

and Response to Petitioner's Motion for Ruling on the Record opining that this case is not appropriate for compensation and petitioner's Motion for Ruling on the Record should be denied. R Rule 4 (c) Report and Response to Petitioner's Motion for Ruling on the Record (hereinafter R Rule 4(c) Report and Response), filed Oct. 10, 2006. Petitioner filed an expert report from Dr. Marcel Kinsbourne, M.D. on March 12, 2007. Petitioner's Exhibit (hereinafter P Ex) 20, filed March 12, 2007. Respondent filed an expert report from Dr. John T. MacDonald, M.D. R Ex A, filed May 11, 2007. A Hearing was held in this matter on September 14, 2007, in Boston, MA. After the Hearing, at the undersigned's urging due to the belief that the damages were relatively minor, the parties discussed the possibility of informally resolving this matter. The parties were unable to informally resolve this matter and post-Hearing briefs were ordered. Order filed July 24, 2008. The parties filed post-Hearing briefs in compliance with the court's order. P Post-Hearing Brief (hereinafter P PH Brief), filed Sept. 5, 2008; R Post-Hearing Brief (hereinafter R PH Brief), filed Oct. 28, 2008. See Order filed July 24, 2008; Order filed October 10, 2008; Order filed October 24, 2008 (Non-pdf Order). The case took an odd procedural route thereafter.

After reviewing the evidence in the case and having prepared an opinion, the undersigned saw a major gap in petitioner's case, that is petitioner's expert relied upon statements by the mother that were not contained in the record. In addition, those statements conflicted with the medical records. Petitioner had been forewarned of the undersigned's concerns about the lack of factual development of this case. See Transcript of September 14, 2007, Hearing at 40; see also September 25, 2007, Order ("If settlement is not possible, Mrs. La Londe's testimony is essential.") Petitioner repeatedly rejected the exhortation to put on factual testimony. See Order filed April 17, 2009; see also Respondent's Response to Petitioner's Second Closing Brief at 1-7 (respondent accurately and succinctly details the procedural history of this case.)

However, the undersigned "encouraged settlement of the matter based upon petitioner's expert's testimony that the damages are limited." April 17, 2009 Order. At petitioner's request and to assist the parties in settling the dispute, the undersigned shared the draft decision with the parties. Id. This proved to be a mistake in that it unnecessarily prolonged the litigation of this case.

Petitioner's response effectively began a new litigation of this matter. Petitioner filed the unsigned narrative of Ms. La Londe, P Ex 52, medical literature, P Exs 53 and 54, and a life care plan, P Ex 55. In addition, reacting to the draft decision's criticism of her expert, petitioner requested time to consult with a new pediatric neurologist and "if appropriate, file a new, 'untainted,' expert opinion." Petitioner's Status Report/Response to the Court's Order of April 17, 2009 at 4. Respondent objected to this request. Respondent's Response filed June 11, 2009. Over respondent's objections, the undersigned ordered petitioner to file a status report indicating how petitioner would like to proceed. Order filed June 11, 2009.

Petitioner filed her status report on July 9, 2009. Moving away from her previous request to present an "untainted" report from a pediatric neurologist, petitioner requested an opportunity to address M.L.'s April 25, 2005, EEG. Petitioner's Status Report filed July 7, 2009, at 3. Respondent objected to this request. Respondent's Status Report filed August 4, 2009.

Petitioner filed on September 25, 2009, a supplemental expert report from Dr. Kinsbourne. P Ex 56. This was a major surprise to the undersigned, and respondent, because prior to its filing this report, petitioner's counsel represented to the court during a status conference that petitioner would have the EEG reviewed by an expert qualified to address EEGS. See Minute Entry of October 6, 2009 status conference. This assurance was given in response to

the undersigned's and respondent's questioning of Dr. Kinsbourne's admitted lack of qualifications to interpret EEGs. See Tr.at 28.

Following the filing of this report, petitioner spent seven months gathering medical records. Over respondent's objections, petitioner obtained a neurological evaluation of M.L. On October 13, 2010, petitioner filed the neurological evaluation of Dr. Ronald Davis. P Ex 64. In that report, Dr. Davis, relying on histories provided by the parents, opined that the vaccinations were the cause of M.L.'s neurological deficits. Id.

A status conference was conducted on April 29, 2011, during which petitioner requested a ruling on the record. May 2, 2011, Order. Respondent was given an opportunity to respond to Dr. Kinsbourne's supplemental report. Id. Respondent filed a supplemental report from Dr. MacDonald responding to both Drs. Kinsbourne and Davis on July 14, 2011. R Ex J.

During a status conference conducted on August 1, 2011, petitioner requested an opportunity to file a responsive report from Dr. Kinsbourne to Dr. MacDonald. Order filed August 2, 2011. After expressing concerns about Dr. Kinsbourne's expertise with EEGs, the undersigned suggested a hearing to fully ventilate the EEGs issues. Id. Petitioner was given an opportunity to consider how to proceed. Id. Petitioner ultimately opted to file a closing brief. Order filed September 19, 2011. Petitioner filed her closing brief; respondent responded and petitioner replied. The case has finally exhausted all procedures and is now ripe for decision.

The undersigned re-reviewed the entire record, including all materials filed subsequent to the drafting of the draft decision given to the parties after the April 9, 2009, status conference, and reaffirms the findings made in that draft decision. Despite the multitude of efforts, many of which will face legitimate arguments regarding their reasonableness, petitioner has failed to advance this case in any meaningful way. The essential defect, never cured, is that the factual predicate for the experts' opinions is information provided by the mother, which contains significant medical information that is not contained in the medical records. This defect was communicated to petitioner on several occasions, with strong advice to correct the deficiency by making mom available to testify. Petitioner for whatever reason rejected that advice. The second major defect, which petitioner simply ignores in her subsequent filings, is that the initial reaction to the immunizations resolved. That was the finding in the contemporaneous medical records, the testimony of Dr. Kinsbourne and the testimony of Dr. MacDonald. Dr. Kinsbourne's theory of the case was that there was a secondary, delayed anaphylactic reaction that caused M.L.'s injuries. However, there was no evidence to support such a delayed reaction, and more importantly there was no evidence showing that Dr. Kinsbourne's theory of harm was "at work" in M.L. Moberly v. Sec'y of Dept. of Health & Human Servs., 592 F.3d 1315, 1324 (Fed. Cir. 2005); see also Broekelschen v. Sec'y of the Dept. of Health & Human Servs., 618 F.3d 1339, 1345 (Fed. Cir. 2010), reh'g en banc denied (Dec. 8, 2010)("[A] petitioner must provide a reputable medical or scientific explanation that pertains specifically to petitioner's case.") That is because the harm would result from a hypoxic or anoxic event, or inflammation of the brain. Neither Dr. Kinsbourne, nor Dr. MacDonald saw any evidence in the medical records of such occurrences.

Petitioner was given multiple opportunities, more than would be reasonably given, or arguably should have been given, to prove her case. Petitioner rejected the opportunity to establish her factual case. Petitioner rejected the opportunity to put on additional expert testimony. In the end, petitioner relies upon additional filings from Dr. Kinsbourne addressing

one segment of the case, the issue of interpreting the EEGs, an area which he conceded he no longer practices. Tr. at 28. Petitioner also relies upon the evaluation and opinion of Dr. Davis, which in turn relies upon the statements of the parents. In doing so, petitioner perpetuates a deficiency in her case – the contemporaneous medical records conflict in meaningful ways with the parents’ histories. Lastly, petitioner advances arguments by counsel that contain counsel’s interpretation of the medical testing of M.L. Counsel is not a doctor. In essence, counsel is attempting to produce evidence through argument. Counsel’s efforts were discussed during status conferences. See Minute Entries for status conferences conducted on October 6, November 13 and November 30, 2009. It was explained therein that counsel’s interpretations conflicted with the experts’, including her own, interpretations of the tests. Thus, counsel would need to produce expert testimony to support her arguments. Special Master’s notes from the status conferences conducted on November 13 and 30, 2009. As seen, petitioner for whatever reason demurred. Counsel’s arguments thus remain unsupported and remain merely arguments, not evidence.

Accordingly, the undersigned rests on the previously drafted opinion. That opinion is reproduced, without substantive change, immediately below.

II. FACTUAL BACKGROUND

M.L. was born in autumn 2003 in Leesburg, Florida. M.L.’s mother suffered from pregnancy induced hypertension and on the day of M.L.’s birth, she experienced contractions and increased blood pressure and labor was augmented. P Ex 1 at 8, 11. M.L. was delivered via vaginal delivery at 37 weeks, 3 days gestation. P Ex 1 at 8. M.L. had Apgar scores of nine and nine at one and five minutes respectively. P Ex 1 at 8. M.L. had two episodes of apnea soon after his birth that resolved with stimulation and were attributed to elevated magnesium levels from medication given to M.L.’s mother. P Ex 3 at 261, 263, 250, 276. M.L. was discharged at three days of age. P Ex 3 at 254.

M.L. was seen two days later at Silver Lake Pediatrics and diagnosed with jaundice and weight loss. P Ex 4 at 3, 13. M.L. returned to have his weight re-checked on October 6, 2003 and was doing well. P Ex 4 at 3, 13.

Mr. La Londe reported that on November 9, 2003, he noticed around noon that day that M.L. was lethargic and around 5 PM M.L. became pale in color. P Ex 3 at 241. That day M.L.’s parents brought him to the Emergency Department (ED) at Leesburg Regional Medical Center (LRMC). P Ex 3 at 238-244. At the ED M.L. was noted to be alert, active and crying, with good color. P Ex 3 at 242-243. M.L. had a follow-up visit at Silver Lake Pediatrics where he was assessed with hypothermia. P Ex 4 at 12.

On December 1, 2003, during a two-month well-child check M.L. received the following immunizations: DTaP; HiB, Hep B, IPV, PCV7. P Ex 4 at 22-23. The office records from this visit the doctor noted mild postural stridor. P Ex 4 at 22. On January 26, 2004, M.L. received the following immunizations: DTaP, HiB, Hep B, IPV, and PCV7. P Ex 4 at 23. Thereafter, M.L. went in for a well-child visit at 12 months of age where he was noted to be speaking 1-3 words and using furniture to walk. P Ex 4 at 18. M.L. received his measles/mumps/rubella (MMR) immunization at this office visit. P Ex 4 at 18; see also P Ex 4 at 23. M.L. was seen at 15 months for a well-child visit where the records from his visit note that he is walking, but regarding speech it is noted that M.L. “[does not] want to talk.” P Ex. 4 at 17.

M.L. was seen on April 14, 2005 for an 18-month well-child visit. P Ex 4 at 15. At this visit while he did have a temperature of 99 degrees, his examination appeared to be otherwise normal. P Ex 4 at 15. He received the following immunizations on this visit: varicella, DTaP, HiB, and PCV7. P Ex 4 at 23.

The next morning, on April 15, 2005, M.L.'s parents brought him back to Silver Lake Pediatrics with the complaint of vomiting and fever. P Ex 4 at 10. The history recorded in the office records note that M.L. experienced a fever of 104.8 degrees at 4:00 PM on April 14 and he was given Tylenol. P Ex 4 at 10. By 8:00 PM his fever was down to 102-103 degrees, and later went back up. P Ex 4 at 10. Also noted was some facial, lip and tongue swelling, though time of onset of these symptoms is not documented in the office record. P Ex 4 at 10. During the April 15 visit M.L. was given intravenous (IV) Decadron, in addition to ephedrine, and an ambulance was called. Emergency Medical Services (EMS) received the call at 9:18 AM. The EMS history notes that M.L. was "showing skin pigmentation problems, red blotches, had febrile skin temp and edema of tongue, mouth and lips. [Patient's] airway is open." P Ex 5 at 5. M.L. was placed on oxygen and transported to the Emergency Department (ED) with an assessment of "allergic reaction." P Ex 5 at 5.

M.L. arrived at the ED at 10:10 AM at LRMC. P Ex 3 at 178. Triage notes indicate that M.L. had "reaction onset at [4:00 PM] 4/14/05; became flushed, swelling face/tongue, red/fever." P Ex 3 at 178. The ED physician saw M.L. at 10:40 AM, and the history taken notes tongue swelling and facial redness with onset that morning, in addition to being unable to swallow and displaying drooling accompanied with lip swelling. P Ex 3 at 176. Shortness of breath, rash and swelling were also noted during examination. P Ex 3 at 176. M.L. was treated with Tylenol and IV fluids. P Ex 3 at 176. Though M.L.'s temperature went down to 100.2 degrees when the physician reassessed him, he was admitted for further treatment and observation. P Ex 3 at 177. Notably, the physician wrote that "symptoms however have resolved." P Ex 3 at 177. An admission diagnosis of "**vaccine adverse reaction with secondary fever, angiodema, and anaphylactoid reaction**" was given. P Ex 3 at 174 (emphasis added). M.L. was treated with Decadron and Benadryl. P Ex 3 at 173. M.L. was discharged on April 16, 2005 and his parents were instructed to have him follow-up with his pediatrician in a week. P Ex 3 at 184.

On April 17, 2005, EMS was contacted again by M.L.'s mother, who called at 9:30 AM for hypothermia. P Ex 5 at 10. M.L.'s assessment was essentially normal with notations that his skin was "warm, flush" and that he was "agitated." P Ex 5 at 10. M.L. was transported to the hospital for "low temp." P Ex 5 at 8. While at the hospital various episodes of "becoming still, grinding his teeth jaw clenched" occurred during feeding attempts. P Ex 3 at 55. M.L. also experienced staring episodes that lasted a duration of ten seconds per episode. P Ex 3 at 55. M.L. was admitted with a diagnosis of "suspected seizures." P Ex 3 at 50. During M.L.'s stay at the hospital he underwent various testing, including a neurological evaluation from Dr. Eugene Tan on April 20, 2005. P Ex 3 at 158-60. An initial EEG was given, that was repeated due to an artifact, on April 20, 2005. P Ex 3 at 159; see also P Ex 3 at 73-74, 156. The repeat EEG showed "intermittent sharp-like wave and rhythmic slow wave in the left posterior region...suspicious of focal activity in that area." P Ex 3 at 72. M.L.'s parents reported two additional "seizure like" episodes during his stay at the hospital, which were followed by normal behavior. P Ex 3 at 86. Also on April 20, 2005, M.L.'s pediatrician, Dr. Cheas, completed and submitted a VAERS report, listing angiodema and possible seizures as adverse events. P Ex 4 at

79. The progress notes from April 21 state “no more seizure-like activity since Monday. Acting more like his normal self. [Vital signs] stable, afebrile ...,” and include the question as to whether the seizures are “idiopathic vs. vaccine related?” P Ex 3 at 48. On April 21, 2005, M.L. was discharged from the hospital with a prescription for Tegretol, an anti-convulsant medication, and instructions to schedule a MRI within one to two weeks and schedule a follow-up visit with Dr. Tan within three weeks. P Ex 3 at 75.

As directed, M.L. had a MRI performed on April 26, 2005, which was normal. P Ex 4 at 77. On May 12, 2005, Dr. Tan noted that M.L.’s parents reported that his episodes continued, for example when M.L. is trying to put blocks together he would suddenly clench his hands, grind his teeth and his facial muscles tense. P Ex 6 at 1-2. M.L. followed-up with his pediatrician Dr. Cheas on May 5, 2005 and experienced “so far no [seizures] now.” P Ex 4 at 10. His parents commented on his “abnormal behavior” which included M.L. holding his head and screaming, restlessness throughout the night, a lack of interest in books, toe-walking and constipation. P Ex 4 at 9-10. Dr. Cheas’ impression was a history of seizures and that it was questionable whether any of these signs or symptoms or his condition relates to his vaccination. P Ex 4 at 9.

M.L. continued to see Dr. Tan. The plan was to continue Tegretol, adjusting the dosage as necessary, and refer M.L. to a speech therapist for evaluation. P Ex 6 at 1-2 (May 12, 2005 office visit, parents reported continued episodes, Dr. Tan noted that the episodes occurred “only while he’s in high chair strapped down”); P Ex 6 at 3 (July 12, 2005 office visit, seizures controlled, starting to talk more, parents concerned not at level he was at prior to seizures).

M.L. saw a speech therapist on July 21, 2005. P Ex 9 at 14-15. The speech therapist was unable to perform formal testing as M.L. did not follow oral or motor commands. P Ex 9 at 14-15. M.L.’s parents informed the doctor that M.L. lost most of his vocabulary, currently saying only three words, and reported that previously M.L. was advanced with spatial relations. P Ex 9 at 14-15. Speech therapy once a week was recommended in an effort to increase M.L.’s vocabulary. P Ex 9 at 15.

In August 2005, Mrs. La Londe called Dr. Tan’s office on two occasions and on both occasions Dr. Tan increased M.L.’s Tegretol based on behaviors Mrs. La Londe described. P Ex 6 at 2 (Mrs. La Londe called on Aug. 1 and Aug. 30, frequent “seizures” described as grinding his teeth, sometimes staring, sometimes facial grimacing followed by lethargy). M.L. was evaluated by a pediatric neurologist, Dr. Renato Gonik, on September 26, 2005. P Ex 18 at 1-2. Her impression was that M.L. may have Landau-Kletler syndrome based on his history of seizure disorder and language regression. An EEG was recommended. P Ex 18 at 2.

M.L. followed up with Dr. Tan on November 10, 2005, at which time M.L.’s parents reported that M.L.’s hair was falling out and he was having spells almost daily. P Ex 6 at 4. Dr. Tan’s assessment was “complex partial seizures.” P Ex 6 at 4. M.L.’s Tegretol was decreased and M.L. was started on Keppra. P Ex 6 at 4.

Shortly thereafter, on November 25, M.L. was admitted to Arnold Palmer Children’s Hospital in Orlando, Florida for evaluation after episodes of screaming, seizure-like response to colors and shapes on the walls, possibly due to hallucinations, sensitivity to light and a temperature high of 102 degrees. P Ex 7 at 8-9. It was reported that M.L.’s seizures have changed from simple partial seizures to complex partial seizures in the past few weeks, and were

occurring with increased frequency. P Ex 7 at 8-9. It was noted that M.L. did not verbalize during his examination. P Ex 7 at 12. While at the hospital M.L. had a normal CT scan and MRI except for pan-sinusitis. P Ex 7 at 12. A lumbar puncture was unsuccessfully attempted due to the patient being too active. P Ex 7 at 12. An EEG was also performed, and was found within normal limits except for bi-posterior quadrant slowing, “possibly indicating postictal state,” with no epileptic discharges. P Ex 7 at 83. A 24-hour video EEG was performed the following day, on November 27, 2005. P Ex 7 at 83. The impression was that the EEG result was mildly abnormal, secondary to presence of mild intermittent bi-posterior quadrant slowing. P Ex 7 at 83. Again, no epileptiform discharges were seen. P Ex 7 at 83. M.L.’s spells of fisting of the right hand, shivering-like of the mouth and clenching of the teeth “did not have electrographic seizure correlate.” P Ex 7 at 80-81. M.L. was weaned off Tegretol and Kepra during his hospital admission. P Ex 7 at 5. His discharge diagnosis was “stereotypic behavior, no infection, no seizure activity.” P Ex 7 at 5.

M.L. had his hearing tested by Dr. Clifford Dubbin on December 27, 2005. P Ex 10 at 8. Dr. Dubbin’s impression was that M.L. had fluid behind both ears that was causing conductive hearing loss. P Ex 10 at 8. He noted that in his experience he has seen nerve deafness from a vaccination, at which point he recommended an audiogram. P Ex 10 at 8.

M.L. had a pediatric neurological evaluation performed by Dr. Jasna Kojic on January 9, 2006. P Ex 11 at 2-4. The history notes that M.L. stopped talking and experienced regression in his fine motor skills subsequent to his hospitalization in April 2005. P Ex 11 at 2. Dr. Kojic’s impression was:

It appears at this point that his developmental delay, repetitive and ritualistic behavior would probably categorize him in autistic spectrum disorder category, most likely pervasive developmental disorder- not otherwise specified. It is puzzling that apparently his development was age appropriate up until 18 months when he had his routine immunization resulting in severe allergic reaction.

P Ex 11 at 3-4. Dr. Kojic recommended that M.L. continue with speech therapy and occupational therapy. P Ex 11 at 3-4.

M.L. was seen by a pediatric developmental specialist, Dr. Joseph Keeley on January 24, 2006. Since being weaned off the antiepileptic medication his screaming and screeching episodes completely stopped. P Ex 11 at 2. Dr. Keeley’s impression was that M.L. has had steady improvements since he was last seen by Dr. Kojic and no longer “makes the diagnosis of pervasive developmental disorder.” P Ex 12 at 8. Dr. Keeley’s impression was that of expressive aphasia, with M.L. continuing to have “steady difficulties with expressive language. But otherwise, is functioning very well.” P Ex 12 at 8. M.L. was noted to have a preference for walking on his toes. Additionally, he noted “no sign of central nervous problems at this time.” P Ex 12 at 1-2.

M.L. had a normal EEG on March 30, 2006. P Ex 12 at 4. It is noted that he “has shown improvement in all areas” and his speech has improved. P Ex 12 at 2. M.L. continued to make progress, following up with doctors as well as receiving speech, occupational and physical therapy. See P Ex 23 at 2 (speech evaluation); P Ex 44 at 3-4 (occupational therapy evaluation); P Ex 31 at 1 (physical therapy evaluation); P Ex 31 at 1 (office visit with Dr. Keeley); P Ex 42 at 1-4 (M.L. continues to receive speech therapy); P Ex 44 at 2 (M.L. completed occupational

therapy, seen 33 times, discharge diagnosis of hypotonia); P Ex 43 at 2 (M.L. continues to receive physical therapy).

III. LEGAL STANDARD

Causation in Vaccine Act cases can be established in one of two ways: either through the statutorily prescribed presumption of causation or by proving causation-in-fact. Petitioners must prove one or the other in order to recover under the Act. According to §13(a)(1)(A), claimants must prove their case by a preponderance of the evidence.³

For presumptive causation claims, the Vaccine Injury Table lists certain injuries and conditions which, if found to occur within a prescribed time period, create a rebuttable presumption that the vaccine caused the injury or condition. 42 U.S.C. §300aa-14(a). The undersigned finds that petitioner did not provide evidence of a Table Injury occurring within the prescribed time frame required by the Vaccine Injury Table. Thus, petitioners must prove that the vaccinations in-fact caused M.L.'s injuries, a so-called "off-Table" case.

To demonstrate entitlement to compensation in an off-Table case, petitioners must affirmatively demonstrate by a preponderance of the evidence that the vaccination in question more likely than not caused or significantly aggravated the injury alleged. See, e.g., Bunting v. Sec'y of Dept. of Health & Human Servs., 931 F.2d 867, 872 (Fed. Cir. 1991); Hines v. Sec'y of Dept. of Health & Human Servs., 940 F.2d 1518, 1525 (Fed. Cir. 1991); Grant v. Sec'y of Dept. of Health & Human Servs., 956 F.2d 1144, 1146, 1148 (Fed. Cir. 1992). See also §§11(c)(1)(C)(ii)(I) and (II). To meet this preponderance of the evidence standard, "[petitioners must] show a medical theory causally connecting the vaccination and the injury." Grant, 956 F.2d at 1148 (citations omitted); Shyface v. Sec'y of Dept. of Health & Human Servs., 165 F.3d 1344, 1353 (Fed. Cir. 1999). A persuasive medical theory is shown by "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury." Hines, 940 F.2d at 1525; Grant, 956 F.2d at 1148; Jay v. Sec'y of Dept. of Health & Human Servs., 998 F.2d 979, 984 (Fed. Cir. 1993); Hodges v. Sec'y of Dept. of Health & Human Servs., 9 F.3d 958, 961 (Fed. Cir. 1993); Knudsen v. Sec'y of Dept. of Health & Human Servs., 35 F.3d 543, 548 (Fed. Cir. 1994). Furthermore, the logical sequence of cause and effect must be supported by "[a] reputable medical or scientific explanation" which is "evidence in the form of scientific studies or expert medical testimony." Grant, 956 F.2d at 1148; Jay, 998 F.2d at 984; Hodges F.3d at 960;⁴ see also H.R. Rep. No. 99-908, Pt. 1, at 15 (1986), reprinted in 1986 U.S.C.C.A.N. 6344.

³ A preponderance of the evidence standard requires a trier of fact to "believe that the existence of a fact is more probable than its nonexistence before the [special master] 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, 372-73 (1970) (Harlan, J. concurring) (quoting F. James, CIVIL PROCEDURE, 250-51 (1965)). Mere conjecture or speculation will not establish a probability. Snowbank Enter. v. United States, 6 Cl. Ct. 476, 486 (1984).

⁴ The general acceptance of a theory within the scientific community can have a bearing on the question of assessing reliability while a theory that has attracted only minimal support may be viewed with skepticism. Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579, 594 (1993). Although the Federal Rules of Evidence do not apply in Program proceedings, the United States Court of Federal Claims has held that "Daubert is useful in providing a framework for evaluating the reliability of scientific evidence." Terran v. Sec'y of Dept. of Health & Human Servs., 41 Fed. Cl. 330, 336 (1998), aff'd, 195 F.3d 1302, 1316 (Fed. Cir. 1999), cert. denied, Terran v. Shalala, 531 U.S. 812 (2000). In Daubert, the Supreme Court noted that scientific knowledge "connotes more than subjective belief or unsupported speculation." Daubert, 509 U.S. at 590. Rather, some application of the scientific method must have been employed to validate the expert's opinion. Id. In other words, the "testimony must be supported by appropriate validation – i.e., 'good grounds,' based on what is known." Id. Factors relevant to that

While petitioners need not show that the vaccine was the sole or even predominant cause of the injury, petitioners bear the burden of establishing “that the vaccine was not only a but-for cause of the injury but also a substantial factor in bringing about the injury.” Shyface, 165 F.3d at 1352-53. Petitioners do not meet their affirmative obligation to show actual causation by simply demonstrating an injury which bears similarity to a Table injury or to the Table time periods. Grant, 956 F.2d at 1148. See also H.R. Rep. No. 99-908, Pt. 1, at 15 (1986), reprinted in 1986 U.S.C.C.A.N. 6344. Nor do petitioners satisfy this burden by merely showing a proximate temporal association between the vaccination and the injury. Grant, 956 F.2d at 1148 (quoting Hasler v. United States, 718 F.2d 202, 205 (6th Cir. 1983), cert. denied, 469 U.S. 817 (1984) (stating “inoculation is not the cause of every event that occurs within the ten day period [following it]. . . . Without more, this proximate temporal relationship will not support a finding of causation”)); Hodges, 9 F.3d at 960. Finally, petitioners do not demonstrate actual causation by solely eliminating other potential causes of the injury. Grant, 956 F.2d at 1149-50; Hodges, 9 F.3d at 960.

In Althen v. Sec’y of Dept. of Health & Human Servs., 418 F.3d 1274,1278 (Fed. Cir. 2005), the Court of Appeals for the Federal Circuit reiterated that petitioners’ burden is to produce “preponderant evidence” demonstrating: “(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 the vaccination and injury.” The Federal Circuit stated further that “requiring that the claimant provide proof of medical plausibility, a medically acceptable temporal relationship between the vaccination and the onset of the alleged injury, and the elimination of other causes – is merely a recitation of this court’s well established precedent.” Id. at 1281. The Federal Circuit concluded that to support petitioners theory of causation, there is no requirement in the Vaccine Act’s preponderant evidence standard that petitioners submit “objective confirmation,”

determination may include, but are not limited to:

Whether the theory or technique employed by the expert is generally accepted in the scientific community; whether it’s been subjected to peer review and publication; whether it can be and has been tested; and whether the known potential rate of error is acceptable.

Daubert v. Merrell Dow Pharmaceuticals, Inc., 43 F.3d 1311, 1316 (9th Cir. 1995) (Kozinski, J.), on remand from, 509 U.S. 579 (1993); see also Daubert, 509 U.S. at 592-94.

However, the court also cautioned about rejecting novel scientific theories that have not yet been subjected to peer review and/or publication. The court pointed out that the publication “does *not* necessarily correlate with reliability,” because “in some instances well-grounded but innovative theories will not have been published.” Daubert, 509 U.S. at 594. However, the Supreme Court’s only guidance to lower courts in determining the reliability of a novel proposition is that

. . . submission to the scrutiny of the scientific community is a component of “good science,” in part because it increases the likelihood that substantive flaws in methodology will be detected. The fact of publication (or lack thereof) in a peer reviewed journal thus will be a relevant, though not dispositive, consideration in assessing the scientific validity of a particular technique or methodology on which an opinion is premised.

Id. at 593-94; see Althen, 418 F.3d at 1280 (“The purpose of the Vaccine Act’s preponderance standard is to allow the finding of causation in a field bereft of complete and direct proof of how vaccines affect the human body.”); see also, Gall v. Sec’y of Dept. of Health & Human Servs. , No. 91-1642V, 1999 WL 1179611, at *8 (Fed. Cl. Spec. Mstr. Oct. 31, 1999).

such as medical literature. Id. at 1279. The Federal Circuit explained that requiring medical literature “prevents the use of circumstantial evidence envisioned by the preponderance standard and negates the system created by Congress, in which close calls regarding causation are resolved in favor of the injured claimants.” Id. at 1280 (citing Knudsen, 35 F.3d 543, 549 (Fed. Cir. 1994)); see also Capizzano v. Sec’y of Dept. of Health & Human Servs., 440 F.3d 1317, 1325 (Fed. Cir. 2006) [hereinafter “Capizzano III”]. Moreover, the Federal Circuit stated, “The purpose of the Vaccine Act’s preponderance standard is to allow the finding of causation in a field bereft of complete and direct proof of how vaccines affect the human body.” Id.

The Federal Circuit affirmed Althen’s three-part test in Capizzano III and in Pafford v. Sec’y of Dept. of Health & Human Servs., 451 F.3d 1352 (Fed. Cir. 2006). The panel in Pafford, however, explained that the three prongs in Althen “must cumulatively show that the vaccination was a ‘but-for’ cause of the harm, rather than just an insubstantial contributor in, or one among several possible causes of, the harm.” Pafford, 451 F.3d at 1355. Fairly interpreted, the Pafford court held that it is petitioner’s burden to rule out other competing possible causes of the injury in establishing that the vaccine was the “but-for cause of the harm.” Id. at 1355, 1357; see also Althen at 1281. (“[T]he elimination of other causes [] is merely a recitation of this court’s well-established precedent.”). But see Walther v. Sec’y of Dept. of Health & Human Servs., 485 F.3d 1146, 1150 (Fed. Cir. 2007) (“[W]e conclude that the Vaccine Act does not require petitioner to bear the burden of eliminating alternative causes when the other evidence on causation is sufficient to establish a prima facie case.”).

However, the legal requirement that petitioners support their proposed causation theory with a “sound and reliable medical or scientific explanation” is undisturbed. Knudsen, 35 F. 3d 543, 548 (Fed. Cir. 1994); see also Grant, 956 F.2d at 1148 (“A reputable or scientific explanation must support this logical sequence of cause and effect.”). Thus, when considering the evidence in a case, the special master is to “consider all relevant and reliable evidence, governed by the principles of fundamental fairness to both parties.” Vaccine Rule 8(c); see also Campbell v. Sec’y of Dept. of Health & Human Servs., 69 Fed. Cl. 775, 781 (2006) (Althen’s requirement of a “reputable medical or scientific explanation” “[l]ogically [] requires a special master to rely on reliable medical or scientific evidence”); Manville v. Sec’y of Dept. of Health & Human Servs., 63 Fed. Cl. 482, 491 (2004) (“Daubert adequately serves the gatekeeping function for analysis of the admissibility of evidence; once evidence has passed that test, the trier of fact’s process, simply, is to determine the probativeness of that evidence.”); DeBazan v. Sec’y of Dept. of Health & Human Servs., 70 Fed. Cl. 687, 699 n.12 (2006) *rev’d* 539 F.3d 1347 (2008) (reversed on other grounds). Petitioner’s case is measured against these standards.

IV. DISCUSSION

A. Summary of Experts’ Positions

The following is a brief overview of the experts’ background and opinions.

Marcel Kinsbourne, M.D.

During the Hearing, held on September 14, 2007, the parties stipulated to Dr. Kinsbourne’s qualifications to testify as a pediatric neurologist. Tr. at 5. Dr. Kinsbourne is certified in neurology and other medical specialties in the U.K. Tr. at 29. This certification was

accepted by Duke University Medical Center to qualify Dr. Kinsbourne for a tenured professorship and the head of the division of child neurology when he first moved to the United States. Tr. at 29. Dr. Kinsbourne stated that he sees approximately 15-20 patients per year, mostly pediatric. Tr. at 26. Generally, the nature of his patients' neurological disorders are disorders of mental development, for example attention deficit disorder, language delay, autistic type manifestations. Tr. at 27. Dr. Kinsbourne added that beginning in 1992 he radically reduced his patient related practice to his current numbers. Tr. at 27-28. Dr. Kinsbourne acknowledged that he has always been an academic neurologist and since "the early 90's divided his time between teaching, research, and seeing patients." Tr. at 27.

Dr. Kinsbourne's theory of this case is that M.L. suffered from a persistent anaphylactic reaction that did not go away and consisted of two-phases, the acute early phase and a late-phase. Dr. Kinsbourne clarified in his testimony that in this case he considered the first stage to be a delayed onset anaphylactic reaction, which appeared to clinically resolve, with the second stage also presenting as a late-phase reaction. Tr. at 88. He testified that although the initial reaction clinically resolved, "we don't know what was happening on the tissue level. But something could have been going on." Tr. at 88. He clearly states that there is no evidence regarding this issue. Tr. at 88. He theorizes that the on-going anaphylactic reaction caused brain damage that resulted in M.L.'s alleged injuries. Dr. Kinsbourne, while acknowledging that he cannot detail the actual mechanism in this case, stated that based on M.L.'s anaphylactic reaction there exist several reasonable mechanisms for brain damage; continuing wide-spread inflammation, cytokine release, and edema induced loss of oxygen. Tr. at 22-24; see also P PH Brief filed Sept. 5, 2008. Dr. Kinsbourne states that these mechanisms can cause organ damage and in this case the damaged organ was M.L.'s brain. P Ex 20.

Dr. Kinsbourne points to the abnormal EEGs as evidence of left hemisphere damage resulting in speech loss. Tr. at 12. Dr. Kinsbourne also attributes M.L.'s right-sided clumsiness with a left hemisphere injury. Tr. at 52; P Ex 20 at 5. Dr. Kinsbourne stated that a compelling reason for finding that the vaccination caused M.L.'s injury was "that until the vaccination, the child was talking at an age-appropriate level, and after the vaccination, his expressive language development was found to be considerably behind." Tr. at 78.

John T. MacDonald, M.D.

Dr. MacDonald is board-certified in child neurology and has been practicing in the field of pediatric neurology for over thirty years. Tr. at 93. Dr. MacDonald's current practice involves spending three days a week teaching and supervising residents at a university; two of those days are half-day clinics seeing patients. Tr. at 94. Additionally, Dr. MacDonald maintains a partial private practice two half-days a week where he sees private patients. Dr. MacDonald estimated that he sees about 40-50 patients a week, not including inpatients. Tr. at 94. His experience in diagnosing and treating children with seizure disorders involves seeing patients both in private practice and at the university. Dr. MacDonald stated that although he sees "a fair amount of newborns" the majority of the "children are between four months and four years and then some older children." Tr. at 95. Further, he stated that his experience with reading electroencephalograms, (hereinafter EEG), entails reading pediatric EEGs roughly 3-4 days a week, which has been consistent throughout his practice as a pediatric neurologist. Tr. at 95.

In his report, Dr. MacDonald opined that while M.L. did experience an allergic reaction to the vaccinations given on April 14, 2005, “he never had anaphylactic shock and was never in any significant cardio-respiratory distress.” R Ex A at 3. Dr. MacDonald stated that M.L. was treated appropriately for his allergic reaction, which may have been a mild anaphylactic reaction with some indicators of a hypotonic/hyporesponsive, and made a good initial recovery. R Ex A at 3. Dr. MacDonald testified that there were no clinical signs of shock and “no measurement to suggest that [M.L.] suffered a severe compromise to maintain blood flow and oxygen.” Tr. at 97-98. He stated that M.L. never really had any significant respiratory compromise that would make M.L. hypoxic enough to result in brain damage. Tr. at 98. In fact, his initial testing was fine; there was nothing to suggest hypotension. Id. In addition, he was described as doing “fairly well.” Id. M.L. did experience “spells” which his doctors interpreted as seizures, and he was subsequently placed in antiepileptic medication for a period of time. R Ex A at 3. Dr. MacDonald suggests that M.L.’s speech delays and behaviors may be explained, in part, as secondary to medication and mild conductive hearing loss due to some existing middle ear fluid during this time period. R Ex A at 3. In addressing the abnormal EEGs Dr. MacDonald details various reasons why the abnormalities, if determined to be real and significant, “don’t really speak to an acquired aphasia.” Tr. at 106-11. In conclusion Dr. MacDonald opines that M.L.’s speech delays, involving delayed expressive speech skills, “is not typically the result of an acute brain injury in the absence of other neurological signs and symptoms of pathology noted in brain imaging studies,” and he does not think M.L.’s current neurological status is the result of his allergic reaction following the vaccinations given on April 14, 2005. R Ex A at 3.

B. Analysis

Petitioner’s Claim of a Table Injury and Causation-in-Fact

Petitioner initially asserted a table injury in this case, alleging an anaphylactic reaction within four hours of receipt of the DTaP vaccination. Tr. at 6. Anaphylaxis or anaphylactic shock must occur within 0-4 hours to qualify as a Table Injury for Tetanus-toxoid containing vaccines (e.g., DTaP, Tdap, DTP-Hib, DT, Td, TT) according to the Vaccine Injury Table. National Childhood Vaccine Injury Act Vaccine Injury Table, available at: <http://www.hrsa.gov/vaccinecompensation/table.htm>. It became clear through Dr. Kinsbourne’s testimony that he relied on statements provided from the mother that were not in the record in determining that M.L. experienced a reaction within four hours of vaccination. Tr. at 32 (referring to P Ex 20 at 1 “[M.L.]’s vaccination had been scheduled for 10:30, but there was a delay, so it was administered at 12:00”); see also P Ex 4 at 10 (pediatrician’s notes from April 15, “events happened at 4:00PM). Dr. Kinsbourne did not look at the medical records for a time of vaccination and did not believe the timing of onset was material to his opinion or important due to the parties being in agreement that an anaphylactic reaction occurred. Tr. at 20-21; see also Tr. at 35. When questioned, he agreed that he should have considered that the time of vaccination is listed on the VAERS report as 10:45 and in the EMS record as 10:30. Tr. at 33, 47; P Ex 4 at 79; P Ex 5 at 5. The records indicate that no evidence of an anaphylactic reaction appeared until about five hours after the vaccination. See P Ex 4 at 10; see also Tr. at 19-20. Thus, the record does not support onset of anaphylaxis or an anaphylactic reaction within four hours of vaccination, and accordingly petitioner cannot establish a table-injury of anaphylactic shock.

In the alternative, Dr. Kinsbourne asserted that petitioner suffered causation-in-fact injuries. Thus, the issue to be resolved is whether petitioner demonstrated by the preponderance

of the evidence that the DTaP vaccination M.L. received on April 14, 2005 was the cause-in-fact of his alleged injuries of expressive aphasia.⁵ For the following reasons the undersigned finds that although petitioner proved causation-in-fact that M.L. experienced an anaphylactic reaction this reaction resolved. Petitioner was unable to prove a six-month sequelae, that is that M.L.'s current injuries are due to his vaccination.

Petitioner Failed to Provide a Logical Sequence of Cause and Effect

After evaluating Dr. Kinsbourne's general causation theory the undersigned finds that petitioner failed to provide a logical sequence of cause and effect. Dr. Kinsbourne's general causation theory is that:

In the great majority of cases of anaphylaxis, an acute reaction may even happen sooner after the administration of the agent that it did in [M.L.]. And that reaction may either kill the child or otherwise, typically the child will completely recover without sequelae. But in 1 cases in 10, the anaphylactic reaction does not go away and can continue for up to several days. In other words, the inflammation which is part of the mechanism of the reaction may continue and may remain evident in terms of the various organ-related deficits over that period of time. So it is well-recognized that there is at times a late phase of anaphylaxis as well as the acute early reaction.

Tr. at 7. Dr. Kinsbourne testified that M.L. experienced a two-stage reaction, the first stage described as his admission to the hospital on April 15, 2005 and the second stage presented on April 17, 2005. Tr. at 8. Dr. Kinsbourne supplied literature that addresses two-stage reactions, notably the stages described consist of an immediate response followed by a second late-phase response that "sets in 2-24 hours later...and may last for several days." P Ex 20, Tab A at 2 (unnumbered); see also P Ex 20 at 5 (Dr. Kinsbourne's Expert Report). The literature described the two-phase reactions as follows:

Many local type I hypersensitivity reactions have two well-defined phases. The immediate, or initial, response is characterized by vasodilation, vascular leakage, and depending on the location, smooth muscle spasms or glandular secretions. These changes usually become evident within 5 to 30 minutes after exposure to an allergen and tend to subside in 60 minutes. In many instances (e.g., allergic rhinitis and bronchial asthma), a second, late-phase reaction sets in 2 to 24 hours later without additional exposure to antigen and may last for several days.

P Ex 20, tab A at 2 (unnumbered). While the literature clearly states that a late-phase reaction can last for several days, Dr. Kinsbourne was unable to point to anywhere in the literature that describes the sequence that presented in this case. Tr. at 22-23; P Ex 20, tab A at 2 (unnumbered). Dr. Kinsbourne testified that the literature submitted does not support the notion of anaphylactic shock causing the type of injury in this case. Tr. at 21. Moreover, Dr. Kinsbourne was unable to find any case reports in support of this type of sequelae. Tr. at 22-24.

⁵ Although Dr. Kinsbourne testified that M.L.'s injury was expressive aphasia at times he purports in his testimony that M.L.'s injuries included right-sided clumsiness. Tr. at 12-13; Tr. at 52-53("So my opinion is mainly centered on speech ability at some level which abruptly decreased and still hasn't recovered into the normal range"); Tr. at 54 (Dr. Kinsbourne notes "right hand somewhat clumsy," "right leg gives way" when walking).

He stated “[h]ad I had a reference available which said something of the order of one in 1000 anaphylactic reactions results not in death but in some permanent focal injury to the brain, I would have filed it with the Court.” Tr. at 22-23. Dr. Kinsbourne acknowledged that he has never seen this sequence before and it is beyond rare. Tr. at 23. Dr. Kinsbourne explained that the mechanism of the April 17 event, almost three days post-vaccination was not totally clear to him and his understanding of how the reaction could evolve in this way is incomplete. Tr. at 23, 25.

Lacking Factual Basis in Support of Theory

Even assuming reliable support for Dr. Kinsbourne’s theory there is no factual basis to support a sequence of events occurring as Dr. Kinsbourne theorized. Dr. Kinsbourne relies on the mother’s narrative in support of his theory. Tr. at 39. (The undersigned took judicial notice of the fact that Dr. Kinsbourne got his information from the mother’s narrative.). This narrative, for reasons that will be addressed below, was not filed into the record. It is clear from both the experts’ testimony and experts’ reports that both experts agree that M.L. suffered from an anaphylactic reaction to the vaccinations he received on April 14, 2005. The critical issue of contention is what was the duration and severity of the anaphylactic reaction? Petitioner’s theory of causation hinges on the anaphylactic reaction not resolving and persisting in smoldering form. Dr. Kinsbourne’s theory is that M.L. suffered from a two-stage late-onset anaphylactic reaction; the first stage starting sometime between April 14 and April 15, which appeared to clinically resolve on April 16, with the anaphylactic reaction still present in smoldering form, which subsequently developed into the second stage of the anaphylactic reaction on April 17, 2005. Dr. Kinsbourne opined that the extended anaphylactic reaction caused brain damage that resulted in M.L.’s alleged injury of expressive aphasia and right-sided weakness. Tr. at 11. Dr. Kinsbourne proposed three general mechanisms for the anaphylactic reaction causing brain damage: (1) inflammation (2) cytokine release (3) loss of oxygen. Tr. at 7 (inflammation part of mechanism); Tr. at 11 (diminution of blood flow, oxygenation).

Dr. Kinsbourne proposed that “the anaphylactic reaction sometimes progresses into a phase in which widespread inflammation as well as anoxia causes organ damage. Tr. at 11-12. In [M.L.]’s case, the damaged organ was the brain.” P Ex 20 at 5; Tr. at 12. Dr. Kinsbourne suggested inflammation as a reasonable mechanism because it is a well-known feature of delayed anaphylaxis. Tr. at 68. He opined generally that when reactions occur, “two things are apt to occur in various parts of the body,” inflammation and anoxia. Tr. at 74. In immune reactions of this kind, Dr. Kinsbourne testified that vasculitis is a component of the reaction. Tr. at 70. As such “it still remains medically possible that blood vessels supplying the left back of the cerebral hemisphere constricted to the point that the blood flow was insufficient to that particular area.” Tr. at 70. He stated that if the blood flow were indeed cut off a stroke may have occurred which would be evident on a CT scan or MRI. Tr. at 70-71. In this case there was no evidence of a stroke on either the CT scan performed or the MRI. Tr. at 71.

Dr. MacDonald disagrees with Dr. Kinsbourne’s theory that M.L. suffered a two-stage late-phase anaphylactic reaction. Dr. MacDonald opined that M.L.’s anaphylactic reaction was self-limited and clinically resolved by April 16, 2005. Tr. at 96 (M.L. had an anaphylactic reaction, then “the process clears”); Tr. at 99 (“I assume [the doctors] felt he was okay to go home, that he was back to his baseline, that the initial reaction had run its course.”). Dr. MacDonald stated that even if it was assumed that M.L.’s anaphylactic reaction consisted of two-stages and persisted beyond April 16, 2005, Dr. MacDonald does not believe M.L. displayed

the clinical signs during this time frame that one would expect to see if general mechanisms for brain damage Dr. Kinsbourne proposed were actually taking place. Tr. at 98 (“There are no clinical signs,” no evidence of either hypoxia or anoxia, “**clinically, he is described as doing fairly well**”). Dr. MacDonald also found that the EEGs from M.L.’s medical record did not present in the manner of being diffusely and grossly abnormal which would be expected if M.L. truly went into anaphylactic shock and that shock was persistent enough to cause any permanent brain damage. Tr. at 100.

In the undersigned’s review of the facts in this case it is clear M.L. experienced an initial anaphylactic reaction that is documented in the medical records. P Ex 3 at 158 (documented history given during 04/19/05 hospital admission). M.L.’s parents noticed fever during the afternoon of the date of M.L.’s vaccinations and through the night. P Ex 3 at 158. They called his pediatrician Dr. Cheas several times and Dr. Cheas told them in effect to hang in there according to the mother’s unfiled narrative. Tr. at 48. Dr. Kinsbourne relies on the mother’s narrative in describing M.L. as limp and floppy like a rag doll on the evening of his vaccinations. Tr. at 46-47. Respondent questioned Dr. Kinsbourne regarding support for the presence of these symptoms:

RESPONDENT: Let me ask you, what do you think a pediatrician would advise if they were given a description of a child who was limp and sloppy like a rag doll, wheezing, not focusing with his eyes glazed and rolled up? What would a pediatrician tell a parent to do?

DR. KINSBOURNE: I think a reasonable doctor would at least want to see the child or have the child go to the hospital. But I don’t know what the parents told the doctor.

Tr. at 48. Dr. Kinsbourne later notes that there are a number of manifestations described by the mother, such as wheezing and pallor and limpness, which are not recorded in the medical records. Tr. at 51; see P Ex 19 (transcription of notes from Dr. Cheas from April 15, 2005 office visit, “no wheezing,” “no focal neurological signs”).⁶ The parents brought M.L. to Dr. Cheas’ office the morning of the April 15 and M.L. was given Decadron and Benadryl and EMS was summoned. P Ex 3 at 158. M.L. was admitted for 24-hour observation at the hospital and was **discharged in good condition** on April 16. P Ex 3 at 158 [emphasis added]. During this admission M.L.’s father “claimed that while he was inside his crib, he was acting a bit groggy and slumped forward and hit is right face on the railing of the crib, but no loss of consciousness.” P Ex 3 at 158.

The following morning, April 17, his father noticed M.L. to be hypothermic and kind of unresponsive. P Ex 3 at 158. His parents took his temperature, which was noted to be 96 degrees Fahrenheit and placed a call into Dr. Cheas. After following Dr. Cheas’ instructions to give M.L. a warm bath and some juice his parents attempted to feed him. P Ex 3 at 158. It was during this feeding attempt that the parents observed M.L. experiencing “episodes” which they described as occurring when M.L. was sitting in a high-chair, his face tensing, lower lips pulled down, turning head left then right with eyes staring straight and unresponsive and then he would

⁶ Dr. Kinsbourne relies on the mother’s unfiled narrative as evidence that these symptoms were present. Petitioner’s counsel made a decision not to file the narrative. See infra 17-18. As stated in the Procedural Background of this decision, Mrs. La Londe’s unsigned narrative was filed as P Ex 52. It is assumed that this is the narrative that Dr. Kinsbourne relied upon for his factual information.

appear lethargic after each episode. P Ex 3 at 158. The parents decided to bring M.L. back to the ED. P Ex 3 at 158. During M.L.'s second admission to the hospital his blood tests and urinalysis all came back within normal limits. P Ex 3 at 158. Additionally, his CT scan came back normal and spinal tap fluid was negative. P Ex 3 at 158. M.L.'s parents related that he is not as active as "he always has been and he seemed to be holding the right occipital area and whine a bit as if he is having some pain there." P Ex 3 at 158. During this hospital visit M.L. had another episode of staring and facial and neck tensing that lasted about ten seconds and was followed with vomiting. P Ex 3 at 159. M.L. had an EEG performed on April 18, 2005 which was complicated by mechanical artifact and is reported as showing slow waves over the left posterior cortex which were occurring rhythmically. Tr. at 8-9, P Ex 3 at 158. Dr. Eugene Tan, who interpreted the EEG as grossly normal diagnosed possible seizures, but noted that he "could not rule out a reaction to the vaccines." P Ex 3 at 159.

It is richly documented throughout the medical records, and not disputed that M.L. suffered an anaphylactic reaction to his vaccination. Petitioner, in petitioner's Post-Hearing Brief, stated that M.L.'s treating doctors attributed his loss of speech and motor skill problems to the DTaP vaccine and cites to numerous locations in the medical records as support that M.L.'s doctors identified no other explanation for his injuries. P PH Brief at 23. Upon closer review of the citations it is evident that this statement is misleading. See P Ex 3 at 14 (urgent care General Information sheet on seizures, stating most common cause is idiopathic epilepsy, other causes may include head injury, withdrawal from alcohol or other drugs, a high fever, a brain tumor or an infection, or cause unknown); P Ex 3 at 49 (lists possible causes as 1) ? clinical seizures, 2) viral syndrome, 3) vaccine adverse reaction); see P Ex 3 at 48 ("idiopathic vs. Vaccine related?"); P Ex 3 at 59 (possible seizures, possibly related to vaccine versus primary epilepsy); P Ex 4 at 57-58 (assessment included diagnosis of a seizure disorder and language regression, possibility of Landau-Kleffner syndrome); P Ex 4 at 53 (M.L. has fluid behind both ears, causing a conductive hearing loss); P Ex 12 at 8 (doctor acknowledged per parents history M.L. presented to hospital in what appeared to be clinical shock, which would need to be correlated with the medical records). There are numerous records where the doctors question what may be causing M.L.'s seizures and lack of speech. At one point Dr. Kojic's impression was that M.L.'s "developmental delay, repetitive and ritualistic behavior would probably categorize him in autistic spectrum disorder category, more likely pervasive developmental disorder-not otherwise specified." P Ex. 4 at 54. M.L. was referred to Dr. Keeley, a pediatrician specializing in development, whose impression was that M.L. did not have PDD but instead had expressive aphasia with normal receptive vocabulary. P Ex 12 at 1-6 ("[M.L.] is a young man who continues to recover from whatever insult it was that he had.").

It is clear that there is a temporal relationship between M.L.'s vaccinations and his seizure-like activity and lack of speech. While establishing a temporal relationship is a critical prong in proving by the preponderance of the evidence that petitioner's injuries were caused-in-fact by his vaccinations, a temporal relationship alone is not enough to establish causation. The medical records and histories, often per the parents recollection, indicate that M.L. was developing normally prior to April 14, the date of his vaccinations and began to experience complex medical problems after his vaccinations.

There is one notation regarding M.L.'s speech in the medical records prior to vaccination that is worth mentioning. At M.L.'s 15-month visit to his pediatrician under the Developmental Assessment category Speech is circled and notation next to it is **[does not] want to talk**. P Ex 4 at 18. This notation was addressed at the Hearing, Tr. at 31-32, where Dr. Kinsbourne felt this

notation was ambiguous. The undersigned pointed out that it was not so much an issue of if M.L. could not or did not want to talk. Instead the point to take away was that the doctor made a notation regarding M.L.'s lack of speech prior to vaccination. Tr. at 32.

Addressing Dr. Kinsbourne's Reliance on the Mother's Narrative

Petitioner attempts to address the issue of Dr. Kinsbourne relying on the mother's narrative by stating in petitioner's Post-Hearing brief, the following:

In his Order of July 24, 2008, the Chief Special Master asked the parties to indicate in their briefs "whether additional proceedings are necessary" or whether they believe "the record is complete." Order at 1. At the hearing, Dr. Kinsbourne testified that he bases his opinions not on the parents' statements, but on the medical records alone. Tr. 90. In these, circumstances, the petitioner does not believe additional proceedings are necessary and believes the record is complete.

P PH Brief at 19 fn12. Petitioner opted not to file the mother's narrative, which Dr. Kinsbourne reviewed in formulating his opinion. The undersigned's review of the transcript at 90 does not support petitioner's statement that Dr. Kinsbourne bases his opinion on the medical records alone. Tr. at 90. Dr. Kinsbourne was questioned by petitioner's counsel as follows:

Q: Dr. Kinsbourne, based upon the medical records that you have seen, and let's disregard what the mother has told you, does that change your opinion that this child had an anaphylactic reaction to a vaccine that he received?

A: Not at all.

Q: And the severity of the symptoms, based on what the mother says, that doesn't change your opinion?

A: No. I don't stipulate to a particular level of severity of clinical manifestation to arrive at my opinion.

Tr. at 90. The undersigned finds it important to note that there is not a dispute as to if M.L. experienced an initial anaphylactic reaction. In fact, Dr. McDonald agrees that M.L. suffered from an initial anaphylactic reaction, the key distinction being that he opines that this reaction clinically resolved. Tr. at 96. The first question does not address this distinction. Thus, the first answer from Dr. Kinsbourne does not elicit anything new. It is not clear how petitioner's counsel extrapolated from Dr. Kinsbourne's second response that Dr. Kinsbourne bases his opinion solely on the medical records. Dr. Kinsbourne appears to say that he did not rely on a certain level of severity of clinical manifestations to come to his opinion, but does not clearly say that he bases his opinion on the medical records alone. Tr. at 90. **The only basis in the medical records that Dr. Kinsbourne appears to rely on in formulating his theory is the existence of M.L.'s speech problems.** Tr. at 12; Tr. at 53 ("My opinion is mainly centered on speech ability at some level which abruptly decreased...").

Symptoms to expect if any of the proposed mechanisms were causing brain damage

What type of symptoms or clinical signs would be expected if any of the general mechanism were occurring at a level where brain damage may occur? A key point deals with distinguishing between an anaphylactic reaction and anaphylactic shock. In this matter Dr. Kinsbourne stated that M.L.'s reaction was not a severe reaction and there was no claim that his reaction was severe. Tr. at 51. Dr. Kinsbourne also said he is "not convinced that [M.L.] was actually in shock." Tr. at 59. Dr. MacDonald opined that M.L. did experience an initial episode of an anaphylactic reaction, and was not in shock "as far as the clinical cardiovascular part of this." Tr. at 97. His testimony indicates that if brain damage existed, persistent anaphylactic shock would be expected to present with various clinical signs. Tr. at 97-100.

When describing symptoms one might expect to see during anaphylaxis Dr. Kinsbourne stated that if the child was grey, limp, and with a hoarse voice these would be manifestations which can arise in anaphylaxis and angioedema. Tr. at 69. Dr. Kinsbourne opined generally that his point about wide-spread inflammation was to show that anaphylaxis and angioedema "have components which potentially could damage parts of the brain." Tr. at 71. He described that he has seen cases where a part of M.L.'s sequence appeared singularly, i.e. anaphylaxis or angiodema, but no case sharing all the elements M.L. experienced. Tr. at 23. Dr. Kinsbourne stated that he found literature enough for his opinion, but not enough to be able to explain the mechanism of everything that transpired. Tr. at 24. Dr. Kinsbourne acknowledged that in a more severe reaction than M.L. experienced one would expect to see continued fever, a broad level of consciousness, and as previously mentioned the possibility of stroke. Tr. at 25, 70-71. Dr. MacDonald detailed that if one were in shock, cardiovascular collapse would occur. Tr. At 97. This would result in hypotension, low blood pressure, and a decline in respiratory status. Tr. at 97. Clinical signs may indicate severe compromise to maintain blood flow and oxygen. Tr. at 98.

Dr. MacDonald opined further that if M.L. truly went into shock and the shock persisted enough to cause brain damage one would expect to see signs through his test results. Tr. at 100. For instance, if encephalopathy occurred one would expect to see significant altered degree of consciousness, coma or semicoma, and development of signs of brain swelling, focal neurological signs and brain stemming. Tr. at 99. Most significant would be evidence of a significantly altered mental status. Tr. at 99.

Interestingly, Dr. Kinsbourne does not opine that M.L. was in anaphylactic shock though his theory relies on a reaction severe enough to cause brain damage. The undersigned highlighted the problems with Dr. Kinsbourne theory:

THE COURT: The problem I am having is that, going back to the questions I asked you much earlier in terms of support for your testimony, you conceded to me that one, you've never seen a case like this, two you cannot find support for a case like this, and three is that you cannot find any medical literature that would support this theory. And then, as Ms. Brodrick is taking you through the factual underpinnings for your theory as it is laid out in your report, it's not there either. So, when you take this all out, quite frankly, it's beginning very quickly to amount to conjecture.

Tr. at 72. The thrust of Dr. Kinsbourne's response is that it is essential that M.L. should not have experienced a severe anaphylaxis, because he is not asserting severe brain damage. Tr. at 72. He continued "But **I think it would be magical if you have a disorder which either kills you**

or necessarily leaves you completely normal. I mean, there always are potentially intermediate states between being dead and walking away.” Tr. at 73 (emphasis added). Dr. Kinsbourne’s response does not adequately address the undersigned’s concerns. It seems clear that M.L. did experience symptoms during an anaphylactic reaction. This reaction resolved clinically and M.L. was discharged in good condition from the hospital on April 16, 2005. The remaining issue is Dr. Kinsbourne’s reliance on inflammation or anoxia as the bridge to get from the initial reaction to the resulting injuries. Tr. at 73. The undersigned pointed out, “if you don’t have support for that [inflammation or anoxia], where is the bridge?” Tr. at 73. Dr. Kinsbourne candidly stated that he doesn’t know the mechanism by which this happened. Tr. at 73. Further, and more importantly, he testified that he cannot show in this child that there was inflammation or that the blood flow was limited causing anoxia in the place where the damage was done. Tr. at 73. In addressing whether the medical records support widespread inflammation or anoxia he replied:

DR. KINSBOURNE: I absolutely agree that **the medical records give me no specific test results or examination results that would support either of those events** at the time he was at the hospital.

Tr. at 79 (emphasis added). It became clear throughout the undersigned’s questioning of Dr. Kinsbourne that, in the absence of several key factors not supported in the medical records, the one reason Dr. Kinsbourne pointed to the vaccination as the cause of M.L.’s injuries is “the fact that the situation [] until the vaccination, [was that] the child was talking at an age-appropriate level, and after the vaccination, his expressive language was found to be considerably behind.” Tr. at 77-78. The Federal Circuit has explicitly rejected this formulation of evidence. In Moberly, the Federal Circuit characterized the essence of the evidence before the special master as follows:

[The vaccine] was healthy before she received her second DPT vaccination; she suffered seizures within 36 hours of receiving the vaccine; DPT is capable of causing seizures and permanent brain damage; and no alternative cause of her condition has been identified.

Moberly, 592 F.3d at 1323. The Circuit affirmed the special master’s denial of compensation based upon the insufficiency of this proof, noting, as did the special master, that the “problem with that evidence is that it amounts at most to a showing of temporal association between a vaccination and a seizure, together with the absence of any other identified cause for the ultimate neurological injury.” Id. That formulation of proof proffered by petitioner in this cases – a literal temporal relationship, plausible connection between vaccines and injury, and absence of other causes – is the same considered and rejected in Moberly, and accordingly must likewise be rejected by the undersigned.

Dr. Kinsbourne’s Reliance on M.L.’s Speech Problems in Support of his Theory

Even still Dr. Kinsbourne relies on M.L.’s speech issues as evidence of a vaccine related injury. He relies heavily on M.L.’s abnormal EEGs as support for focal brain damage. P Ex 20 at 2. Dr. MacDonald, while noting that the EEGs were mildly abnormal, stated that if shock was persistent enough to cause permanent brain damage an EEG results would typically be diffusely and grossly abnormal, which is not present in this case. Tr. at 100. The undersigned notes that Dr. MacDonald has far more experience reading EEGs than Dr. Kinsbourne. Tr. at 95 (Dr.

MacDonald presently reads pediatric EEGs 3-4 days a week); Tr. at 28 (Dr. Kinsbourne stated that he hasn't "been concerned with EEGs since 1980"). Additionally, petitioner attempts to link M.L.'s abnormal EEGs with his seizure-like episodes. Tr. at 14. Whether there is a link or not is not wholly clear. Worthy of notice Dr. Kojic documented that the typical spells of right hand fisting, teeth grinding and shivering-like episodes of the mouth when captured on the long-term EEG did not have electrographic seizure correlates. P Ex 4 at 53-54. There were also occasions where the EEGs did not show any epileptic discharges to indicate M.L. was in-fact experiencing epileptic seizures. Tr. at 100-1. Another critical point is Dr. Kinsbourne and Dr. MacDonald's differing viewpoint on the type of brain damage that would result in a child being unable to speak. Dr. Kinsbourne stated that M.L.'s "expressive aphasia" is consistent with left hemisphere damage of his brain. Tr. at 13. Dr. MacDonald disagrees, stating that aphasia presents in two primary forms. Tr. at 108. The first form is called Broca aphasia, which is more towards the front of the brain and presents with the patient not talking and decreased speech, but normal understanding. Tr. at 108. The other form is Wernicke's aphasia, which is more towards the posterior part of the brain and the patient talks endlessly, but is unable to understand what is going on. Tr. at 108. Dr. MacDonald notes **that even if the EEGs were accepted as insightful, Broca's aphasia is an anterior abnormality and in this case M.L.'s EEGs intermittent sharp-like wave and rhythmic slow wave are presented in the left posterior hemisphere region.** Tr. at 108-09; P Ex 3 at 32. Dr. MacDonald clearly states that **"even the EEG abnormalities, if it's real and if it is significant, it doesn't really speak to an acquired aphasia at all."** Tr. at 109; see also Tr. at 110 ("it's not really in the right location"); Tr. at 111 ("it's not in the right area"). But see Tr. at 111 (Dr. McDonald acknowledges that the November 26, 2005 EEG is not "wholly consistent with the first two" and he accepts that there "might have been something there, but it's very non-specific"). The undersigned found Dr. MacDonald's testimony discussing how the EEG results do not support petitioner's theory very persuasive.

Dr. MacDonald also suggested that M.L.'s documented ear fluid, that was noted to be causing conductive hearing loss, may have attributed to his speech issues. Tr. at 118; P Ex 4 at 53 (M.L. has fluid behind both ears, causing a conductive hearing loss). He also proposed that M.L.'s anti-convulsant medication may be a factor contributing to M.L.'s issues. Tr. at 118. In addressing M.L.'s behavioral issues, he stated that it is hard not to think the drugs were playing a role. Tr. at 120. Dr. MacDonald opined that there has been a lot of trouble with Keppra, one of the medications M.L. was on. Tr. at 120. Dr. MacDonald has had older children who have hallucinated on Keppra. Tr. at 120. Further he opined Tegretol can make certain seizures worse. These medications, including Depakote can cause strange behaviors. Tr. at 120.

Even if the undersigned accepted petitioner's theory as plausible the above issues cast further doubt on petitioner's theory. Given the lack of a plausible medical theory that demonstrates a logical sequence of cause and effect, defining and determining the extent of M.L.'s problems that presented after his anaphylactic reaction clinically resolved is not necessary for purposes of this decision. M.L. has a complex medical history and fortunately appears to be making steady improvements in his development. Tr. at 82-83.

Medical Expert Credibility

The undersigned finds it necessary to address the credibility of petitioner's expert. Upon further review of this case the undersigned found Dr. Kinsbourne's testimony suffered from extreme deficits. The testimony in this case was as poor as any the undersigned has experienced

in twenty years. Dr. Kinsbourne's testimony highlighted his willingness to testify in a case where he proffered a theory that he conceded he has never seen before, he went to the literature and could not find specific support for, and he did not completely understand. Tr. at 23-25. Further, in applying his theory to the facts of this case there is no support in the medical records that the events transpired as he proposed.

It was established during the Hearing that Dr. Kinsbourne relied on the mother's unfiled narrative in formulating his opinion. At this time petitioner's counsel took responsibility for not filing the mother's narrative into the record. Tr. at 42-43 ("And I'll take responsibility for that."). Petitioner's counsel stated that Dr. Kinsbourne's reliance on the mother's narrative, which petitioner's counsel chose not to file, should not go to his credibility. Tr. at 42. While the undersigned does not blame Dr. Kinsbourne for petitioner's counsel's decision not to file the mother's narrative that does not discharge him of his obligation as a medical expert to provide reliable support for his medical opinion. An expert should not blindly accept the statements of parents, but must reconcile such statements with the medical records. In this matter, Dr. Kinsbourne stated that the mother's narrative did not conflict with his read of the medical records. Yet from the beginning it became clear that Dr. Kinsbourne failed to look at the medical records in at least one occasion. Dr. Kinsbourne relied on the mother's statement that M.L. suffered an anaphylactic reaction within four hours of vaccination, thus within the time frame of a Table Injury. The medical records clearly state otherwise and Dr. Kinsbourne himself acknowledged that he should have considered various notations regarding the timing of M.L.'s vaccination. Tr. at 6, 15-18, 44. This lack of review of the records, in conjunction with the many other deficiencies in Dr. Kinsbourne's theory seriously undermines his credibility. There exists a serious question as to whether Dr. Kinsbourne should be paid for his efforts.⁷

Petitioner's theory must meet the three prongs of laid out in Althen. Althen at 1278. Dr. Kinsbourne attempts to lay out a medical theory causally connecting the vaccination to the injury. Unfortunately for petitioner this theory fails to pass any reasonable test of reliability. Dr. Kinsbourne has testified as a medical expert in the Program for many years with varying degrees of success. In this case, Dr. Kinsbourne speculates to a theory which addresses the unique sequence of events in M.L.'s case in an effort to try to fill in the causative blanks and link M.L.'s injuries to his vaccination. Dr. Kinsbourne himself, is unable to specifically explain or opine to any of the three mechanisms he proposes as causing M.L.'s brain damage. This is not reliable testimony. Further, the medical records do not show clinical signs of any of the three mechanisms taking place. Thus, petitioner has failed to establish a "medical theory casually connecting the vaccine and the injury," nor has petitioner demonstrated by a preponderance of the evidence that M.L.'s vaccinations were the "but for" cause of his injuries as required by the Federal Circuit. Althen at 1278, Capizzano III at 1352, Pafford, 451 F.3d at 1355; see also Moberly, 592 F.3d at 1324.

This concludes the opinion as contained in the draft sent to the parties. At this point, the undersigned will address several points raised by petitioner in her Closing Brief filed November 23, 2011.

Miscellaneous Arguments

Petitioner's Closing Brief is a combination of "facts" provided by the mother in her affidavit and unsigned narrative, snippets of Dr. Kinsbourne's testimony and the unqualified

⁷ Dr. Kinsbourne was paid for his services as part of the interim fees payment.

medical interpretation of this information by counsel. What the brief ignores is the decisive point that Dr. Kinsbourne's medical theory is nothing more than a theory – it is untested, he does not know if it is medically accepted, there is no supportive literature that he is aware of and he has never seen such a medical sequence in his experience. Tr. at 22-23. In short, it is unreliable. In addition, while first testifying that the damage in this case was caused by a diminution in oxygen to M.L.'s brain and an ongoing inflammatory process, he later conceded that other than the mother's statements there is no evidence in this record of hypoxia or inflammation. Id. at 67-9. The unreliability of Dr. Kinsbourne's testimony can be seen through this issue; he actually testified earlier that to the diminution in blood flow. Thus, he stated "there can be a diminution of blood flow, oxygenation, which particularly affects a focal area in the brain rather than the whole brain. . . ." Id. at 11. He stated emphatically that "I believe that occurred in this case." Id. However, later in his testimony, Dr. Kinsbourne testified that he was not testifying to anoxia actually occurring in this case. Tr. at 69 (Responding to whether there is evidence of anoxia, Dr. Kinsbourne responded "Only as a reasonable mechanism. But I certainly couldn't in this case opine that that occurred.")

However, counsel argues that the "duration and severity" of Mathew's anaphylactic reaction were sufficient to produce neurological sequelae. P Closing Brief at 19. Petitioner's supporting narrative relies upon untested information from the mother and counsel's, not the experts', interpretation of the medical records. See generally Id. at 19-21. Counsel concludes with a review of the medical records for clinical evidence of hypoxia and inflammation. Id. at 21-22. These are the mechanisms of harm that Dr. Kinsbourne postulated. Tr. at 71. The problem with counsel's argument is just that, it is counsel's argument. No doctor supported that proposition. Dr. Kinsbourne could not have been clearer in stating that there were no signs of inflammation. Id. at 67. Regarding anoxia, the undersigned asked if there was evidence of anoxia and Dr. Kinsbourne responded "only as a reasonable medical mechanism. But I certainly couldn't in this case opine that that occurred." Id. at 69; see also Id. at 74 ("I cannot show in this child that there was inflammation in the place where the damage was done, and I cannot show that the blood flow was limited causing anoxia in the place in which it was done.") Dr. McDonald saw no evidence of inflammation or anoxia. Id. at 98, 128-29. Petitioner cites to no treating doctor supporting the assertion of inflammation or anoxia. What we are left with is lawyer argument, which is not evidence.

Finally, counsel attempts to build a case for vaccine causation based upon M.L.'s seizures and speech issues. Petitioner's Closing Brief at 24. In doing so, counsel is ignoring or distancing herself from her expert's testimony. Counsel focuses on the initial vaccine reaction, which the records and both experts agree resolved, and assumes that anything that occurred following is due to that reaction. The problem with that reasoning is that it ignores the medical theory expounded in this case that there was a two phase reaction, it ignores the medical theory of harm and the complete absence of any proof of that theory, that is the inflammation and anoxia, and it ignores the fact that no treating doctor supported such an occurrence. In making her argument, petitioner focuses her attention on what respondent's expert, Dr. MacDonald was unable to disprove. However, it is petitioner's burden to establish causation, and it is petitioner's expert, Dr. Kinsbourne, who was unable to support the proposed medical theory of harm. In fact, he clearly and emphatically stated that there was no evidence to support his theory of how the brain would be damaged from the anaphylactic reaction – that is, there was no evidence of inflammation or anoxia. As the Federal Circuit has made clear, the proposed theory of causation must be shown to be "at work" in the case presented. Moberly v. Sec'y of Dept. of Health & Human Servs., 592 F.3d 1315, 1324 (Fed. Cir. 2005); see also Broekelschen v. Sec'y of the

Dept. of Health & Human Servs., 618 F.3d 1339, 1345 (Fed. Cir. 2010), reh'g en banc denied (Dec. 8, 2010) (“[A] petitioner must provide a reputable medical or scientific explanation that pertains specifically to petitioner’s case.”) As in Moberly, even assuming that Dr. Kinsbourne’s medical theory of two phase anaphylactic reaction is reliable, a fact that Dr. MacDonald hotly disputes, Dr. Kinsbourne admittedly stated that there is no support in this record that the “proposed mechanism was at work” in M.L.’s case. Moberly, 592 F.3d at 1325.

There has been much process and argument devoted to the proper interpretation of M.L.’s EEGs. This has been an unfortunately time-consuming and unproductive effort. Petitioner has focused upon one aspect of the EEGs, the correlation of M.L.’s expressive aphasia with the area of damage shown on the EEG. The undersigned addressed the issue of the EEGs in the draft decision, which is reproduced and affirmed here. What must be noted is that the EEG issue was further evidence of the deficiency in petitioner’s case for causation, it cast further doubt. Supra at 20-21. What petitioner ignores and did not address in her subsequent submissions regarding the EEGs is that Dr. MacDonald noted that the EEGs were mildly abnormal and that “if shock was persistent enough to cause permanent brain damage and EEG results would typically be diffusely and grossly abnormal, which is not present in this case. Supra at 20 (citing Tr. at 100). In addition, Dr. MacDonald questioned whether or not the EEGs were indicative of seizures. Tr. at 100-01; see also P Ex 4 at 53-54. These issues were discussed in the draft opinion and discussed with counsel during status conferences. Counsel indicated that a qualified doctor would be enlisted to address the EEG issues. Instead, counsel submitted a further expert report from Dr. Kinsbourne addressing one, the speech issue, of the several EEG issues. What was left not addressed is the critical issue linked to the medical theory of this case, that is signs on the EEG of diffuse brain damage which would support the permanent brain damage petitioner is alleging. Dr. MacDonald did not see such damage, the treating doctors did not discuss such damage and Dr. Kinsbourne, whose qualifications to read EEGs is questionable, did not testify to such damage, and petitioner did not produce further evidence of such damage. In addition, Dr. Kinsbourne’s medical theory of how the focal damage would be caused was the diminution of blood flow. Tr. at 11. As discussed above, he conceded that there was no evidence to support that lack of blood flow. Id. at 69, 74. Given these findings, it is unnecessary to resolve the continued disagreements regarding the aphasia and associated findings on EEG. Compare P Ex 56; R Ex J. The correlation of the speech issue to the EEGs is a difficult issue, which requires adequate development. The undersigned suggested an evidentiary hearing to air out this issue; petitioner’s counsel opted for closing arguments instead. Given the many deficiencies in petitioner’s causation claim, it makes eminent sense to forego deciding this one issue.

Lastly, petitioner submitted the independent neurological examination of Dr. Ronald Davis. P Ex 64. While it is an examination, Dr. Davis includes a summary opinion that M.L.’s neurological issues were caused by his “profound anaphylactic event.” Id. at 5. He states that the anaphylaxis caused “ischemic changes as a consequence of oxygen deprivation secondary to postinflammatory events” caused by the immunizations. Id. As discussed above, there is no support whatsoever for oxygen deprivation or inflammation. Dr. Davis cites no support. However, he does cite to historical events provided by the family. Id. at 2. The undersigned does not credit this untested familial history. The Federal Circuit has found that an expert’s opinion is only as good as its factual predicate. Perreira v. Sec’y of the Dept. of Health & Human Servs., 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994). Given the deficient factual predicate, Dr. Davis’ opinion must fail.

Petitioner's case has suffered from several deficiencies that petitioner failed to cure despite having the benefit of the undersigned's draft opinion and, over respondent's reasonable objections, several years of time. First and foremost, the factual discrepancies between the medical records and the mother's account were never addressed. This issue was so critical because the mother's statements undergirded the experts' opinions. Despite numerous exhortations, mom was not made available for examination. Petitioner was aware from the draft opinion that Dr. Kinsbourne's testimony was flawed and his qualifications were questioned. Petitioner represented that she would have a qualified neurologist review the case, and a qualified expert would review the EEGs. Neither occurred. Over respondent's repeated reasonable objections, the undersigned continued to give petitioner time to correct these deficiencies. In the end, petitioner offered counsel's interpretations of the medical records. These interpretations were offered during status conferences, and counsel was informed that they differed from the medical experts and represented argument, not evidence. Yet petitioner rested her case on counsel's interpretations of the records. Simply stated, petitioner's case did not change appreciably in the four plus years following the September 14, 2007, Hearing in this matter. As such, the undersigned's findings represented in the draft decision did not change. Thus, the draft decision now becomes final.

Accordingly, the undersigned finds that petitioner has not established by a preponderance of the evidence that M.L.'s April 14, 2005 vaccinations were the legal cause of his injuries. Petitioner's claim is **denied**. The Clerk shall enter judgment accordingly.

IT IS SO ORDERED.

s/ Gary J. Golkiewicz
Gary J. Golkiewicz
Special Master