clear fluid and an elevated protein level of 133[10] mg/dL in the cerebral spinal fluid. Pet. Ex. 3 at 20. Tala was intubated due to the mottling and lack of color. *Id* at 3. Attempts to obtain a pulse reading were unsuccessful. *Id.* Additional attempts to start the IV were unsuccessful. *Id*. An interosseous[11] line was subsequently inserted and Tala was started on IV fluids. *Id*. At 3:00 p.m. Tala went into asystole[12] and resuscitative efforts were begun. *Id.* After 30 minutes of resuscitation, Tala was unresponsive and was declared dead. *Id.*

On 29 April 1997 starting at approximately 9:30 a.m., eighteen hours after Tala died, an autopsy was performed. Pet. Ex. 6 at 3. The autopsy revealed a soft and slightly sunken fontanelle and a slightly sunken appearance to the eyes . Pet. Ex. 6 at 3. Toxicology results revealed vitreous electrolyte levels as follows: Na (sodium) 129 mEq/L; K (potassium) 10.5 mEq/L; Cl (chloride) 115 mEq/L. *Id*. at 5. Additionally, Tala had mildly elevated levels of beta tryptase consistent with mass cell activation. *Id.* A microscopic examination of the brain revealed a mild perivascular pallor compatible with a cerebral edema. *Id.* at 6. Tala weighed 13 pounds at the time of the autopsy and her brain weight was 737 g. *Id*. at 3.

A body cavity internal examination uncovered a 6.5 x 6 x 5.3 cm fleshy pink-tan mass located anterior to the aorta, just below the kidneys situated predominantly on the left side, but also

---

[9] Cyanotic is defined as "pertaining to or characterized by cyanosis." DORLAND'S *supra* at 415. Cyanosis is defined as "a bluish discoloration, applied especially to such discoloration of skin and mucous membranes due to excessive concentration of reduced hemoglobin in the blood. *Id*.

[10] Normal levels of protein in the central spinal fluid range from 15 to 45 mg/dL. Pet. Ex. 3 at 20.

[11] Between bones. DORLAND'S *supra* at 846.

[12] Cardiac standstill or arrest - absence of a heartbeat. DORLAND'S *supra* at 160.

extending slightly right to the midline. *Id*. at 4. Adherent to the mass there were closely matted apparent lymph nodes also present. *Id.* The mass appeared at least focally encapsulated and did not invade the aorta or other pelvic structures grossly. *Id.* The mass was diagnosed as a Neuroblastoma[13] *Id* at 5. The medical examiner conducting the autopsy found that the cause of death was the Neuroblastoma. *Id.* at 1.

III. DISCUSSION AND ANALYSIS

Petitioner can prove she is entitled to compensation under the Program in one of two ways. She can prove entitlement through a statutorily prescribed presumption of causation or, by proving causation-in-fact. First, Petitioner may prove that Tala suffered an injury or condition listed in the Vaccine Injury Table within the statutorily prescribed time period. § 11(c)(1)(C)(i). If Petitioner establishes that Tala suffered such injury by a preponderance of the evidence, Petitioner is entitled to a presumption of causation. § 13(a)(1)(A). If Tala qualifies under this presumption, she will be said to have suffered a "Table injury." The burden would then shift to the Respondent to prove that the injury or condition "is due to factors unrelated to the administration of the vaccine described in the petition." § 13(a)(1)(B).

If Petitioner fails to satisfy the requirements under the Act for demonstrating a Table injury, Petitioner may prove by a preponderance of the evidence that the vaccination in question, more likely than not, caused the alleged injury. §§ 11(c)(1)(C)(ii)(I) and (II). This causation-in-fact standard, according to the Federal Circuit, requires proof of a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." *Grant v. Secretary of HHS*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). Once again, if Petitioner is successful in that showing, the burden shifts to Respondent to prove that the injury or condition "is due to factors unrelated to the administration of the vaccine described in the petition." § 13(a)(1)(B).

In the present case, Petitioner does not allege that Tala suffered a Table injury.[14] Petitioner alleges that Tala's death was sequela to an encephalopathy that "was in fact caused by the DPT vaccination." Pet. Closing Argument at 1. Thus, Petitioner's claim is one of causation-in-fact.

A. Causation-In-Fact

In order to demonstrate entitlement to compensation in a causation-in-fact claim, a petitioner must affirmatively demonstrate by a preponderance of the evidence that the vaccination in question *more likely than not* caused the injury alleged. *See* 11(c)(1)(C)(ii)(I) and (II); *Grant v. Secretary of*

---

[13] Sarcoma of nervous system origin, composed chiefly of neuroblasts and affecting mostly infants and children up to 10 years of age. DORLAND'S *supra* at 1127. Sarcoma is a tumor made up of a substance like the embryonic connective tissue; tissue composed of closely packed cells embedded in a fibrillar or homogenous substance. Sarcomas are often highly malignant. *Id.* at 1485. Neuroblasts are any embryonic cell which develops into a nerve cell or neuron; an immature nerve cell. *Id*. at 1127.

[14] In fact, Petitioner's expert, John J. Shane, M.D., testified that Tala did not experience the requisite 24 hour decreased level of consciousness within the 72 hour time frame required to meet the definition of an acute encephalopathy as found in the Vaccine Injury Table's aids to interpretation at 42 C.F.R. § 100.3(b)(2)(A). Tran. at 46-47.

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, which summarized the legal criteria required to prove causation-in-fact under the Vaccine Act, requires that every petitioner:

> 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.

This Court has organized the legal criteria in *Grant* by means of a two-part test. *First*, a petitioner must provide a reputable medical theory causally connecting the vaccination and the injury. In fine, can DPT cause the type of injury alleged? *Second*, a petitioner must also prove that the vaccine actually caused the alleged symptoms in her particular case.

Under the first prong, a petitioner must demonstrate the biologic plausibility of their theory. This may be accomplished in a number of ways. First, a petitioner must proffer a scientific pathogenesis underlying the alleged causal relationship. Reliability and plausibility are found by providing evidence that a sufficient minority of physicians have accepted the theory. In addition, epidemiological studies and an expert's experience, while not dispositive,[15] lend significant credence to the claim of plausibility. Articles published in respected medical journals, which have been subjected to peer review, are also persuasive.

The second prong of the causation-in-fact test is difficult but not impossible. Petitioner must show, by a preponderance of the evidence--as this special master is wont to say, a test based on 50% and a feather--that the vaccine caused the symptoms that manifested in this case. A petitioner does not meet this affirmative obligation by merely showing a temporal association between the vaccination and the injury. Rather, a petitioner must explain *how* and *why* the injury occurred. *Strother*, 21 Cl. Ct. at 370; *see also Hasler v. United States*, 718 F.2d 202, 205 (6th Cir. 1993), *cert. denied*, 469 U.S. 817 (1984) (inoculation is not the cause of every event that occurs within a ten day period following it).

B. Applicability of the Two Part test in Tala Woolf's Case

In Tala's case, the Court follows the two pronged causation in fact analysis tailored as: (1) Can DPT, specifically the pertussis element, cause an encephalopathy?; and, (2) Did Tala's DPT vaccination result in an encephalopathy that led to Tala's death?

1. Can the pertussis element in DPT cause an encephalopathy

[15] This first prong of the Court's test meets easily with cases where epidemiological or case study reports are already available. Beginning with this prong is practical when there is epidemiological evidence, for it avoids the tautalogical reasoning that would result when one attempts to answer *Can It?* without having reports and studies that previously would have answered *Did It?*

The Institute of Medicine (IOM) has written extensively about this relationship between pertussis and encephalopathy. Respondent usually relies on the IOM's publications to confirm respondent's own positions, generally that there is a lack of causation between a particular vaccine and an alleged injury. In discussing pertussis encephalopathy, the IOM cites the National Childhood Encephalopathy Study (NCES), a large British case-control study begun in 1976, which concluded that receipt of DPT vaccine results in an increased risk of, *inter alia*, encephalopathy. INSTITUTE OF MEDICINE, ADVERSE EFFECTS OF PERTUSSIS AND RUBELLA VACCINES, 101 (1991). The IOM concluded, "The evidence is consistent with a causal relation between DPT vaccine and acute encephalopathy. . . ." *Id.* at 118. Thus, the question of whether pertussis can cause encephalopathy is answered in the affirmative.

2. Under the facts of the record as a whole, did the pertussis element in the DPT cause Tala's death?

Petitioner's medical expert, John J. Shane, M.D., opines that Tala's death was caused by an encephalopathy precipitated by the DPT administration on 24 April 1997. Pet. Ex. 13 at 2. Dr. Shane posits the following sequence of events, allegedly caused by the 24 April 1997 DPT administration: The pertussis component of the DPT vaccination caused a cerebral edema, leading to prolonged vomiting and mild dehydration. Pet. Ex. 16 at 3. The prolonged vomiting led to metabolic alkalosis which caused Tala to increase her respiratory rate to lower the pH. *Id.* The prolonged respiratory rate led to hypoxia, asphyxia and metabolic acidosis. *Id.* The metabolic acidosis led to increased levels of potassium in Tala's blood, resulting in hyperkalemia causing cardiac arrest and Tala's resultant death. *Id*.

a. Cerebral Edema

Tala's autopsy results state that "sections of the hippocamus and parietal lobe reveal patchy, mild perivascular pallor compatible with a mild cerebral edema." Pet. Ex. 6 at 6. In his testimony, Dr. Shane stated that he examined the autopsy slides and agreed that the child had a "mild to moderate cerebral edema." Tran. at 7. He opined that the elevated level of protein in Tala's cerebral spinal fluid was indicative of an irritative process in the central nervous system causing the cerebral edema. Tran. at 33. Additionally, Dr. Shane found that Tala's brain weight of 737 g. was "arguably top limit of normal, or slightly above normal [a]nd that would certainly coincide with the finding of a cerebral edema." Tran. 11.

While Respondent's medical expert, Virginia M. Anderson, M.D., acknowledged that literature has associated the onset of cerebral edema after the administration of pertussis, Tran. at 125, she found that there was no mild cerebral edema in this case. Tran. at 107-08. She stated that the inexperience of the Medical Examiner in performing autopsies on children could have caused her to wrongly conclude that the microscopic impression was a cerebral edema. Tran. at 70. In addition, she stated that "[i]f you are going to have cerebral edema sufficient enough to cause symptoms and death, you're going to see [it] grossly." Tran. at 69. Dr. Anderson also stated that a brain weight of 737 g. "really may be the top limit of normal" but is not anything that needs to be focused on in this case. Tran. at 71. Finally, Dr. Anderson stated that there are many conditions that can cause elevated protein levels in the cerebral spinal fluid. Tran. at 67.

Petitioners did not submit any medical literature objectively validating the claim that elevated levels of protein in the cerebral spinal fluid indicates a cerebral edema. The Court also found a paucity of authority for this claim in its own research.[16] However, Dr. Anderson never refuted Dr. Shane's statement that elevated levels of protein in the spinal fluid "are reflective [of an] irritative process in the CSF." Trans. at 14. Dr. Anderson did theorize that the elevated protein levels could be from a period of hypertension, Trans. at 68-69, and the blood pressure reading of 96 over 80 found in the medical records, Pet. Ex. 3 at 48, was "hypertension . . . for sure." Trans. at 93-94. Dr. Shane stated that a systolic reading of "92 " [sic] is not hypertension but the dystolic "reading of 80 is high." Trans. at 128.

In this matter, the coroner's findings are not dispositive. Autopsy results or a coroner's report "shall not be binding on the special master . . . ." *Lampe v. Secretary of Health and Human Services,* 42 Fed.Cl. 632, 640 (1998). Thus, the Court must weigh contrasting experts' opinions as to whether a cerebral edema did indeed exist. Because Dr. Anderson is a pediatric pathologist the Court gives added deference to her testimony in this matter. Although Dr. Shane is eminently qualified, his expertise is not with children. Therefore, the Court finds that Dr. Anderson was more persuasive here. Also, Petitioner's failure to file any medical literature bolstering her claim regarding this issue leaves the Court little recourse but to rely more heavily upon the expert testimony.

b. Prolonged Vomiting

Dr. Shane opined that cerebral swelling, resulting from the cerebral edema, caused Tala's vomiting along with the irritation of the central nervous system as indicated by the elevated protein levels. Tran. at 27. Dr. Shane stated that a "very, very small amount of cerebral swelling causes vomiting." *Id.* He also stated that "swelling certainly was present" in this case. *Id.*

Dr. Anderson stated that vomiting is a nonspecific symptom in infants. Tran. at 74, 123. She did agree with Dr. Shane that increased intracranial pressure can lead to vomiting. Tran. at 123. However, Dr. Anderson testified that any increase in intracranial pressure in a child Tala's age would not be enough to cause vomiting. Tran. at 124. "Significant brain edema with resultant increased intracranial pressure significant to cause vomiting does not occur in very young infants who have an open anterior fontanelle." Resp. Ex. A. (Dr. Anderson's expert report). The open fontanelle allows the baby's head to "expand" and "[t]hat's why you don't get herniation" in infants.[17] Tran.

---

[16] In its independent research, the Court found a website publication that is somewhat supportive of Petitioner's claim: "Elevated white blood cells and protein in the CSF may indicate . . . encephalitis." *at* http://atoz.iqhealth.com/HealthAnswers/encyclopedia/HTMLfiles/3084.html. Encephalitis is an "inflamation of the brain," DORLAND'S *supra* at 548. Telling, however, is that Tala's CSF white blood cell count was not elevated. Pet. Ex. 3 at 20.

[17] "The skull of the newborn and older infant is not a rigid box but rather consists of several membranous bones, with fontenelles and unfused bony structures providing outlets for increases in intracranial pressure . . . ." PEDIATRIC NEUROLOGY: PRINCIPLES AND PRACTICE 951 (Kenneth F. Swaiman ed., 2d ed. 1994); Increased intracranial pressure can be partially vented in infants and newborns by expanding the volume of the skull. GERALD M. FENICHEL, CLINICAL PEDIATRIC NEUROLOGY 93 (3d ed.1997). Vomiting is not listed as a feature of increased intracranial pressure in infants. However, it is listed as a feature of such in children. *Id.* at 92; *But see* an earlier text, STATIC ENCEPHALOPATHIES OF INFANCY AND CHILDHOOD 289 (Miller & Ramers eds., 1992), which lists vomiting as a symptom of increased intracranial pressure in both an infant and child. The Court notes, however, that a full

at 123-24.

Petitioner's claim falls short of the preponderance threshold here as well. In addition to Dr. Anderson's refutation, the contemporaneous medical records rebut Dr. Shane's pronouncement that intracranial swelling was certainly present in this case. A bulging or swelling fontanelle may be a sign of intracranial pressure in an infant.[18] Here, the medical records conclusively indicate that Tala's fontanelles were normal and flat. "[T]he hospital . . .check[ed] Tala's fontanelles several times, with no indication of bulging to suggest intracranial pressure." Res. Clos. Arg. at 10.[19] Additionally, neither the medical records nor the fact witness affidavits state that Tala was projectile vomiting, which is telling. Projectile vomiting is often associated with increased intracranial pressure.[20] Thus, Petitioner has not met its preponderance burden in her claim that intracranial swelling was the cause of Tala's chronic vomiting.

c. Mild Dehydration

Dr. Shane opined that the chronic vomiting led to mild dehydration. Pet. Ex. at 2. Firstly, Dr. Shane referred to the autopsy notation that Tala's "anterior fontanelle is soft and slightly sunken" as indicative of dehydration. Tran. at 15; Pet. Ex. 6 at 3. Additionally, Dr. Shane stated Tala's sunken eye appearance at autopsy could be reflective of dehydration, however, certain other conditions or storage could bring about the same effect. *Id.*; Pet. Ex. 6 at 3. Further, Tala's weight loss was another indication of mild dehydration . On 10 April 1997, Tala weighed 13 pounds, 8 ounces during a well-baby visit to her pediatrician. Pet. Ex. 1 at 2. Upon her admission to Wadley Regional Medical Center, Tala's weight was recorded at 12 pounds, 15 ounces (5.86 kg.). Pet. Ex. 3 at 36. That indicates a decrease in weight of approximately 4.167%.[21] Clinical symptoms of dehydration may become clinically apparent at a 3-4% decrease in overall body weight.[22] Res. Ex.

---

fontanelle is also listed as a symptom. *Id.*

[18] Features of increased intracranial pressure in infants includes bulging fontanelles. GERALD M. FENICHEL, CLINICAL PEDIATRIC NEUROLOGY 92 (3d ed.1997); "[A] fontanelle that bulges above the level of the bone edges and is sufficiently tense to cause difficulty in determining where bone ends and fontanelle begins is abnormal and indicates increased intracranial pressure. A full fontanelle, which is clearly distinguishable from the surrounding bone edges, may indicate increased intracranial pressure" but can also indicate other things such as "crying." *Id*. at 93.

[19] "The anterior fontanel was quite soft, and no evidence of neurologic symptoms was present." Pet. Ex. 3 at 2. (Wadley Regional Medical Center Discharge Summary); "[F]ontanel soft and flat." *Id*. at 45 (Nurse's notes at 9:20 p.m. on 4/27/97); and, "Fontanels are soft [and] flat." *Id*. at 46 (Nurse's notes at 11:30 p.m. on 4/27/97).

[20] "If vomiting is 'projectile'--*i.e.,* with tremendous force--it may be (but is not always) indicative of intracranial pressure, which in turn is a potential sign of encephalopathy." *Grimes v. Secretary of Health and Human Services*, 1991 WL 274234 (Cl. Ct. 1991).

[21] Calculated as follows: (13.5-12.9375)/13.5 = 0.04167 or 4.167%. 13.5 is equal to 13 lbs., 8 ounces and 12.9375 is equal to 12 lbs, 15 ounces.

[22] Angela MacKenzie et al., *Clinical Signs of Dehydration in Children*, *in* THE LANCET 605, 607 (Sep. 9, 1989).

G at 3. Finally, Tala had a delayed capillary filling time in all of her extremities upon admission, Pet. Ex. 3 at 46, and a 5-7 second delay in capillary filling time about two hours before her death.[23] *Id*. at 48.

Dr. Anderson stated that nowhere in Tala's clinical course in the hospital was there an indication that hospital personnel were impressed enough to do something to abate possible dehydration. Tran. at 117. Additionally, the medical records state that Tala's mucus membranes were moist and pink upon admission and on the morning of her death. Pet. Ex. 3 at 8, 10, 44. Dr. Anderson stated that the numerous times that hospital personnel checked Tala's mucus membranes indicates they were aware of the possibility of dehydration and that the mucus membranes would be an early sign. Tran. at 116. Additionally, it appears that Tala had gained weight while in the hospital. *Cf.* Pet. Ex. 3 at 43 (weight 12 pounds, 15 ounces upon admission), with Pet. Ex. 3 at 36 (weight 13 pounds, 2 ounces).

Another factor that impacts the question on whether Tala was dehydrated is the Wadley Regional Medical Center - Emergency Dept. Record which indicates that Tala weighed 13 pounds,[24] 4 ounces at approximately 7:33 p.m. on the night of 27 April 1997. Pet. Ex. 3 at 28. This weight measurement was not cited by either party and would indicate only a 1.85%[25] decrease in weight from her 10 April 1997 well-baby doctor visit. It may add credence to Respondent's claim that "we know nothing about how the weight was taken, . . . whether Tala was dressed, . . . or about any other factor which may cause variations in weight . . . ." Res. Closing Argument at 11.

Common sense may indicate that chronic vomiting could lead to mild dehydration. However, Petitioner's claim that such is supported by Tala's weight loss is suspect. The medical records indicate that Tala gained weight while in the hospital. Pet. Ex. 3 at 36. Additionally, Petitioner's claim that Tala weighed 12 pounds, 15 ounces upon admission to the hospital is in doubt due to another contemporaneous record that puts Tala's weight upon admission at 13 pounds, 4 ounces. Pet. Ex. 3 at 28. Further, each time that Tala's mucous membranes were checked, they were noted as "moist and pink." Pet. Ex. 3 at 8, 10, 44. Thus, Petitioner has not met her burden by a preponderance of the evidence that Tala was dehydrated.

d. Metabolic Alkalosis[26] led to an Increased Respiratory Rate leading to Metabolic

---

[23] Through the Court's own research it was able to learn that capillary fill time should be less than two seconds. *See* http://felidaeworld.com/homeexam.html; http://gucfm.georgetown.edu/welchjj/netscut/fen/dehydration_intro.html. Petitioner did not submit any medical literature explaining the importance of delayed capillary refill time.

[24] WT 13#4o. Pet. Ex. 3 at 26. The # sign is a medical abbreviation for pound. Neil M. Davis, *Medical Abbreviations: 8600 Conveniences at the Expense of Communications and Safety*, 189 (6th ed. 1993).

[25] Calculated as follows: (13.5-13.25)/13.5 = 0.0185 or 1.85%. 13.5 is equal to 13 lbs., 8 ounces and 13.25 is equal to 13 lbs, 4 ounces.

[26] A disturbance in which the acid-base status of the body shifts toward an alkaline side because of retention of base or loss of noncarbonic, or fixed (nonvolatile), acids. DORLAND'S *supra* at 48.

Acidosis[27]

Chronic vomiting can cause metabolic alkalosis. *See generally* Pet. Ex. 16B.[28] Dr. Shane states that Tala's chronic vomiting resulted in metabolic alkalosis which caused Tala to increase her respiratory rate in order to lower the blood's pH. Pet. Ex. 16A at 2. This increase in respiratory rate is not supported by the contemporaneous medical records. Upon admission to Wadley Regional Medical Center, Tala's respiratory rate was 63. Pet. Ext 3 at 43. In subsequent measurements from the night of 27 April 1997 to the afternoon of 28 April 1997, Tala's respiratory rate was measured at 42, 40, 48, 40, Pet. Ex. 3 at 36, and 44. *Id.* at 48. Additionally, notations in the nurses records indicate that Tala's respiration was "regular [and] unlabored," Pet. Ex. 3 at 46, "regular [and] even," *Id*., and "unlabored." *Id*. at 47.[29]

The mechanism of an increased respiratory rate by which Dr. Shane posits as the cause of the metabolic acidosis is not objectively supported. Petitioner has not met her preponderance burden here.

e. Hyperkalemia[30]

Dr. Shane stated that the alleged metabolic acidosis led to hyperkalemia, Tran. at 35, as evidenced by the 10.5 mEq/L vitreous potassium level found in the autopsy's toxicological screening. Pet. Ex. 6 at 5; Pet. Ex. 16 at 1. Dr. Shane went on to state that a vitreous potassium level of 10.5 mEq/L "is inconsistent with life," Trans. at 28, 35, and caused Tala to suffer cardiac arrest and die. Pet. Ex. 13 at 2.

Metabolic acidosis can indeed result in hyperkalemia: "An elevated serum potassium may occur with transcellular redistribution of potassium, which is seen typically in metabolic acidosis and shortly before death or in severely ill patients"[31] Pet. Ex. 16A. However, raised levels of vitreous potassium are a natural phenomenon postmortem.[32] Pet. Ex. 16C at 5. "[P]otassium . . . [has] been

---

[27] A disturbance in which the acid-base status of the body shifts toward an acid side because of loss of base or retention of noncarbonic, or fixed (nonvolatile), acids. DORLAND'S *supra* at 16.

[28] Thomas D. Dubose, Jr., *Principles of Internal Medicine: Acidosis and Alkadosis* 278 (14th ed., 1998).

[29] Contrary to an increase in respiratory rate, a decrease in the respiration rate is a compensatory mechanism for metabolic alkalosis. Pet. Ex. 16B at 6 (Thomas D. Dubose, Jr., *Acidosis and Alkalosis, in* HARRISON'S PRINCIPLES OF INTERNAL MEDICINE 282 (14th ed. 1998)).

[30] Abnormally high potassium concentration in the blood, most often due to defective renal function. DORLAND'S *supra* at 794.

[31] RICHARD E. BEHRMAN, M.D. ET. AL., TEXTBOOK OF PEDIATRICS 198 (16th ed.).

[32] "Because the value of vitreous potassium rises with an increase in postmortem time . . . ." MEDICOLEGAL INVESTIGATION OF DEATH GUIDELINSE FOR THE APPLICATION OF PATHOLOGY TO CRIME INVESTIGATION: PART 2 CHEMICAL CONSIDERATIONS 54,55 (3rd ed.).

shown to exhibit postmortem increase in concentration in vitreous humor in a linear fashion."[33] Res. Ex. E at 1. That phenomenon may be more pronounced in infants: "Several investigators have found that the postmortem level of vitreous potassium rises much more rapidly in the infant than in the adult."[34] Pet. Ex. 16C at 8.

Dr. Shane's claim that a potassium level of 10.5 mEq/L is inconsistent with life, Trans. at 28, 35, was not refuted by Dr. Anderson. Dr. Anderson testified that the upper level for normal for serum potassium level is 5 mEq/L. Trans. 80. However, Dr. Anderson doubted whether the level of potassium in the vitreous humor taken at autopsy was representative of the serum potassium level just prior to Tala's death. Dr. Anderson stated that "vitreous potassium can be higher than serum potassium; [t]hey're not exactly one to one." Trans. at 82. Additionally, and as indicated by medical literature filed by Respondent, potassium levels in the vitreous humor may vary between 5-15 mEq/L at 10-20 hours after death.[35] Resp. Ex. E at 3. Tala's autopsy, when the vitreous humor was extracted, was eighteen (18) hours after her death.[36] Dr. Shane presumably disagrees with this literature yet he did not provide this Court with any literature validating his disagreement. To the contrary, Petitioner's own article submission, "Medicological Investigation of Death," indicates that a "relatively low level of potassium" is "less than 15 mEq/L . . . ."[37] Pet. Ex. 16C at 6.

High levels of potassium in the serum can cause cardiac arrest. However, Petitioner's claim that the level of potassium in the vitreous humor extracted eighteen hours after Tala's death indicate the level of serum potassium just prior to Tala's death does not hold up under the scrutiny of the medical literature submitted by both Respondent and Petitioner. Again, Petitioner has not met her preponderance burden here.

f. Alternative Causation

When the Court, as it does here, finds that a petitioner's theory of causation does not meet the required preponderance of the evidence burden, an analysis of the respondent's proffered theory of alternative causation may be of value.[38] If the Court was to find Respondent's theory more

---

[33] Ross A James, et al. *Determination of Postmortem Interval by Sampling Vitreous Humor,* 18(2) AM. J. FORENSIC MEDICINE AND PATHOLOGY 158 (1997).

[34] John I. Coe, *Chemical Considerations*, MEDICOLEGAL INVESTIGATION OF DEATH: GUIDELINES FOR THE APPLICATION OF PATHOLOGY TO CRIME INVESTIGATION 56 (3rd ed.)

[35] Ross A. James, et al., *Determination of Post Mortem Interval by Sampling of the Vitreous Humor*, AM. J. FORENSIC MEDICINE 160 (1997).

[36] Tala's time of death was 3:26 p.m. on 28 April 1997 and the time of her autopsy examination was 9:30 a.m. on 29 April 1997. Pet. Ex. 6 at 3.

[37] Coe, *supra*, at 54.

[38] Finding that the neuroblastoma was or was not the cause of Tala's death would have no bearing on the outcome of this decision. Additionally, when petitioner has failed to meet their burden of proving causation by a preponderance of the evidence, respondent does not have the burden of proving alternative causation. *See* § 300aa-13(1).

probable than not the cause of Tala's death, it may at least resolve some of Petitioner's lingering doubt resulting from this opinion.

Here, Respondent, in agreement with the coroner's[39] report, proffered that Tala's cause of death was from "Neuroblastoma."[40] Pet. Ex. 6 at 1. To bolster her theory, Respondent filed one medical literature exhibit which lists the possible symptoms of neuroblastoma.[41] The exhibit states that symptoms of neuroblastoma will vary depending upon the site of the tumor. Res. Ex. C at 2. However, the exhibit does not give an in depth description of which symptoms occur with which location. Additionally, Respondent did not put forth any analysis as to which symptoms were more likely to be present due to the location of Tala's tumor. Thus, if the Court was to endeavor to analyze whether it was more likely than not that the neuroblastoma caused Tala's death, the Court would have to play the part of a medical expert. The Court is not qualified to do such.

When, as here, the petitioner fails to meet its burden, the Court does not have to augment its holding by finding an alternative cause of injury or, in this case, death. "[W]hen the special master concludes that a petitioner has *not* demonstrated by a preponderance of the evidence . . . causation, . . . [he] is not required to make a finding regarding alternative causation." *Bradley v. Secretary of Department of Health and Human Services*, 991 F.2d 1570, 1575 (D.C. Cir. 1993). Thus, having found that Petitioner has not met her burden, the Court ends its inquiry.

IV. Conclusion

This Court is painfully aware that Tala's family has suffered and will continue to suffer as a result of Tala's passing. However, the Court finds that the woof and warp of Petitioner's argument lacks sufficient evidence by a preponderance to prove that Tala's sudden death resulted from the DPT vaccine administered on 24 April 1997. Regretfully, entitlement must be denied for the foregoing reasons.

Accordingly, this petition is **DISMISSED** with prejudice, pursuant to Vaccine Rule 21, for failure to prove a *prima facie* case for entitlement under the Vaccine Act. In the absence of a motion for review filed pursuant to RCFC, Appendix B, the clerk is directed to enter judgment accordingly.

IT IS SO ORDERED.

Richard B. Abell

[39] Karen F. Ross, M.D., Medical Examiner for the Southwestern Institute of Forensic Sciences at Dallas was the coroner in this case. Pet. Ex. 6 at 1.

[40] Sarcoma of nervous system origin, composed chiefly of neuroblasts and affecting mostly infants and children up to 10 years of age. Most of such tumors arise in the automatic nervous system or in the adrenal gland. DORLAND'S *supra* at 1127.

[41] The filing is Respondent's Exhibit C (Res. Ex. C). The exhibit can also be found on the web at http://www.nlm.nih.gov/medlineplus/ency/article/001408.htm.

Special Master