IN THE UNITED STATES COURT OF FEDERAL CLAIMS
OFFICE OF SPECIAL MASTERS
No. 07-81V
Filed: February 28, 2011
To be Published

* *		
Austin Taylor Huffman, by SHANNON CELESTE HUFFMAN, his mother and next friend,	*	
Petitioner,	*	Entitlement; Pertussis; Table Encephalopathy; Trauma
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	
* *		

Michael G. McLaren, Esq., Memphis, TN, for petitioner.
Chrysovalantis P. Kefalas, Esq., U.S. Dept. of Justice, Washington, DC, for respondent.

DECISION[1]

VOWELL, Special Master:

On February 1, 2007, Ms. Shannon Huffman ["petitioner" or "Ms. Huffman"][2] filed a petition for compensation under the National Vaccine Injury Compensation Program,

[1]Because this decision contains a reasoned explanation for the action in this case, I intend to post this decision on the United States Court of Federal Claims' website, in accordance with the E-Government Act of 2002, Pub. L. No. 107-347, § 205, 116 Stat. 2899, 2913 (codified as amended at 44 U.S.C. § 3501 note (2006)). In accordance with Vaccine Rule 18(b), petitioner has 14 days to identify and move to delete medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, I agree that the identified material fits within this definition, I will delete such material from public access.

[2] Although Mr. Chris Huffman and Ms. Huffman were married at the time the petition was filed, only Ms. Huffman filed the petition on Austin's behalf. By the time of the initial fact hearing, the Huffmans had separated and a divorce action was pending. *See* Transcript of April 16, 2009 hearing ["Tr.1"] at 6-7, 59, 73.

42 U.S.C. § 300aa-10, *et seq*.[3] [the "Vaccine Act" or "Program"], on behalf of her minor son Austin Huffman ["Austin"]. The petition alleged that Austin's neurological devastation[4] was caused by his four-month vaccinations. Because Austin received a pertussis-containing vaccine within the 72 hours prior to his cardiopulmonary collapse and resultant coma, vaccine causation is, at least initially, presumed in this case. Notwithstanding this presumption in petitioner's favor, I deny her claim because I find by preponderant evidence that Austin's coma was caused by trauma.

In order to prevail under the Program, a petitioner must prove either a "Table" injury or that a vaccine listed on the Table was the cause in fact of an injury. A "Table" injury is an injury listed on the Vaccine Injury Table, 42 C.F.R. § 100.3, corresponding to the vaccine received within the time frame specified. One of the conditions specified on the Table for compensation is an "encephalopathy" [5] within 0-72 hours after receipt of a

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[3] National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755. Hereinafter, for ease of citation, all "§" references to the Vaccine Act will be to the pertinent subparagraph of 42 U.S.C. § 300aa (2006).

[4] Austin is severely developmentally delayed. Ms. Huffman testified that he is unable to hold his head up, sit, roll over, or walk. He has little ability to control movement of his extremities. He is not toilet trained. He receives all his nutrition through a gastrostomy tube. He cannot speak, and he is minimally communicative through crying and vocalizing vowel sounds. He is legally blind. Austin receives a variety of medications daily, including several for seizure management. Tr.1 at 7-11, 14, 16-19, 22.

[5] The Vaccine Injury Table must be interpreted by reference to the Table's Qualifications and Aids to Interpretation ["QAI"] definition of key terms. *Terran v. Sec'y, HHS*, 195 F.3d 1302, 1307 (Fed. Cir. 1999). The QAI define "encephalopathy" as follows:

(2) Encephalopathy. For purposes of [the Vaccine Injury Table], a vaccine recipient shall be considered to have suffered an encephalopathy only if such recipient manifests, within the applicable period, an injury meeting the description below of an acute encephalopathy, and then a chronic encephalopathy persists in such person for more than 6 months beyond the date of vaccination.

(i) An acute encephalopathy is one that is sufficiently severe so as to require hospitalization (whether or not hospitalization occurred).

(A) For children less than 18 months of age who present without an associated seizure event, an acute encephalopathy is indicated by a significantly decreased level of consciousness lasting for at least 24 hours. . . .

(D) A "significantly decreased level of consciousness" is indicated by the presence of at least one of the following clinical signs for at least 24 hours or greater (*see* paragraphs (b)(2)(i)(A) and (b)(2)(i)(B) of this section for applicable timeframes):

(1) Decreased or absent response to environment (responds, if at all, only to loud voice or painful stimuli);

(2) Decreased or absent eye contact (does not fix gaze upon family members or other individuals); or

pertussis-containing vaccine.[6] Austin experienced a coma on June 15, 2004, approximately 25 hours after receiving a diphtheria, tetanus, and acellular pertussis ["DTaP"] vaccination. This coma met the Table's definition of "encephalopathy." However, the same section of the Vaccine Injury Table that defines encephalopathy includes a caveat:

> An encephalopathy shall not be considered to be a condition set forth in the Table if in a proceeding on a petition, it is shown by a preponderance of the evidence that the encephalopathy was caused by an infection, a toxin, a metabolic disturbance, a structural lesion, a genetic disorder or trauma (without regard to whether the cause of the infection, toxin, trauma, metabolic disturbance, structural lesion or genetic disorder is known). If at the time a decision is made on a petition filed under section 2111(b) of the Act for a vaccine-related injury or death, it is not possible to determine the cause by a preponderance of the evidence of an encephalopathy, the encephalopathy shall be considered to be a condition set forth in the Table.

42 C.F.R. § 100.3(b)(2)(iii).

As I discuss at greater length in Section V below, this case is more complicated than most Table cases, because the Vaccine Act and the Vaccine Injury Table's QAI are less than clear about the allocation of the burdens of production and persuasion in Table encephalopathy cases. Nevertheless, after considering the record as a whole,[7] I hold that petitioner has failed to establish Austin's entitlement to compensation. Respondent has established by preponderant evidence[8] that Austin's injuries were

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(3) Inconsistent or absent responses to external stimuli (does not recognize familiar people or things).

42 C.F.R. § 100.3(b)(2) (2009).

[6] Petitioner initially claimed a Table injury caused by all of the vaccinations Austin received on June 14, 2004, as well Austin's February 7, 2004 hepatitis B vaccination. Petition ["Pet."] at ¶¶ 25-29. However, of the vaccines received on those dates, only pertussis-containing vaccines have encephalopathy as an associated Table injury. **Her claim for compensation based on a Table injury for vaccines other than DTaP is therefore summarily denied**.

[7] *See* § 300aa–13(a) ("Compensation shall be awarded...if the special master or court finds on the record as a whole–(A) that the petitioner has demonstrated by a preponderance of the evidence the matters required in the petition by section 300aa-11(c)(1)."). *See also* § 300aa–13(b)(1) (indicating that the court or special master shall consider the entire record in determining if petitioner is entitled to compensation).

[8] As respondent acknowledges in her Post Hearing Brief, [Res. Post-Hearing Br."] at n.1, her exhibits were erroneously labeled such that there are duplicate exhibits A and B. The report of Dr. Wiznitzer and his curriculum vitae were originally designated as Respondent's Exhibit ["Res. Ex."] A and B, respectively. Because of the confusion, I will instead refer to them by their descriptions and filing dates, not as Res. Exs. A and B. The second set of Res. Exs. A and B are the records of the criminal proceedings against Mr. Huffman. Respondent filed these documents as .pdf files, breaking Res. Ex. B into two separate files.

caused by trauma; petitioner's attempts to undercut the nonaccidental trauma ["NAT"][9] diagnosis made by Austin's treating physicians were unsuccessful.

I. Procedural History.

Ms. Huffman filed her petition *pro se*,[10] alleging both a cause in fact claim (Pet., Part I) and a Table injury claim (Pet., Part II). Medical records and causation opinions from several physicians accompanied the petition.[11] These medical opinions attributed Austin's condition to an encephalopathy resulting from the vaccinations Austin received on June 14, 2004 and the hepatitis B vaccination he received at birth.

Ms. Huffman later secured representation, resulting in a shift in the focus of her case from vaccine causation of Austin's injuries (the cause in fact claim) to reliance on the Table encephalopathy claim.[12] With this shift in focus, petitioner filed an additional expert report, that of Dr. Uscinski,[13] challenging the NAT diagnosis. Thereafter,

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Unfortunately, this Res. Ex. A contains no page numbers. I will cite to Res. Ex. A using the page number of the .pdf file. Res. Ex. B is paginated, but the pagination begins over again in the second file, rendering those page numbers useless. I will cite Res. Ex. B, then, as "Res. Ex. B, Part 1" and "Res Ex. B, Part 2," and will cite to the page number of the .pdf file, not the provided page number.

[9] Nonaccidental trauma ["NAT"] is one of the preferred terms for what has been called shaken baby syndrome ["SBS"], nonaccidental traumatic brain injury, abusive head trauma ["AHT"], acquired traumatic brain injury ["ATBI"], acquired brain injury ["ABI"], or inflicted injury. Transcript of June 24, 2009 hearing ["Tr.2"] at 58,152; Transcript of May 11, 2010 hearing [Tr.3"] at 35, 168-69. *See also* C. Christian, et al., *Abusive Head Trauma in Infants and Children*, PEDIATR. 123(5): 1409-1411, 1409 (2009), filed as Res. Ex. EE (recommending the use of a term "inclusive of all mechanisms of injury, including shaking" and suggesting the use of AHT).

[10] It appears from some of the filed medical records that Ms. Huffman had assistance from a law firm, although no member of this firm ever entered an appearance on her behalf. *See, e.g.*, Petitioner's Exhibit ["Pet. Ex."] 5, p.1 (reflecting that the medical records were requested by an attorney who frequently appears on behalf of Vaccine Act petitioners).

[11] These physicians included Drs. Harold Buttram (*see* Pet. Ex. 9); Edward Yazbak (*see* Pet. Ex. 11); Horace Gardner (*see* Pet. Ex. 13); Michael Innis (*see* Pet. Ex. 10); and Patrick Barnes (*see* Pet. Ex. 12). In her post-hearing brief, filed July 23, 2010 ["Pet. Post-Hearing Br."], petitioner relied only on the reports provided by Drs. Gardner and Barnes, in addition to the later filed report of Dr. Ronald Uscinski, the expert who testified on her behalf.

[12] The petition was never amended to withdraw the cause in fact claim. Although petitioner's pre-hearing memorandum, filed January 14, 2010 ["Pet. Pre-Hearing Memo."], at 4-5, indicated that petitioner was proceeding on a cause in fact claim, at the May 11, 2010 hearing, petitioner's counsel clarified that this was an error, and that petitioner was only proceeding on the Table injury claim. *See* Tr.3 at 5. *See also* Pet. Post-Hearing Br. (arguing only a Table injury claim).

[13] Doctor Uscinski is a board-certified neurosurgeon. Tr.3 at 232, 314; curriculum vitae ["CV"] of Dr. Ronald Uscinski, Pet. Ex. 55, at 2. He has neurosurgical privileges at several Washington, DC area hospitals, including Georgetown, where he also serves as a non-tenure track faculty member. His office is located in Chevy Chase, MD. Tr.3 at 236-37. In the past 10-13 years, he has testified approximately

respondent filed expert reports invoking the trauma exception to the Table presumption of causation and explaining how trauma was, in fact, responsible for Austin's injuries.

Three hearings were necessary to accommodate the witnesses. I heard petitioner's testimony and met Austin in a hearing conducted in Atlanta, GA, on April 16, 2009. I heard the testimony of Austin's father, Mr. Chris Huffman; two of Austin's treating physicians, Drs. Robert Rust and Douglas Willson; and the police officer who investigated Austin's injuries, Investigator Jim Fetterman, in Charlottesville, VA, on June 24, 2009. Finally, I heard the testimony of non-treating expert witnesses, Drs. Max Wiznitzer[14] and Robert Reece[15] for respondent and Dr. Ronald Uscinski for petitioner, in Washington, DC, on May 11, 2010. Because these hearing transcripts are separately paginated, I will use "Tr.1" to designate testimony from the initial fact hearing on April 16, 2009, "Tr.2" to refer to the second proceeding involving treating physicians[16] and other fact witnesses on June 24, 2009, and "Tr.3" to refer to the May 11, 2010 expert witness hearing.

Post-hearing briefs were filed on July 22 and 23, 2010. Neither party filed optional post-hearing reply briefs. Thus, this case is ready for resolution of the issue of entitlement.

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160 times about shaken baby syndrome, mostly in the context of criminal cases. In the last few years, he has derived considerable income from such testimony ($91,000 in 2008; and $46,200 in 2007). Tr.3 at 311-313, 318-19.

[14] Doctor Wiznitzer is a board certified pediatrician and neurologist with a special competence in child neurology. CV of Dr. Max Wiznitzer, filed Oct. 25, 2007, at 5. He is primarily a clinician, seeing pediatric patients at Rainbow Babies & Children's Hospital in Cleveland, Ohio. Tr.3 at 12-13. As a part of his training in pediatric neurology, he examined the neurological consequences of child abuse, and has treated patients with abusive head trauma. Tr.3 at 14-15. He has authored numerous medical journal articles, and sits on the editorial board of several top-ranked neurological journals, in addition to serving as a peer reviewer. Tr.3 at 8-9, 11-12. Additionally, Dr. Wiznitzer teaches medical students about how to identify child abuse. Tr.3 at 15-16. Although he has not appeared as an expert witness in the Vaccine Program on behalf of petitioners, Dr. Wiznitzer has opined as a reviewer for HHS in favor of petitioners, particularly in cases involving Table encephalopathy without evidence of alternate cause. Tr.3 at 18-19.

[15] Doctor Reece is a board certified pediatrician at the Floating Hospital for Children at Tufts Medical Center in Boston, MA. CV of Dr. Robert Reece, Res. Ex. M, at 1; Tr.3 at 160-61. He specializes in identifying cases of child abuse, serving as medical director of the Child Protection Program at Tufts. Tr.3 at 161. He has diagnosed "close to 10,000" cases of child abuse in his career. Tr.3 at 163. His practice includes teaching medical students how to diagnose child abuse. He is the recipient of awards for his work and has published peer-edited journal articles and book chapters on the topic of child abuse. He also edits textbooks and a journal that aggregates articles about child abuse from non-pediatric journals. Tr. 165-66. Although he serves on the board for the National Shaken Baby Center, he receives no compensation for this position. Tr.3 at 214.

[16] Although not a treating physician, Dr. Uscinski did observe Austin while he was hospitalized at UVA on June 23, 2004, about one week after his arrival there. *See* Pet. Ex. 46 at 10-11. His observations were not, however, the sole basis for his opinion. Tr.3 at 252-53.

II. Medical History.

A. Introduction.

The primary factual dispute in this case involves what happened during the afternoon of June 15, 2004. According to his pediatrician, Austin was a healthy infant on the afternoon of June 14, 2004, when he received his four-month vaccinations. Pet. Ex. 5, p. 7. A little over 25 hours later, at about 4 PM on June 15, 2004, emergency medical services ["EMS"] personnel found Austin cyanotic and apneic in his parents' basement apartment, with his father performing CPR. Pet. Ex. 18, p. 1. Austin was in the sole care of his father, Chris Huffman, from the morning of June 15, 2004 when Ms. Huffman departed for work, until EMS personnel arrived.

Austin was initially transported to Rockingham Memorial Hospital ["RMH"], but his condition necessitated treatment at a pediatric intensive care unit, and he was therefore transferred to the University of Virginia's medical center ["UVA"]. Shortly after Austin's arrival at UVA at about 7 PM on June 15, 2004, his treating physicians provisionally diagnosed him as the victim of NAT. *See* Pet. Ex. 8, pp. 361, 379. Testing performed that evening and over the next few weeks solidified that diagnosis. *See, e.g.*, Pet. Ex. 8, pp. 149-50, 155-56, 161-63, 175-76, 179-81.

If the diagnosis of NAT is correct, Austin was injured by his father, probably between about 3:20 PM and 3:47 PM on June 15, 2004.[17] If Austin's injuries are the result of NAT, petitioner loses the benefit of the presumption of causation. Because she has abandoned her causation in fact claim (*see* Tr.3 at 5), the loss of the presumption of causation defeats her compensation claim.[18]

Mr. Huffman has consistently denied injuring Austin.[19] His accounts of Austin's activities on the afternoon of June 15, 2004 as set forth below are taken from his reports

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[17] Evidence seized by police from the Huffmans' apartment during a subsequent criminal investigation included a camera. The developed film contained a photo of Austin taken at 3:20 PM on the date of his collapse. In the photograph, Austin is playing in his bath and appears alert and happy. Tr.2 at 12, 15, 51. The first responders' records place the time of the call for assistance to the Harrisonburg, VA, police department at 3:47 PM. Pet. Ex. 18, p. 1.

[18] Even if the causation in fact claim had not been abandoned, there is no persuasive evidence that vaccines caused Austin's collapse, and Dr. Uscinski, the expert who testified on petitioner's behalf, attributed the bleeding in Austin's brain to prenatal or neonatal trauma, not the vaccines. Tr.3 at 255-56; Pet. Post-Hearing Br. at 17. Although petitioner filed reports from several non-treating physicians, some of whom attributed Austin's collapse to vaccines, petitioner expressly relied on only the Table injury claim. Tr.3 at 5; *see generally* Pet. Post-Hearing Br. (in which vaccine causation was not discussed, except to note, in footnote 4, that it need not be addressed). Even if petitioner had not eschewed reliance on the cause in fact claim, Drs. Rust, Willson, Wiznitzer, and Reece demolished any such claim.

[19] Mr. Huffman reported what transpired on June 14-15 to several individuals, in addition to what he told his wife. He called the police department seeking emergency assistance when Austin stopped breathing on June 15, 2004, and talked with EMS personnel while they were treating Austin. *E.g.,* Res. Ex. J at 17-18. He talked with health care providers at RMH. *E.g.*, Pet. Ex. 5, p. 47. He also spoke with health care

to health care providers, a social worker, and a police investigator within 48 hours of Austin's collapse. The evidence undercutting his denials is medical and circumstantial, and is addressed in Section III below.

Although less central to the issue of causation, there are also disputes concerning Austin's health and behavior after he received his four-month vaccinations. Ms. Huffman provided accounts of what happened from the evening of June 14 through the morning of June 15, 2004 to a number of individuals, as well as providing later statements, affidavits, and testimony.[20] There are conflicts between her earlier accounts (those made to health care providers and others on June 15-16, 2004) and those made after June 16, 2004. These later statements were made at a time when Ms. Huffman began investigating the role Austin's four-month vaccines may have played in causing his collapse on June 15, 2004.[21]

B. Law Governing Resolution of Factual Disputes.

Conflicts between contemporaneous records and testimony given several years later at a hearing are common in Vaccine Act cases, and this case is no exception. Two general legal principles guide the resolution of conflicts between contemporaneous records and later-adduced evidence. The first is that the absence of a reference to specific symptoms in a medical record does not conclusively establish the absence of symptoms during that time frame. *See, e.g.*, *Murphy v. Sec'y, HHS*, 23 Cl. Ct. 726, 733

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providers at the UVA hospital. *E.g.,* Pet. Ex. 8, p. 377. He was interviewed by social worker Erin Sours and Investigator Fetterman on June 16, 2004 at 4:05 PM while at the UVA hospital. *See* Res. Ex. E at 10. Investigator Fetterman testified at least twice concerning what Mr. Huffman told him at this interview. The first testimony was at a preliminary hearing in Harrisonburg, VA, on a criminal complaint for the aggravated malicious wounding of Austin filed against Mr. Huffman. The transcript of this hearing appears in Res. Ex. B, filed June 27, 2007. As indicated above, Investigator Fetterman also testified at the second hearing in this Vaccine Act proceeding. Additionally, Investigator Fetterman's handwritten notes from this interview were filed as Res. Ex. J, and his dictated notes as Res. Ex. E.

[20] The most contemporaneous record is found in the reports made to treating health care providers on June 15-16. *See, e.g.*, Pet. Ex. 8, pp. 377-78. Ms. Huffman also talked with a social worker and Investigator Fetterman on June 16, 2004. Res. Ex. E at 6. According to her testimony, she and her husband also provided accounts to a relative on June 17, 2004. *See* Pet. Ex. 23; Tr.1 at 81-82. Petitioner's Ex. 46 is a handwritten diary of events, beginning on June 15, 2004, but some of the early events were written in a manner that suggests that the accounts were written at a time after the dates on the entries. Petitioner's Ex. 47 is petitioner's initial affidavit, dated March 30, 2007. A second and similar affidavit was filed on March 25, 2009, and was dated March 23, 2009. *See* Pet. Ex. 52. Finally, petitioner testified on April 16, 2009 about what transpired. Although both of her affidavits also described what happened during the day of June 15, 2004, I have attached little weight to those accounts, as Ms. Huffman was not at home when those events transpired. I note that Chris Huffman's testimony was generally consistent with Ms. Huffman's reports of what he told her.

[21] Because she was unwilling to accept the diagnosis of the treating physicians that Austin was the victim of NAT, Ms. Huffman began internet investigations into vaccine causation on June 16, 2004, after talking with Investigator Fetterman. Tr.1 at 111. Portions of her later accounts of what transpired between Austin's June 14, 2004 vaccinations and his cardiac arrest appear to have been shaped by her inquiries.

(1991), *aff'd*, 968 F.2d 1226 (Fed. Cir. 1992) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance." (citation omitted)).

The second principle addresses the degree of reliance commonly accorded to contemporaneous records. Special masters frequently accord more weight to contemporaneously recorded medical symptoms than those recounted in later medical histories, affidavits, or trial testimony. "It has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight." *Murphy*, 23 Cl. Ct. at 733 (citation omitted); *see also Cucuras v. Sec'y, HHS*, 993 F.2d 1525,1528 (Fed. Cir. 1993) (medical records are generally trustworthy evidence). Memories are generally better the closer in time to the occurrence reported and when the motivation for accurate explication of symptoms is more immediate. *Reusser v. Sec'y, HHS*, 28 Fed. Cl. 516, 523 (1993). Inconsistencies between testimony and contemporaneous records may be overcome by "clear, cogent, and consistent testimony" explaining the discrepancies. *Stevens v. Sec'y, HHS*, No. 90-221V, 1990 WL 608693, at *3 (Fed. Cl. Spec. Mstr. Dec. 21, 1990). The facts set forth in this opinion were determined with these legal principles in mind.

These facts were also determined based on the Federal Circuit's mandate that special masters should give weight to the opinions of treating physicians. *See Andreu v. Sec'y, HHS*, 569 F.3d 1367, 1375 (Fed. Cir. 2009) (citing *Capizzano v. Sec'y, HHS*, 440 F.3d 1317, 1326 (Fed. Cir. 2006)). Unlike most Vaccine Act proceedings in which treating physicians' opinions are presented solely through their sometimes ambiguous medical record entries, two of Austin's treating physicians testified. Both were well-qualified as experts in their own right. The evidence regarding their qualifications enhanced the weight I accorded their testimony. One testified twice in Commonwealth of Virginia proceedings concerning the same issues presented in this case.[22] They

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[22] Doctor Willson testified for the Commonwealth at a preliminary hearing (transcript at Res. Ex. A) and at a sentencing hearing (transcript at Res. Ex. B). On February 10, 2005, Chris Huffman entered a plea of guilty in the Circuit Court of Rockingham County, VA, to unlawfully wounding Austin, a lesser offense of the charge of malicious wounding upon which he was indicted. *See* Res. Ex. B, Part 1, at 1 (indictment), 41-45 (plea agreement). His plea was pursuant to an agreement permitting a so-called "*Alford*" plea, referring to the Supreme Court decision in *North Carolina v. Alford*, 400 U.S. 25 (1970). In *Alford*, the Supreme Court ruled that there was no constitutional impediment to a court's acceptance of a guilty plea, even though the defendant continued to protest his innocence, where there was other evidence of guilt. 400 U.S. at 32, 38; *see also* 24 MOORE'S FEDERAL PRACTICE—CRIMINAL PROCEDURE § 611.08 (3rd ed. 1997) (*Alford* plea requires the trial court to find a factual basis for the plea independent of the defendant's plea).

Because Mr. Huffman's plea was based on *Alford*, he was not required to acknowledge responsibility for Austin's injuries, although he acknowledged the substantial likelihood of a conviction, based on the evidence available. *See* Res. Ex. B, Part 1, at 42. The court heard the testimony of Dr. Willson and Dr. Robin Foster to establish a factual basis for the guilty plea and to aid the court in determining an appropriate sentence. The judge sentenced Mr. Huffman to a five year penitentiary term, suspending three years of the sentence. Res. Ex. B, Part 1, at 47.

explained their medical record entries and the bases for their opinions that Austin was the victim of NAT. Unlike many treating physician opinions, their opinions addressed all the factors set forth in *Althen v. Sec'y, HHS*, 418 F.3d 1274, 1278 (Fed. Cir. 2005), establishing: (1) a reliable medical theory; (2) a logical sequence of cause and effect; and (3) a proximate temporal relationship between the cause and the injury.

To challenge the diagnosis of NAT and the bases upon which the diagnosis rested, petitioner also produced expert testimony and medical literature. Respondent produced two experts and medical literature, buttressing the opinions of the treating physicians.

C. Initial Treatment and Testing.

1. Treatment at RMH.

Doctor Steven Turner treated Austin upon his arrival at the RMH emergency department. Pet. Ex. 7, p. 101. On physical examination, Dr. Turner found no evidence of trauma. He ordered two antibiotics for empirical treatment of suspected sepsis,[23] and arranged to have Austin transported to the UVA pediatric intensive care unit ["PICU"]. Pet. Ex. 7, pp. 101-02.

In an addendum to his initial assessment, Dr. Turner noted that a head CT scan[24] "demonstrated findings consistent with diffuse anoxic brain injury, as well as

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Petitioner orally objected to my consideration of Mr. Huffman's plea in a status conference on February 12, 2009, during the discussion concerning a motion to strike assertions regarding Mr. Huffman's plea from the petition. After discussing the basis for this motion, it was apparent to me that neither party understood the nature of an *Alford* plea. *See* Order, filled February 19, 2009. As I indicated to the parties in orally denying the motion, based on my experience as a former prosecutor, defense counsel, and criminal trial and appellate court judge, I fully accept that an innocent individual might plead guilty in this manner to mitigate the consequences attendant upon a trial and the possible conviction of a greater offense. Mr. Huffman alluded to that rationale after entering his guilty plea in telling the court that he did not have the money to produce expert testimony on his behalf. *See* Res. Ex. B, Part 1, at 46; *see also* Tr.1 at 115-17 (testimony of Ms. Huffman regarding the difficulty in obtaining expert testimony on Mr. Huffman's behalf).

As a part of my decision on causation, I have considered the evidence advanced by the Commonwealth of Virginia to establish Mr. Huffman's responsibility for Austin's injuries <u>without considering the guilty plea itself</u>.

[23] The laboratory testing later disclosed that Austin might have had an acute urinary tract infection. *See* Addendum to the Emergency Department Physician's Report, Pet. Ex. 7, p. 105; *but see id*., p. 131 (reflecting no growth in the urine culture). None of the experts considered a urinary tract infection to be a possible cause of Austin's cardiac arrest. *See, e.g.*, Res. Ex. A at 434-35 (testimony of Dr. Willson that a urinary tract infection might conceivably spread to cause meningitis, but would not cause a sudden cessation of breathing and intracranial bleeding).

[24] A CT scan refers to a computed tomography scan of the brain, a test used to diagnose central nervous system disease, including tumors, aneurysms, and hemorrhages. It consists of a computerized analysis

subarachnoid hemorrhage." Pet. Ex. 7, p. 105. There was no evidence of any skull fracture. *See id*., pp. 101-02, 105. In response to the CT findings, Dr. Turner ordered the administration of Dilantin, an anti-seizure medication. *Id*., p. 105. Doctor Turner's records do not reflect a definitive diagnosis.

2. Austin's Condition on Arrival at UVA Medical Center.

Upon his arrival at the UVA hospital on June 15, 2004, Austin was admitted to the PICU. *See* Pet. Ex. 8, p. 377. His initial care and treatment were provided by Drs. Cary Sauer and Douglas Willson. *See* History and Physical Examination form completed by Dr. Sauer, Pet. Ex. 8, pp. 375-76; PICU Attending Note completed by Dr. Willson, *id*., pp. 377-79.

Austin arrived intubated, but with good circulation. Pet. Ex. 8, p. 378; Tr.2 at 149. He had a distended and protruding anterior fontanel.[25] His pupils were small and minimally reactive. He had no doll's eyes or corneal reflexes, and did not respond to painful stimuli. Pet. Ex. 8, p. 378; Res. Ex. A at 417-18; Tr.2 at 150. The only positive neurological signs Austin displayed on arrival were some spontaneous respiratory efforts and some gag reflex. Tr.2 at 150. Austin had no visible burns, bleeding, or rashes. Pet. Ex. 8, p. 378; Tr.2 at 149-50. Based on his condition, Dr. Willson did not expect him to survive. Res. Ex. B, part 2, at 63-64.

Doctors Sauer and Willson made several consultation requests on June 15, 2004. These included requests to ophthalmology to "r/o [rule out] shaken baby," according to the ophthalmology consultant notes (Pet. Ex. 8, p. 361), and to the neurology service (*id*., pp. 362-63). In addition, they ordered an EEG[26] to rule out seizures, scheduled a skeletal survey to look for broken bones, and arranged for a radiologist to read the CT scan taken at RMH. *Id*., p. 376; Res. Exs. A at 418; B, Part 2, at 38.

Austin was minimally responsive during the EEG performed the evening he arrived at UVA. Pet. Ex. 8, p. 510; Res. Exs. A at 418; B, Part 2, at 38. Although there was no evidence of seizure activity on the EEG, the results were abnormal, suggestive of a severe encephalopathy of non-specific etiology. Pet. Ex. 8, p. 510; Res. Exs. A at 418-19; B, Part 2, at 38.

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of x-rays of the brain. MOSBY'S MANUAL OF DIAGNOSTIC AND LABORATORY TESTS (4th ed. 2010) ["MOSBY'S"] at 1080-82.

[25] The fontanel is the soft spot at the top of an infant's head. Tr.2 at 70-71. The spot is soft because the skull bones have not yet grown sufficiently to encase the entire brain, and represents a gap in the bones. *See* DORLAND'S ILLUSTRATED MEDICAL DICTIONARY (31st ed. 2007) ["DORLAND'S"] at 736.

[26] An EEG (electroencephalogram) measures the electrical activity of the brain. *See* Res. Exs. A at 418-19; B, Part 2, at 38.

D. Opinions of Treating Physicians.

Every treating physician who recorded a diagnosis of Austin during his stay at the UVA medical center concurred in the diagnosis of NAT, although there were some differences in their opinions as to the precise mechanism of injury.

1. Treating Physicians Who Did Not Testify.

a. Doctor Cary Sauer.

Doctor Sauer's history and physical examination note, dated at 2200 (10 PM) on June 15, 2004, reflected a differential diagnosis of NAT. Pet. Ex. 8, pp. 375-76. Based on the history taken from the Huffmans, the test results that accompanied Austin from RMH, and his own examination of Austin, Dr. Sauer initiated a child protective service inquiry that same evening. *See* Pet. Ex. 8, p. 351.

b. Doctor Steven Newman.

Doctor Newman, a physician on the UVA ophthalmology service, conducted the eye examination on June 16, 2004. He found diffuse bilateral retinal hemorrhages in Austin's eyes. Pet. Ex. 8, pp. 52, 206-07, 361. He indicated that Austin had definite evidence of optic nerve dysfunction in his left eye, with no obvious increase in intracranial pressure. He attributed the extensive intraretinal hemorrhages he observed to "a shear injury secondary to shaking." *Id*., p. 207.

Doctor Newman re-evaluated Austin's eyes on July 16, 2004. The right eye appeared to be healing, but Austin had a left vitreous hemorrhage, which ultimately required surgical treatment. Pet. Ex. 8, pp. 54; 206-11, 326-30.

2. Testimony by Treating Physicians.[27]

Doctors Willson and Rust both testified as to their diagnoses and treatment of Austin. They relied on the same test results and reports and, in most respects, their observations and assessments were congruent. They agreed that Austin was the victim of NAT, but their opinions diverged to some degree as to the precise mechanism of injury. Doctor Rust opined that Austin's injuries were likely the result of asphyxiation by strangling or smothering, but agreed that shaking caused the subarachnoid bleeding (Tr.2 at 95-97); Dr. Willson opined that Austin's injuries were the result of shaking, or, conceivably, shaking plus an impact (Tr.2 at 167-69; Res. Ex. B, Part 1, at 444-46). I have considered the conflict in their opinions regarding the mechanism of injury in

[27] Transcript references to page numbers in the medical records filed as Pet. Ex. 8 are to the UVA hospital pagination, not Pet. Ex. 8's page numbers. At the time Pet. Ex. 8 was filed, the pages were unnumbered. A paginated copy later replaced the originally filed version of Pet. Ex. 8, but the copies of the hospital records used by the witnesses had only the UVA-assigned page numbers. *See* Tr.3 at 135.

determining how much weight to give their opinions. However, to find that the trauma exception applies in this case, I need not determine a precise mechanism of injury, so long as I find preponderant evidence that trauma caused Austin's encephalopathy. *See* 42 C.F.R. § 100.3(b)(2)(iii).[28]

a. Doctor Douglas Willson.

At the time Austin was admitted to the PICU, Dr. Willson was not only the on-call attending physician, he was the PICU director as well. He held this position for about 17 years.[29] Tr.2 at 145-46. He is board certified in both pediatrics and anesthesiology, and has a subspecialty certification in pediatric critical care.[30] Tr.2 at 147. He treated Austin the night of Austin's arrival before rotating off Austin's case, and then rotated back on the case later that week. Even while not directly responsible for Austin's care, he continued to follow Austin's progress. Tr.2 at 152.

b. Doctor Robert Rust.

Doctor Rust, a physician with a double board certification in pediatrics and neurology and advanced training in neonatal neurology, was the pediatric neurologist on call when Austin was admitted to the UVA medical center.[31] Tr.2 at 53-55, 57. He was Austin's treating neurologist for the first week of Austin's hospitalization. Tr.2 at 57-58.

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[28] That section of the Vaccine Injury Table's QAI provides:

> An encephalopathy shall not be considered to be a condition set forth in the Table if in a proceeding on a petition, it is shown by a preponderance of the evidence that the encephalopathy was caused by an infection, a toxin, a metabolic disturbance, a structural lesion, a genetic disorder or trauma (without regard to whether the cause of the infection, toxin, trauma, metabolic disturbance, structural lesion or genetic disorder is known). (emphasis added).

[29] By the time of the hearing in this case, Dr. Willson had stepped down as the PICU director, but was still actively practicing in the area of pediatric intensive care. Tr.2 at 145.

[30] In addition to his residency training in pediatrics and anesthesiology, Dr. Willson performed a fellowship in pediatric critical care. Res. Ex. A at 415. Doctor Willson also teaches residents and lectures on pediatric care issues at UVA. Tr.2 at 147.

[31] Doctor Rust is a clinician and professor, with an active practice at the UVA medical center and its satellite clinics in rural Virginia. About 95% of his patients are children. Tr.2 at 55. One of his teaching responsibilities involves teaching medical residents how to identify child abuse. He has seen and treated about 40 such cases in the course of his medical career. Tr.2 at 56-57. He has testified before in Vaccine Act cases, twice for petitioners and twice in the Omnibus Autism Proceeding on behalf of respondent. Tr.2 at 87.

3. Diagnosis.

Both specialists diagnosed Austin as suffering from NAT, and testified that they believed with near certainty that NAT was the only possible diagnosis. As Dr. Rust testified:

> The initial history and physical examination suggested a greater than 95 percent likelihood that this was an inflicted injury. And the ensuing evaluation, which has several parts to it, one of which is to exclude alternative diagnoses and another part of it is to actually visualize the images with a variety of modalities, refined that position to my view that there isn't any alternative explanation other than inflicted injury in this case.

Tr.2 at 58. With the additional information obtained from imaging studies, coupled with the exclusion of other possible diagnoses, Dr. Rust said that Austin's case "came as close as I've ever really come to saying this is a 100-percent chance that this is inflicted trauma." Tr.2 at 64. According to Dr. Rust: "[t]he combination of those findings doesn't permit in my view any alternative diagnosis to be considered." Tr.2 at 60.

In addition to his testimony in this case, Dr. Willson twice testified in Virginia court proceedings regarding the cause of Austin's injuries.[32] Doctor Willson also testified before me, but commented that his testimony was drawn primarily from his records because the passage of time had dimmed most of his memories of Austin. Tr.2 at 147. At the two Virginia court proceedings, his testimony appeared to be based on his more recent treatment of Austin, as well as on the medical records. His diagnosis, however, remained the same. He had previously testified that the "constellation of findings" in Austin's case could only be explained by trauma (Res. Ex. A at 424; *see also id*. at 431), and reiterated that testimony in these proceedings (*see* Tr.2 at 168).

Doctor Willson's initial impression on June 15, 2004 was that Austin was the victim of NAT. Tr.2 at 151. All the evidence developed later only solidified his diagnosis. He testified: "This was very clearly nonaccidental trauma. On a scale of one to 10, this was a 9.8 or something like that." Tr.2 at 155.

4. Bases for Diagnosis.

Both Drs. Willson and Rust relied on the same body of information in arriving at their diagnoses of NAT. This data included: (1) reports that Austin was a normal baby

[32] Doctor Willson's testimony appears at Res. Exs. A, at 414-56 (preliminary hearing testimony); B, Part 2, at 34-98 (sentencing hearing testimony).

before his collapse; (2) the CT scan conducted at RMH; (3) the MRI[33] performed on June 16, 2004; (4) subsequent MRI and CT scans of Austin's spine; (5) the ophthalmology examination performed by Dr. Newman; and (6) the probable fracture of Austin's left tibia.

a. Austin's Condition Prior to His Collapse and His Initial Presentation.

Shortly after his arrival at UVA, Austin was examined by staff neurologists, including Dr. Rust. According to Dr. Rust, Austin's physical appearance raised the indicia of suspicion for NAT as a cause. Tr.2 at 58-59. Doctor Rust was influenced by the same findings on examination that Dr. Willson also observed, but attributed the findings to specific areas of brain injury. Austin had a very full fontanel, indicative of increased intracranial pressure. Tr.2 at 59, 70-71; Res. Ex. V. He had "[u]pgaze paresis with lid elevation" (Tr.2 at 71), meaning that his eyes did not fully elevate in response to a stimulus, but that the eyelids did fully elevate. Tr.2 at 61, 71; Res. Ex. V. This is sometimes called "Collier's sign," and signifies a brainstem dysfunction, particularly in the midbrain. Tr.2 at 71.

Austin had markedly increased tone bilaterally, which indicated a central nervous system motor dysfunction. Tr.2 at 72; Res. Ex. V. He also had increased respiratory rate and increased blood pressure, which would be consistent with the increase in intracranial pressure. The presence of all three of these symptoms indicated that Austin's body was attempting to get blood throughout the brain, despite the swelling and hemorrhage. His increased heart rate suggested that he was in pain. Tr.2 at 72; Res. Ex. V.

To Dr. Rust, these findings were indicative of a severe encephalopathy, but there was no evidence of any underlying neurological dysfunction, seizures, or infection as a possible cause. Tr.2 at 58-59. Austin apparently had no clotting disorder, given that he had been circumcised without excessive bleeding. Tr.2 at 60.

Doctor Willson's initial impression was that Austin was the victim of NAT, based on his physical examination, review of the CT scan from RMH, and his interview of Austin's parents. Tr.2 at 151-52. According to Dr. Willson, the account Mr. Huffman provided (that Austin was fine when he put him in the playpen, only to find him blue and not breathing a few minutes later) was not biologically plausible because an infant could not turn blue that quickly, and Mr. Huffman's account did not provide any explanation for Austin's injuries. *See* Tr.2 at 151-52; Res. Ex. A at 424.

[33] "MRI" stands for magnetic resonance imaging. Doctor Willson described an MRI as a "more sophisticated way of imaging [Austin's] brain" than that of the CT scan. Res. Ex. A at 419-20; *see also id*. at 422; Tr.2 at 152.

b. The Initial CT Scan.

Although both Drs. Willson and Rust relied upon the RMH CT scan, it played a more prominent role in Dr. Willson's opinion.

Doctor Willson observed that the CT scan provided him with information regarding the mechanism of injury and allowed him to date when the injury occurred. He explained that, because babies' heads are heavy as compared to the rest of their bodies and their necks lack musculature, the bridging veins on the surface of the brain (below the pia and arachnoid membranes that cover the brain) can be torn by the force exerted in shaking and impact. Tr.2 at 168-69; *see also* Res. Ex. A at 425; Tr.2 at 95 (Dr. Rust's testimony attributing subdural hemorrhage to tearing of the bridging veins due to shaking**).** The bleeding he observed on the RMH CT scan was, in his opinion, caused by shaking.[34] Tr.2 at 151-52.

The CT scan allowed him to date the time the bleeding began, because on a CT scan, new blood, which is dense, blocks more of the x-rays and therefore shows up as very bright on the scan.[35] The brightness decreases as the blood breaks down, and bright blood on a CT scan is certainly from bleeding within the last 24 hours. Tr.2 at 162-63; Res. Ex. A at 422, 441. Although his dictated notes indicated that "some" of the blood appeared to be bright, consistent with new bleeding, he testified that he did not mean to imply by the use of the word "some" that some of the bleeding was older. Tr.2 at 162-63; *see also* Res. Ex. A at 437-39 (consistent testimony explaining his dictated notes).

In addition to the CT scan evidence, other evidence also suggested that the bleeding began shortly before Austin's collapse. Doctor Willson explained that active bleeding in the brain that began earlier would have made Austin very irritable and fussy and Austin appeared normal to his pediatrician a day earlier. Res. Ex. A at 440. The damage seen on the CT (and, later, on the MRI) suggests that Austin lost consciousness immediately after being shaken, but it could have taken from minutes to an hour for him to lose consciousness. Res. Ex. A at 426. The event that caused the bleeding could not have occurred weeks or days earlier. The initial reports of Austin's parents to Dr. Willson and other treating physicians indicated that Austin was somewhat more fussy than usual after his vaccinations, but his father described him as laughing and happy in his bath less than half an hour before the 911 call. *See*, *e.g.*, Pet. Ex. 8, pp. 377-78.

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[34] Doctor Willson testified that he did not find any physical evidence of impact on Austin, but that was not unusual in cases where the child survived. Evidence of impact has been found on autopsy in cases where there was no external evidence of impact. Res. Ex. A at 445; Tr.2 at 169. `

[35] Doctors Uscinski and Wiznitzer provided similar testimony about dating blood based on its appearance on a CT scan. *See* Tr.3 at 121, 268.

c. The Initial MRI Scan and Second CT Scan.

Austin's initial MRI was performed on June 16, 2004, the day after his admission. It revealed an acute ischemic[36] infarction in the area of the anterior and posterior cerebral arteries. It also revealed subdural hemorrhage in the parafalcine interhemispheric dura.[37] Pet. Ex. 8, pp. 149-53. A second CT scan, performed on June 20, 2004 showed worsening cerebral edema. *Id*., pp. 155-56.

Doctor Willson described the initial MRI scan as "distressing, horrible," and indicated that it showed "diffuse bilateral areas of edema and probably stroke." Res. Ex. A at 420. He explained that edema results from a lack of oxygen supply to the brain cells, causing the cells to leak their intracellular fluid outside the cell wall. Although edema is a fairly nonspecific finding, the distribution of edema on Austin's MRI was "most consistent with a period of the baby not having oxygen or blood supply to the brain." Res. Ex. A at 421. The MRI showed blood collected "over the right hemisphere of the brain, blood all through the spinal fluid[38] and the subarachnoid space and also demonstrated in between the two hemispheres the further collection of blood, subdural blood." Res. Ex. A at 421. Diffuse dead brain tissue could be seen in the frontal and posterior areas of both hemispheres of Austin's brain. Res. Ex. B, Part 2, at 39; *see also* Tr.3 at 59-60 (Dr. Wiznitzer explaining that both the front and the back of Austin's brain were injured, and that the injuries could represent circulation problems and a coup or coup-contrecoup injury, and also noting that damaged tissue is more susceptible to further injury from poor oxygenation).

Doctor Rust explained that an ischemic brain injury[39] occurs when the heart continues to pump, but blood pressure is low. Tr.2 at 66; *see also id.* at 60. This happens when there is an obstruction of blood output from the heart, when a child is dehydrated, or as the result of strangulation. Strangulation interferes with blood flow through arteries that supply the brain and the Circle of Willis.[40] Tr.2 at 60, 66. Austin's

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[36] "Ischemia" means insufficient delivery of nutrients to involved tissue as the result of low blood pressure or impaired circulation. Tr.3 at 24-25. In Austin's case, it means that his brain did not have adequate circulation. Tr.3 at 25.

[37] The "parafalcine interhemispheric dura" refers to the part of the dura situated in the crease dividing the two halves of the brain near the falx. *See* DORLAND'S at 689, 962, 1396.

[38] Spinal fluid exists in the brain as well as in the spinal column. *See* GRAY'S ANATOMY (S. Standring ed., 40th ed. 2008) [GRAY'S ANATOMY"] at 425.

[39] Doctor Rust referred to brain swelling that was parasagittal, with the worst swelling in the anterior lobes and posterior occipital lobes. He indicated that this is the pattern seen with an ischemic injury. Tr.2. at 61. The parasagittal tissue is found near the middle of the brain, involving the inner portion of both hemispheres. Tr.3 at 59.

[40] The Circle of Willis is an area of the brain that connects several arteries permiting arterial blood flow to one hemisphere of the brain to reach the other hemisphere as well. GRAY'S ANATOMY at 251-52.

eye movement abnormalities also supported the existence of an ischemic injury. Tr.2 at 61.

d. The Spinal CT and MRI Scans.

Austin also had a CT on June 24, 2004 and an MRI on June 25, 2004, both imaging his cervical and thoracic spine. Pet. Ex. 8, pp. 175-88. Doctor Rust requested these studies to look for other evidence of strangulation or suffocation. *See* Tr.2 at 73, 75, 152-53.

The spinal MRI disclosed bleeding in Austin's cervical, thoracic, and lumbar spine.[41] Tr.2 at 153. Doctor Rust testified that there was some, although not definitive, evidence of neck vertebrae abnormality as well. Tr.2. at 59. Doctor Rust explained that this is present in "strangulation injuries." Tr.2 at 81. Doctor Willson characterized the spinal MRI results as "just classic again from shaking." Tr.2 at 153-54. According to Dr. Willson, the finding of blood in the spinal column, coupled with the previous findings of brain bleeding, made it "hard to imagine blood collecting like that without trauma." Res. Ex. B, Part 2, at 40.

e. Retinal Hemorrhages.

In addition to Dr. Newman's findings, described above, Dr. Rust also found widespread intraretinal and preretinal (subhyaloid) hemorrhages. He testified that these are a strong indicator of inflicted injury. Tr.2 at 59, 71-72; Res. Ex. V.

According to Dr. Willson, Dr. Newman was very cautious in his assessments, and that Dr. Newman told Dr. Willson that these were some of the worst retinal hemorrhages he had seen. Tr.2 at 153. Doctor Willson testified that retinal hemorrhages like Austin's could only be caused by severe trauma. Res. Ex. A at 428-29, 449.

f. The Left Tibial Fracture.

Although the initial skeletal survey done on admission was read as showing no evidence of fractures (Pet. Ex. 8, p. 148), another study performed 12 days later showed a periosteal reaction in the left ankle. Pet. Ex. 8, pp. 50, 190-91. Repeated ankle films taken a month later on July 15, 2004 showed a healed fracture in Austin's left ankle. Pet. Ex. 8, pp. 54; 198-99. Austin's discharge summary from the UVA hospital reflected a diagnosis of a healing, non-displaced left tibia fracture. Pet. Ex. 8, p. 49.

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[41] The testing revealed several separate and distinct areas of hemorrhage. There were subdural hemorrhages (1) extending from the posterior fossa to the C3 vertebra, (2) extending along the dorsum from T7 to T10, (3) along the ventral cord from T5 to T6, and (4) a ventral hemorrhage extending from T12 to L3. Pet. Ex. 8, p. 180.

Because there was no evidence to suggest that Austin's bones were fragile, Dr. Rust found the tibial fracture to be additional evidence that Austin had been the victim of NAT; he commented that the type of fracture was "virtually pathognomonic" of an inflicted injury in a child of Austin's age. Tr.2 at 59-60, 99. Doctor Willson testified that the radiologist who read Austin's skeletal survey believed there was a small, non-displaced fracture of his left tibia, but Dr. Willson referred to it as a "soft call," not a certainty. Res. Ex. A at 423.

5. Differences in the Treating Physicians' Opinions.

Focusing primarily on the bleeding observed in Austin's brain, Dr. Willson believed that Austin's injuries were, more likely than not, the result of shaking. Tr.2 at 151-52, 155. Focusing on the dead or dying brain tissue as well as his physical examination findings, Dr. Rust considered strangulation or smothering as a possible cause of Austin's ischemic injuries, but did not rule out shaking in addition, indicating that shaking was likely responsible for the subdural hematoma.[42] Tr.2 at 95.

About 12 days after Austin was admitted, doctors observed glottic swelling (swelling in the upper airway), leading Dr. Rust to suspect a strangulation injury. Tr.2 at 66-67. Although this swelling could have been the result of Austin's breathing tube, Dr. Rust explained that this type of swelling was not typically caused by intubation. He looked for other evidence sometimes found in strangulation cases, such as bruising on Austin's neck, or hemorrhage in his glottis, but did not find it. The lack of this evidence did not rule out strangling or smothering as a mechanism of injury. Tr.2 at 67. Even if Austin's injuries were not produced by smothering or strangling, NAT remained his diagnosis. Tr.2 at 67-69.

Doctor Willson believed that smothering or strangulation was a possible explanation for some of Austin's injuries, but that shaking was definitely involved. He believed that the retinal hemorrhages and bleeding in the brain and spinal column had to have come from trauma. Tr.2 at 167-68.

6. Austin's Discharge Diagnoses.

Austin's discharge summary from the UVA hospital reflected*, inter alia*, diagnoses of NAT, intermittent small cortical bleeding secondary to severe cortical atrophy and secondary tearing of the bridging veins, subdural bleeding in his brain and spine, bilateral retinal hemorrhages with vitreous bleeding in the left eye, and a healing, non-displaced left tibial fracture. At discharge, Austin had hypertonia, gastroesophogeal

[42] Doctor Rust testified that he had conducted a good deal of research into the areas of the brain most vulnerable to various types of injury: asphyxial, ischemic, and hypoxic-ischemic. Tr.2 at 96. Based on his experience, ischemic injuries are not produced by shaking. Tr.2 at 96. He attributed the subdural bleeding to shaking, caused by injury to bridging veins. Tr.2 at 95.

reflux, and seizures, and was dependent on gastrostomy tube feeding. Pet. Ex. 8, p. 49.

7. Petitioner's Challenges to the NAT Diagnosis.

Petitioner contends that the NAT diagnosis is not correct and bases her contention on four categories of evidence. First, she contends that Austin began reacting to his vaccinations shortly after they were administered, and experienced reactions that continued and intensified the following day, leading to his cardiopulmonary collapse.[43] Pet. Post-Hearing Br. at 8. Second, Ms. Huffman contends that the UVA doctors jumped to the conclusion that Austin was the victim of NAT and thus failed to consider and test for alternative diagnoses to explain Austin's comatose state. Pet. Post-Hearing Br. at 8-9. Third, she relies on Chris Huffman's denial that he injured Austin. Pet. Post-Hearing Br. at 7. Finally, she relies on expert testimony challenging the basis for the treating physicians' opinions that Austin's constellation of injuries could only be explained by NAT. Pet. Post-Hearing Br. at 16-20. She contends that this evidence undercuts the treating doctors' diagnosis of NAT and thus restores to her case the presumption of causation for a Table encephalopathy.

The first two categories of evidence do not require any extended discussion because the evidence overwhelmingly demonstrates that her assertions are either irrelevant or incorrect. The third and fourth categories, Mr. Huffman's denial and her expert evidence, are discussed in Section III, below.

a. Vaccine Reaction.

As signs of Austin's vaccine reaction on the evening before and the day of his collapse, Ms. Huffman points to unusual crying, a decreased response to his environment, decreased appetite, and increased vomiting.[44] *See* Pet. Post-Hearing Br.

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[43] Although this evidence looks like circumstantial evidence of vaccine causation, and it is clear from Ms. Huffman's statements that she believed Austin's vaccines caused his collapse, this evidence was used to undercut the NAT diagnosis, rather than as substantive evidence of a vaccinal cause. *See* Pet. Post-Hearing Br. at 20. Petitioner also used some of this evidence to challenge Dr. Wiznitzer's opinion that only NAT could produce the injuries Austin experienced. *Id*. at 12.

[44] She also relies on evidence related to head circumference changes to suggest that something other than trauma may have caused the problems in Austin's brain. Although Austin's head circumference increased significantly between his birth measurement of 13 ¾ inches and the 15 ¼ inches at his first well baby visit eleven days later (*see* Pet. Exs. 1, p. 30; 5, p. 5), I accept the explanation of Drs. Rust and Willson that this increase was not medically significant. As Drs. Willson and Rust explained, the unfused bones of the skull permit the bones to overlap temporarily, easing the passage of a baby through the birth canal. Head measurements taken at birth may reflect this "head molding," and the next head circumference measurement may reflect a substantial increase over the birth measurement, without generating any cause for concern. Res. Ex. A at 434; Tr.2 at 101-02. Austin's birth records reflect a significant degree of head molding with overriding sutures. Pet. Ex. 1, p. 30; *see also* Tr.3 at 372-73 (Dr. Wiznitzer's testimony that the head circumference measurements were not a cause for concern).

at 3 (citing to her testimony). However, her contention that Austin had signs of a vaccine reaction the evening before and the day of his collapse is based solely on her testimony and on affidavits or statements she made on or after June 17, 2004. *See* Pet. Post-Hearing Br. at 3 (citing Tr.1 at 79-81; Tr.2 at 113-14; Pet. Ex. 52 at 1); *see also* Pet. Exs. 6; 23 at 2; 46 at 7; 47 at 2.

Reliance on Ms. Huffman's testimony and affidavits is problematic. This evidence conflicts with other evidence provided by the Huffmans at the time of Austin's admission and initial treatment at UVA, when the need for accurate reporting was most acute and memories the freshest. Most of the specific events petitioner relies upon were not mentioned to health care providers on the evening of or the day after Austin's collapse or to the social worker and police investigator who interviewed the Huffmans on June 16, 2004. The later-produced evidence is unpersuasive.[45]

In both her testimony and affidavits, Ms. Huffman indicated that Austin was less responsive than usual the evening after he received his vaccinations,[46] but other evidence suggests that neither parent had concerns about his behavior that evening. Ms. Huffman's statement to Dr. Cary Sauer at UVA hospital[47] indicated that Austin was "somewhat fussy" after his vaccinations on June 14, 2004, but the Huffmans denied "any nausea, vomiting, diarrhea, or poor feeding, or fever." Pet. Ex. 8, p. 375. Although Ms. Huffman testified that this was not an accurate report of their conversation (Tr.1 at 132-33), I adopt the account provided in the contemporaneous medical record.

Investigator Jim Fetterman of the Harrisonburg Police Department and Social Services caseworker Erin Sours interviewed Ms. Huffman at the UVA hospital on June 16, 2004. According to Investigator Fetterman's notes, Ms. Huffman stated that Austin

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Austin's head circumference increased from 16 inches on April 7, 2004 to 17 ½ inches on the day of his four-month vaccinations. *See* Pet. Ex. 5, pp. 6- 7. Austin's pediatrician did not consider this a matter for concern. Tr.1 at 77; Res. Ex. E at 8. Doctor Rust testified that Austin's head measurement on June 14, 2004 was "beginning to get into the region where we consider external hydrocephalus," but he also indicated that at Austin's age, hydrocephalus would manifest with head circumference measurements "on the order of 120th to 130th percentile," not at Austin's percentile. Tr.2 at 102. He noted that even if hydrocephalus played a role in causing Austin's subdural hemorrhaging, it could not explain the other findings consistent with abuse. Tr.2 at 103.

[45] Even if this later version of events is correct, the presence of a vaccine reaction does not negate the likelihood that trauma caused Austin's brain injuries and retinal hemorrhages.

[46] *See* Tr.1 at 79. In both her affidavits, Ms. Huffman indicated that Austin had a decreased response to her and his environment. Pet. Exs. 47 at 2; 52 at 1; *see also* Pet. Ex. 23 at 2 (describing Austin as "uninterested to his environment" on the morning of June 15, 2004). A "decreased or absent response to environment" is a recognized sign of a Table encephalopathy. 42 C.F.R. § 100.3(b)(2)(i)(A) and (D)(1).

[47] The encounter note was written at about 10:00 PM on the night of Austin's collapse. Pet. Ex. 8, p. 375. Ms. Huffman testified that she talked with Dr. Sauer at about 8:00 PM that evening. Tr.1 at 104-05, 129-30. She and Chris Huffman also talked with Dr. Douglas Willson that same evening. Tr.1 at 105, 134-38.

awoke several times on the night of June 14, 2004, with his father attending to him.[48] Res. Ex. E at 6. Her hearing testimony was very similar to the accounts contained in her affidavit. *See* Tr.1 at 79-80; Pet. Exs. 47 at 2; 52 at 1. She testified that he woke up twice during the night, crying loudly each time. He settled down after being given a pacifier on one occasion and after a diaper change the other time. Tr.1 at 79-80.

However, the evidence indicates that Ms. Huffman's accounts of what transpired that evening were based only on Mr. Huffman's reports. Investigator Fetterman's notes indicate that Ms. Huffman did not wake up when Austin did on the night after his four-month vaccinations. The handwritten notes read: "Chris said he had been up a couple times during night." Res. Ex. J at 3. Investigator Fetterman's dictated and transcribed notes read: "Chris had told her that he had been up a couple times during the prior night with Austin." Res. Ex. E at 6. Mr. Huffman testified that he got up with Austin that evening. He described Austin's cry as "screaming" and "wailing" (Tr.2 at 113); he did not mention a shrill, high-pitched cry.

On the morning of June 15, 2004, Ms. Huffman fed Austin at about 6:30 AM. Res. Ex. E at 6; Tr.1 at 85. Although she previously reported to Investigator Fetterman that there was nothing unusual about Austin's behavior during this feeding (Res. Ex. E at 7), she testified that Austin vomited more copiously and more forcefully than usual that morning (Tr.1 at 149-50).[49]

Mr. Huffman remained home with Austin while Ms. Huffman departed for work at about 7:30 AM. Res. Ex. E at 7. From the time Ms. Huffman departed for work, Austin was in the sole care of his father, Chris Huffman. According to Mr. Huffman, he and Austin did not leave the apartment or see anyone until the arrival of EMS personnel. Res. Exs. B, Part 1, at 396-97; E at 11.

At about 10:00 or 11:00 AM, Mr. Huffman attempted to feed Austin a bottle with formula and rice cereal, but Austin refused to eat it. He then fed Austin about six

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[48] In accounts on and after June 17, 2004, Ms. Huffman described Austin as having a "shrill" or "high-pitched" cry upon awaking the evening after his four-month vaccinations. *See* Pet. Ex. 23 at 2 (describing cry as "shrill"); Pet. Exs. 47, p. 2; 52, p. 1 (affidavits reflecting that Austin awoke during the night "with a shrill scream" and resisted going back to sleep). She testified that she reported this shrill cry to all the health care providers after Austin was hospitalized. Tr.1 at 119-20, 137-38. However, this information does not appear in any of the medical histories in Austin's medical records, only in accounts made by Ms. Huffman after she began researching vaccine reactions as a possible cause of Austin's condition. Based on my experience in the Vaccine Program, I am aware that a shrill or high-pitched cry is often cited as evidence of a vaccine reaction.

[49] In other statements, Ms. Huffman indicated that Austin threw up two-four ounces of the formula she gave him. *See* Pet. Exs. 23 at 2; 47, p. 2. This amount was consistent with her report to the physician at Austin's check-up the prior day. However, her testimony indicated that this bout of emesis was more serious than the spitting up she had described to the physician. *See* Tr.1 at 149-50. In view of her denial to Dr. Sauer that Austin had vomited between his vaccinations and his collapse, I do not accept her testimony that Austin vomited more copiously on the morning after his vaccinations.

ounces of plain formula. Res. Exs. E at 10; B, Part 1, at 396 (Investigator Fetterman's reports of his June 16, 2004 interview with Chris Huffman). Mr. Huffman did not describe any difficulties with this feeding, either to Investigator Fetterman or in his testimony.[50] Res. Ex. E at 10; Tr.2 at 114. Mr. Huffman described Austin's behavior in the afternoon as normal, using the terms "playing," "laughter," and "bubbly" in his interview with Investigator Fetterman. Res. Ex. B, Part 1, at 397; *see also* Tr.2 at 114, 118. These descriptions are inconsistent with Austin exhibiting a decreased response to his environment.

I rely more heavily on the health care providers' and Investigator Fetterman's interviews of the Huffmans on June 15-16, 2004, than on petitioner's affidavits and hearing testimony.[51] I find that Austin was somewhat more fussy than usual and that he did not sleep as well as usual after his four-month vaccinations. Otherwise, I find that Austin's behavior after these vaccinations was generally in accordance with his behavior before the vaccinations. He continued to spit up after feeding and to resist taking cereal along with his formula. He interacted normally with his parents and others.[52] I find no reliable evidence that Austin awoke with a shrill or high-pitched cry the evening after his vaccinations, although his waking up at all was somewhat unusual.

b. Failure to Consider Alternative Diagnoses.

Contrary to Ms. Huffman's assertions that the UVA physicians never considered causes other than NAT, Dr. Rust's initial note after examining Austin (Res. Ex. V) indicated several other possible mechanisms of injury, although he considered them "unlikely alternative explanations." Tr.2 at 64; *see also* Tr.2 at 72. These included a clotting disorder, an organic acid disorder, an inflammatory condition such as Reyes syndrome, or a vascular abnormality in the brain. Tr.2 at 72-73; Res. Ex. V; *see also* Pet. Ex. 8, p. 516 (note written by Dr. Rust on June 16, 2004, indicating need to rule out the same possible causes).[53] Doctor Rust testified that dengue fever might produce a similar constellation of symptoms, but there was no evidence of Austin being vaccinated for or exposed to dengue fever, which is found primarily in Africa. He therefore excluded it as a possibility. Tr.2 at 65.

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[50] However, Pet. Ex. 23 at 2 reflects that, consistent with the report to Austin's pediatrician the day before about routine spitting up after feeding, Austin again spit up about two to four ounces of the formula.

[51] I note that Mr. Huffman's testimony was more or less consistent with his account of events immediately following Austin's hospitalization.

[52] Investigator Fetterman also interviewed Steven Stultz, Ms. Huffman's uncle, on June 21, 2004. He indicated that at about 8:00 PM on June 14, 2004, Shannon brought Austin upstairs to the main portion of the residence, where Mr. Stultz held the baby. According to Mr. Stultz, Austin appeared fine and was not fussy or crying. Res. Ex. E at 17.

[53] Doctor Rust's handwriting is difficult to read. For that reason, respondent's counsel had him read this and other notes he authored into the record at the June 24, 2009 hearing. *See* Tr.2 at 73-75.

Testing ruled out all of these as possible explanations for Austin's condition. Tr.2 at 64, 75-76; Pet. Ex. 8, p. 519. When the testing was negative, Dr. Rust wrote: "I feel that in the absence of other explanations this is clearly inflicted injury. Would have preferred to have MRA and MRV[54] as well, but cannot imagine a natural process that could produce this pattern." Pet. Ex. 8, p. 519 (emphasis original); Tr.2 at 75.

Nevertheless, it is true that the UVA treating physicians did not consider vaccine causation to be a plausible explanation for Austin's condition. Both testified that they had ruled out a vaccine injury. *See* Tr.2 at 68, 156-57; *see also* Res. Ex. A at 440 (Dr. Willson's testimony that it was not medically possible for vaccines to have caused the injuries he observed).

Doctor Rust acknowledged that recent vaccination is sometimes associated with injuries attributed to NAT, but is not the biological mechanism of injury. He explained that the vaccinations often cause irritability in the child, which provokes frustrated parents to inflict injury. Tr.2 at 61, 63. Dr. Willson also testified to this association. Tr.2 at 156. There was nothing about Austin's condition that in any way suggested that his vaccines played a biologically causal role. Retinal changes are not produced by vaccines. Tr.2 at 62. Intracranial hemorrhage can be the result of vaccinations, but the vaccines that can cause hemorrhage were not ones Austin received.[55]

III. Expert Testimony on the Validity of Austin's Trauma Diagnosis.

A. Overview.

1. Mr. Huffman's Denials.

Mr. Huffman's consistent denials that he injured Austin stand in stark contrast to the opinions of the treating physicians.[56] Mr. Huffman's explanation of the events of June 15, 2004 is entirely inconsistent with Austin's ultimate injuries. If the treating physicians' diagnosis of NAT is correct, the trauma exception defeats the Table injury claim. If their diagnosis is shown to be incorrect, then Austin's injuries are presumed to be vaccine-caused. Thus, the expert testimony contradicting and supporting the

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[54] MRA, magnetic resonance angiography, and MRV, magnetic resonance venography, are used to image blood vessels. *See* MOSBY'S at 1166.

[55] Doctor Rust referred to Pasteur's (rabies) vaccines as capable of producing acute hemorrhagic leukoencephalopathy, resulting in profound changes in the white matter and brainstem, but not the type of changes seen in Austin's brain. Tr.2 at 62. Doctor Rust also indicated that the old whole cell pertussis vaccine, when accompanied by Freund's complete adjuvant, produced brain changes in laboratory animals, but that whole cell pertussis could not cause brain changes of the type seen in Austin. Tr.2 at 62-63. Austin received an acellular pertussis vaccine. Pet. Ex. 5, pp. 2,6.

[56] I evaluate the persuasiveness of Mr. Huffman's denial as a factual finding in Section IV, below.

treating physicians bears directly on the ultimate question of whether Austin was injured on the day of his collapse.

2. The Expert Opinions.

In every important respect, the testimony of respondent's experts, Drs. Wiznitzer and Reece, supported that of the treating physicians, Drs. Rust and Willson. Respondent's two experts opined that Austin's injuries were almost certainly the result of NAT; both agreed with Dr. Rust that this case was as close to certain as one could get. *See* Tr.3 at 20-21, 82 (Dr. Wiznitzer agreeing that this case was close to 100% certain), 196 (Dr. Reece opining that trauma was "99% likely"). They relied on the same test results and reasoning as did the treating physicians. *See* Tr.3 at 21-24, 80, 180, 182-83, 195-96. Doctor Reece characterized the approach to Austin's care and treatment taken by the treating physicians in working up Austin's case as "outstanding." Tr.3 at 195.

They made three important additional contributions to respondent's case: First, they countered Dr. Uscinski's assertions that shaking alone could not produce sufficient acceleration to cause the injuries that Austin suffered, cogently explaining why Dr. Uscinski's reliance on certain studies was misplaced. Second, they offered additional testimony, strongly supported by a considerable body of medical literature, demonstrating that shaking is the most likely explanation for the constellation of injuries Austin suffered. They explained the medical and scientific basis for concluding that shaking alone or shaking plus impact on a soft surface leaving no external signs could cause the injuries that Austin suffered. Third, they successfully rebutted the primary bases for Dr. Uscinski's opinions that Austin's initial CT scan showed a chronic subdural hematoma and that CPR could explain his retinal hemorrhages. Doctor Wiznitzer used the computer images from the MRI and CT scans performed on Austin to provide a graphic explanation of the significance of Austin's injuries and to explain why Dr. Uscinski was wrong about the presence of a birth injury and a chronic subdural hematoma.

However, the crux of the dispute between the parties' experts concerned whether shaking alone could produce Austin's injuries.[57] Based on research involving the amount of force generated in simulations of shaking performed with doll models, Dr. Uscinski opined that shaking alone cannot produce sufficient force to cause subdural or subarachnoid bleeding, in the absence of an impact. Tr.3 at 240-45, 247. Although not explicitly so stated in their expert reports, Drs. Barnes and Gardner appear to share that

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[57] This framing of the issue ignores, for the moment, the opinions of Drs. Wiznitzer and Reece that Austin may well have sustained both shaking and an impact. Doctor Reece testified that autopsies have disclosed evidence of impact when no external evidence of impact was observable (Tr.3 at 205-06) and Dr. Uscinski acknowledged this as well (Tr.3 at 245). Furthermore, Case, a forensic pathologist, noted that on autopsy, the core injuries are the same in cases of suspected shaking as in cases of shaking plus an impact. M. Case, *Inflicted Traumatic Brain Injury in Infants and Young Children*, BRAIN PATHOLOGY 18: 571-82, 572 (2008) ["Case 2008b"], filed as Res. Ex. KK.

same opinion. Doctor Uscinski acknowledged that his opinion is shared by a minority of physicians, but equated the widespread reliance on shaking as a mechanism of injury as similar to the once widely held belief in a flat earth. Tr.3 at 320-22. He believed that the small amount of medical literature that supported his opinion was the result of "good science" and that the medical literature to the contrary was the result of *post hoc, ergo propter hoc* reasoning. Tr.3 at 321.

In contrast, Drs. Wiznitzer and Reece both subscribe to the generally accepted medical opinion[58] that shaking alone can produce "shearing" injuries, causing both subdural and subarachnoid bleeding, other brain injury resulting in an interference with breathing and circulation (a "diffuse axonal injury"[59]), and diffuse bilateral retinal hemorrhages. Coupled with other evidence of physical abuse, such as the fracture Austin sustained, these injuries are considered diagnostic for NAT, in the absence of any other medically plausible explanation. This mechanism of injury is commonly taught to medical students and residents. Hospitals in all states are required to inform new parents of the dangers of shaking an infant. *See* Tr.3 at 14, 164, 318.

The following subsections address the bases for these opinions in more detail. One important point deserves mention here, however. The bleeding observed on Austin's head CT on June 15, 2004 and on the MRI performed on June 16, 2004 was not itself responsible for Austin's cardiopulmonary collapse. Doctor Uscinski contends that Austin suffered birth trauma that caused bleeding, and that periodically in the months after his birth, the bleeding began anew, accounting for the blood seen on imaging studies. Consistent with petitioner's position that she presented a *prima facie* case for a Table encephalopathy, she made no substantial effort at the hearing to demonstrate a vaccine cause or, indeed, any cause, for Austin's encephalopathy, relying instead on the Table presumption of vaccine causation.[60]

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[58] Doctor Reece testified that about 95% of the relevant medical community accepts that shaking alone can produce serious injury. Tr.3 at 226.

[59] Autopsies have revealed diffuse axonal and shearing injuries to the brain in NAT. As the brain accelerates and decelerates, differential movement of parts of the brain with different tissue consistencies causes shearing injuries at the points of tissue differences. In infants and young children, these shearing injuries produce surface hemorrhages in bridging veins but do not produce hemorrhages within the brain, because the blood vessels are elastic and not, with the exception of the bridging veins, firmly attached to a structure (the dura at the skull) that allows them to snap. However, on autopsy, stained tissues do show damaged axons (*i.e.*, diffuse axonal injury). Case 2008b, Res. Ex. KK at 572, 576.

[60] Doctor Uscinski suggested that Austin's encephalopathy was caused by hypoxia. He based this on "prolonged" CPR and problems with intubation (Tr.3 at 274-75, 293), but he did not propose any reason for Austin to have stopped breathing, other than birth trauma. *See* Tr.3 at 255. In testifying about the length of the CPR, Dr. Uscinski indicated that the 911 call was received at 3:47 and that Austin did not arrive at RMH until 6:14. Tr.3 at 273-74. This was clearly an error. He apparently mistranslated the 24 hour time entries on Pet. Ex. 18 to 12 hour time. The transport record reflects that the 911 call was received at 15:47, the EMS personnel arrived at the Huffmans' apartment at 16:01, and that Austin arrived at RMH at 16:14. Pet. Ex. 18, p.1. Although the emergency records reflect that Austin was successfully intubated on the first attempt at the scene (Pet. Ex. 18, p. 1), Dr. Uscinski seemed to imply that he was not, based on the amount of air in his stomach and bowel. Tr.3 at 273. I note that the RMH

Respondent's experts (and Austin's treating physicians) believed that the blood was a marker that showed Austin's brain had been traumatized, rather than the mechanism by which the brain injury occurred. They were careful to note that the amount of bleeding was small, and insufficient to produce a "mass effect," *i.e*., swelling-produced compression, shifting parts of the brain out of position. Tr.3 at 41, 53, 177, 369-70. The trauma itself caused Austin's cardiopulmonary collapse, which led to the anoxic brain injury, producing swelling and, ultimately, the almost complete breakdown of grey-white matter differentiation in Austin's brain. Tr.3 at 23-24, 61.

In her effort to undercut the evidence that Austin's injuries were the result of trauma, petitioner relied primarily on the testimony of Dr. Uscinski and the reports of Drs. Barnes[61] and Gardner.[62] None of these three physicians attributed Austin's injuries to a vaccine, although Dr. Barnes included "postvaccinial" infection in his differential diagnosis, pending complete medical workup.[63] Pet. Ex. 12 at 1. Doctor Uscinski opined that, in the absence of any external evidence of trauma, Austin was not shaken. He testified that it was more likely than not that Austin suffered some trauma at birth, causing a chronic subdural hematoma. Tr.3 at 254. Doctor Barnes offered a conflicting opinion, stating that there were "no specific findings to indicate trauma, including nonaccidental injury." He indicated that a "combined etiology" such as an infection complicated by hypoxia-ischemia should be considered. Pet. Ex. 12 at 1. Doctor Gardner indicated that the retinal hemorrhages could be the result of intracranial hemorrhages "from little or no trauma" and that this bleeding, plus CPR and an increased bleeding tendency,[64] could account for the eye findings. Pet. Ex. 13 at 2.

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records do not indicate any problems with the endotracheal tube placed by the EMS personnel and do not indicate any re-intubation attempts. Pet. Ex. 7 at 101; *see also id*. at 125 (radiology report confirming the endotracheal tube's proper placement). Alternatively, Dr. Uscinski may have been referring to Mr. Huffman's CPR attempts. Tr.3 at 274.

[61] Petitioner did not file his CV, but Dr. Barnes' letter reporting his causation opinion, Pet. Ex. 12, identifies him as a pediatric neuroradiologist. His signature block indicates he is, or at least was at the time he produced his opinion, Chief of Pediatric Neuroradiology and Director of the Pediatric MRI and CT Center at Stanford University Medical Center. *Id.* at 2.

[62] Doctor Gardner is an ophthalmologist. *See* Pet. Ex. 13. The CV included with his report indicates that at the time he rendered his opinion, he was a rehabilitation consultant for the State of Colorado and a volunteer physician with a community health center system. *Id.* His most recent publications were related to child abuse and a hypothesis on the relationship between immunizations and retinal and subdural hemorrhages. *Id.*

[63] No evidence of infection was found. *See* Tr.2 at 58-59.

[64] Doctor Gardner opined that two of the blood tests performed at UVA showed an increased bleeding tendency. Pet. Ex. 13. Doctor Rust testified that Austin did not have any clotting disorder. Tr.2 at 60, 64. The elevations in Austin's blood tests were mild, according to his UVA medical records. Pet. Ex. 8, pp. 51 ("mild elevations … normalized on repeat one week later"), 53-54, 117, 123, 136-37. A notation on the form indicated that, for reference ranges for children less than one year of age, the laboratory should be contacted. *Id*., p. 117. NELSON'S PEDIATRICS indicates that Austin's PTT (partial thromboplastin time) on June 16, 2004, 35.5 seconds, would be considered normal in a full term infant under one year of age.

Petitioner's evidence undercutting shaking as a mechanism of injury is discussed in subsection B below, followed by the contrary evidence that shaking can cause subdural and subarachnoid bleeding and diffuse axonal injuries. Subsection C discusses the evidence regarding the causes of retinal hemorrhages.

B. Shaking as a Mechanism of Injury.

1. The Evidence that Shaking Cannot Produce Sufficient Force.

Doctor Uscinski's testimony challenged the widely accepted medical belief that shaking alone can produce sufficient force to produce subdural hematomas, diffuse axonal injury, and diffuse bilateral retinal hemorrhages. His opinion relied primarily on two studies, one from 1987[65] ["Duhaime"] and one from 2003[66] ["Prange"]. Tr.3 at 234-36, 241-45, 247-50. Although Dr. Uscinski has authored three papers on shaken baby syndrome, one a very short article published in the AMERICAN JOURNAL OF PHYSICIANS AND SURGEONS in 2004,[67] and two in NEUROLOGIA MEDICO-CHIRURGICA,[68] a Japanese international journal of neurosurgery (Tr.3 at 232-33[69]), none involved original research.[70]

NELSON TEXTBOOK OF PEDIATRICS 2065 (18th ed. 2007). *See also* Tr.3 at 141 (Dr. Wiznitzer's testimony that Austin's readings were normal or near normal, and did not evince a bleeding disorder).

[65] A. Duhaime, et al., *The shaken baby syndrome: A clinical, pathological, and biomechanical study*, J. NEUROSURG. 66:409-15 (1987), filed as Pet. Ex. 56.

[66] M. Prange, et al., *Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants*, J. NEUROSURG. 99: 143-50 (2003), filed as Pet. Ex. 57.

[67] *See* R. Uscinski, *The Shaken Baby Syndrome*, J. AM. PHYSICIANS &SURGEONS 9(3): 76-77 (2004), filed as Pet. Ex. 60. The article is less than two pages long. Although Dr. Uscinski testified that this article was peer reviewed (*see* Tr.3 at 232), articles published in this journal are not subject to peer review prior to publication. *See Dwyer v. Sec'y, HHS*, No. 03-1202V, 2010 WL 892250, *101 n.408 and *104 (Fed. Cl. Spec. Mstr. Mar. 12, 2010) (discussing another article published in this journal); *see also id.* at *9 n.37 (describing the importance of peer review). The article itself does not reflect any peer review, which is usually indicated by a reference to the date the article was submitted, followed by a later date indicating when the article was accepted for publication.

[68] R. Uscinski, *Shaken Baby Syndrome: An Odyssey*, NEUROL. MED. CHIR. 46:57-61 (2006), filed as Pet. Ex. 63; R. Uscinski & D. McBride, *The Shaken Baby Syndrome: An Odyssey II Origins and Further Hypotheses*, NEUROL. MED. CHIR. 48: 151-56 (2008), filed as Pet. Ex. 64.

[69] The name of the latter journal is misspelled in the transcript (Tr.3 at 232-33), but correctly reported in Dr. Uscinski's CV, Pet. Ex. 55 at 2.

[70] Although Dr. Uscinski testified that his second paper in the Japanese journal involved "research" (*see* Tr.3 at 323), the research was conducted on medical literature and did not involve experiments. *See* Pet. Ex. 64.

Doctor Uscinski explained that most papers on shaken baby syndrome were ultimately derived from research published in 1968 by Dr. Ayoub Ommaya.[71] Tr.3 at 235-36. While working at NIH, Dr. Ommaya measured the degree of force necessary to produce concussions in rhesus monkeys subjected to rear-end motor vehicle collision simulations. Doctor Uscinski indicated that Dr. Ommaya and others extrapolated from that data to determine how much force would be necessary to produce similar injuries in humans. Tr.3 at 237-39.

According to Dr. Uscinski, those investigating what caused unexplained subdural bleeding in children without external evidence of impact seized on Dr. Ommaya's findings to hypothesize that these children were being shaken. However, they did so without sufficient evidence that shaking alone could produce enough acceleration to produce those injuries.

Doctor Uscinski relied on the Duhaime and Prange studies[72] as evidence that shaking alone cannot produce acceleration sufficient to cause hemorrhage, concussion, or other direct injury to the brain.[73] Tr.3 at 240-44. The Duhaime study measured shaking alone, as well as shaking plus an impact on various surfaces. The Prange study also included falls from various heights, in addition to shaking and shaking plus impact. Tr.3 at 248-49; Duhaime, Pet. Ex. 56 at abstract; Prange, Pet. Ex. 57 at abstract.

Both studies involved shaking doll models fitted with a device to measure acceleration. Duhaime, Pet. Ex. 56 at abstract, 413; Prange, Pet. Ex. 57 at 144-45. In neither study did shaking alone produce acceleration over the level that the authors estimated was necessary to produce injury. Duhaime, Pet. Ex. 56 at 414; Prange, Pet.

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[71] Doctor Ommaya's name is misspelled throughout the transcript at "Ommaye." His 1968 paper was cited in many of the medical literature exhibits filed by both parties. *See, e.g.*, Pet. Ex. 63 at 61.

[72] The 1987 Duhaime study (Pet. Ex. 56) was co-authored by Lawrence Thibault, a biomechanician. *See* Tr.3 at 235. The 2003 Prange study (Pet. Ex. 57) was co-authored by Duhaime. The witnesses used several authors' names in referring to these two studies, creating some confusion in the hearing transcript. *See, e.g.*, Tr.3 at 201, 203-04, 241, 325, 382. Other studies co-authored by Thibault were referenced in the Duhaime study, Pet. Ex. 56 at 414, and in R. Minns, *Shaken baby syndrome: theoretical and evidential controversies*, J. R. COLL. PHYSICIANS EDIN. 35:5-15, 7-8 (2005) ["Minns"], filed as Res. Ex. HH. It is likely that at times Dr. Uscinski was referencing one of these studies (*see, e.g.*, Tr.3 at 307-08), rather than the 1987 Duhaime study. Other Thibault studies were not filed.

[73] However, Dr. Uscinski acknowledged that these studies demonstrated that shaking, coupled with an impact, could produce sufficient force to "cross the injury threshold." Tr.3 at 242-43. Paraphrasing Dr. Reece's testimony, "injury threshold" can be defined as the level of force necessary to produce an injury, but no injury threshold has been established for infant brains. Tr.3 at 200, 380. Doctor Uscinski also agreed that an impact could produce the bleeding observed on Austin's CT and MRI scans, although he qualified that answer by indicating that he would expect to see soft tissue injury at the point of impact on either CT or MRI scans. Tr.3 at 344.

Ex. 57 at 148. The 2003 Prange study used more sophisticated models and involved impacts against both hard and soft surfaces. Tr.3 at 248; Prange, Pet. Ex. 57 at 147.

Based on Dr. Ommaya's work and the Duhaime and Prange studies, Dr. Uscinski opined that intracranial hemorrhages could not be produced in infants by manual shaking. Tr.3 at 257. He testified that sufficient force to cause subdural hemorrhage in a four month old infant would produce spinal cord injury such as a broken neck. Tr.3 at 257-58. He would also expect to see rib injury, if the child were grabbed around the torso. Tr.3 at 258. He also testified that it would require no force at all to produce new bleeding from a chronic subdural hematoma. Tr.3 at 324.

2. Evidence that Shaking Alone Can Cause Injury.

a. Problems with the Duhaime and Prange Studies.

Doctor Reece testified that the Duhaime and Prange studies were useful, but noted that their doll models were crude[74] and not "biofidelic" (*see* Tr.3 at 391-93), meaning that they did not mimic what would happen inside an infant brain[75] (*see, e.g.*, Tr.3 at 223-24). Doctor Reece took some exceptions to Dr. Uscinski's descriptions of the studies themselves and the conclusions of the researchers, but was in agreement with Dr. Uscinski that, with the models used, vigorous shaking did not demonstrate the force the Duhaime and Prange researchers believed was necessary. *See* Tr.3 at 390-93. Expanding on this point, however, Dr. Reece testified that there is no means to quantify the force necessary to cause brain injury. Tr.3 at 380-81; *see also id.* at 200. He pointed out that there is no established general injury threshold for infants. Different tissues require different levels of force to cause an injury, and nonaccidental trauma may involve several types of tissue. Tr.3 at 380-81; *see also* M. Case, *Forensic Pathology of Child Brain Trauma*, BRAIN PATHOLOGY 18: 562-64, 564 (2008) ["Case 2008a"], filed as Res. Ex. JJ; Case 2008b, Res. Ex. KK. Thus, he did not accept Dr. Uscinski's conclusion that shaking alone could not produce the injuries seen in Austin. *See* Tr.3 at 390-91.

Others have noted the same problem with the degree of force used as the injury threshold in the Duhaime and Prange studies. *See* R. Block, *Child Abuse—Controversies and Imposters*, CURR. PROBL. PEDIATR. 29: 253-72, 254 (1999), filed as Res. Ex. GG (commenting that the threshold was only an extrapolation from adult animal studies). As the Minns paper noted: "Their injury thresholds for concussion, [subdural hematoma] and diffuse axonal injury were derived from studies in adult primates and where the rotational motion was from a single inertial event." Res. Ex. HH

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[74] Doctor Reece called the doll models used in the study "terrible." Tr.3 at 392. He explained that the models used a hinge to hold the head on, with a strain gauge in the head to measure the force levels. Tr.3. at 393. He noted that better models are being used in some current research. Tr.3 at 394.

[75] Doctor Uscinski appeared to agree that the models did not replicate the human brain. *See* Tr.3 at 339.

at 9. Thus, the computed threshold could not address the effect on the brain of repeated acceleration-deceleration injuries produced by shaking.

One of the most significant weaknesses of both studies concerned the method by which the head of the model was attached to the neck. Both studies used metal hinges. Duhaime, Pet. Ex. 56 at 411 (this study also tested a rubber neck); Prange, Pet. Ex. 57 at 144. These hinges allowed for measurement of straight line force, but did not simulate the rotational forces generated by shaking a human infant. *See* Tr.3 at 393.

Finally, Dr. Duhaime herself has acknowledged that reliance on the doll models currently available has serious shortcomings. In an editorial, she listed some of the steps necessary to draw valid predictions from anthropomorphic models to injury in human infants, referring to the need to reach an understanding of injury thresholds in specific tissue types. *See* A. Duhaime & C. Dodge, *Closer but not there yet: models in child injury research*, J. NEUROSURG. PEDIATRICS 2: 320 (2008), filed as Res. Ex. II.

b. Other Evidence that Shaking is a Reliable Hypothesis.

When controlled studies cannot be performed, the medical community frequently relies on circumstantial evidence to determine the cause for medical conditions. For example, there are no studies that involve infecting human beings with the human immune deficiency virus to prove conclusively that it causes AIDS. The same ethical considerations that prohibit infecting humans with a potentially deadly virus apply to conducting controlled studies involving shaking infants. Therefore, physicians and other researchers have relied on other types of evidence to conclude that shaking can and does cause subdural and subarachnoid bleeding and diffuse axonal injury.

Ample circumstantial evidence is available to support the majority opinion in the medical community that shaking does cause serious and long-lasting head trauma in infants. The types of circumstantial evidence include: (1) the physical vulnerability of infants' heads and necks to rotational injuries; (2) confessions of caregivers to shaking infants who present with injuries similar to Austin's; and (3) data acquired from animal and computer models. The fourth type of circumstantial evidence, the co-occurrence of subarachnoid or subdural bleeding and other types of trauma, including diffuse retinal hemorrhages, is addressed in subsections C and D below.

(1) Physical Vulnerability.

Several researchers have detailed the anatomical differences between adult and infant human brains that render infants more vulnerable to rotational injuries, such as those produced by shaking, as opposed to straight-line impacts such as falls.[76] This

[76] Falls from heights or other straight-line head impacts cause external bruising, skull fractures, and focal brain contusions, but do not produce concussion or encephalopathy in the absence of severe brain injury. *See, e.g.*, Minns, Res. Ex. HH at 8, 13.

evidence suggests that the reliance Dr. Uscinski and the Duhaime and Prange researchers placed on studies of degrees of force involving adult cadavers is misplaced.

The infant skull is thin, pliable, and prone to deform, assisting in the birth process, but rendering the infant vulnerable to traumatic injury. Case 2008a, Res. Ex. JJ at 563; Tr.3 at 369. Infant brains are large, compared to body size, comprising about 10-15% of body weight in an infant, compared to 2-3% of body weight in an adult. Case 2008a, Res. Ex. JJ at 563; Minns, Res. Ex. HH at 7. The infant brain is softer, and the subarachnoid space is thin and covers a large surface area. Case 2008a, Res. Ex. JJ at 563. Infant brains are poorly myelinated, which makes diffuse axonal injury from strain more likely.[77] Case 2008a, Res. Ex. JJ at 563; Minns, Res. Ex. HH at 7 (noting that the relative difference in the specific gravity of grey and white matter, which dictates a difference in how the two types of brain matter respond to acceleration, renders sheering injuries more likely). Infant heads contain extra space between the skull and the cerebrum, allowing for more movement of the brain within the skull when the head is subjected to acceleration and deceleration. This extra space is maximized at about five months of age. Minns, Res. Ex. HH at 7; *see also* Tr.3 at 176. Undeveloped neck muscles are unable to support the head, making it more vulnerable to acceleration-deceleration injuries. Case 2008a, Res. Ex. JJ at 563; Minns, Res. Ex. HH at 7.

These anatomic differences in infants create a vulnerability to shearing injuries. The hypothesized mechanism is that dynamic injuries to the head cause inertial movement of the brain within the skull and rotation of the unsupported head at the point where it joins the spine. Subdural hematomas result from the differential movement of the brain and skull, which cause strain and tearing of the bridging veins, resulting in interhemispheric subdural hemorrhage.[78] The inertial movement of the brain also causes the diffuse axonal trauma, making subdural hematomas a marker for the diffuse axonal injuries, which are difficult to detect on neuroimaging. *See* Case 2008a, Res. Ex. JJ at 564.

(2) Confessions of Caregivers.

Doctor Reece testified that in most cases of suspected child abuse, there is either no history that could account for the physical findings or a history incompatible

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[77] Case relied, at least in part, on a study of neonatal pig brains for this point. That study found non-impact rotational velocities caused three times the axonal damage in neonatal brains than in older brains. Case 2008a, Res. Ex. JJ at 563.

[78] The dura is attached to the inner surface of the skull, and the arachnoid layer is attached to the pia on the surface of the brain. The bridging veins run from the cortical surface of the brain to the venous sinuses in the dura. These veins are attached strongly to the arachnoid, but more weakly at the dura. When acceleration is applied to the infant head, differential acceleration between the brain and the skull occurs. The dura moves with the head and the arachnoid moves with the brain, causing strain to the bridging veins. Case 2008b, Res. Ex. KK at 572. The inertial motion of the brain begins in the posterior hemispheric fissure, resulting in the first evidence of bleeding in that area (*id.*), the same area where Austin's bleeding was observed.

with the type of injury seen. *See* Tr.3 at 175. However, some caregivers do confess to shaking the injured child. These caregiver confessions may present some reliability problems, as false or coerced confessions are certainly a possibility and some voluntary admissions may be inaccurate or incomplete. Still, there is a high degree of similarity in the physical injuries in cases with and without confessions of caregivers, which supports a conclusion that shaking causes this constellation of injuries. Tr.3 at 381-82; *see also* S. Starling, et al., *Analysis of Perpetrator Admissions to Inflicted Traumatic Brain Injury in Children*, ARCH. PEDIATR. ADOLESC. MED. 158: 454-58, 457 (2004) ["Starling"], filed as Res. Ex. FF. Confessions to shaking rendered in cases where there are other obvious signs of abuse corroborates confessions in cases lacking these other signs.

The Starling study examined 81 cases of traumatic brain injury in which caregivers admitted abuse. In 69 cases, the perpetrator of the abuse admitted to a specific mechanism of injury, either shaking alone, impact alone, or a combination of the two. Forty-nine perpetrators admitted to some form of shaking, with 32 admitting to shaking as the only mechanism. Children whose caregivers admitted to shaking only were more likely to have subdural hematomas and retinal hemorrhages than those subjected only to impact and were far less likely to have either skull fractures or scalp swelling. Starling, Res. Ex. FF at 456, Table 2. This corroborates the confessions and supports a conclusion that shaking alone can cause the injuries that Austin suffered.

The Minns paper, Res. Ex. HH, discussed a study of 124 cases of suspected NAT in Scotland. In 23% of those cases, adult caregivers admitted injuring the child. Of those admissions cases, 89% of the infants had subdural hematomas, 68% had retinal hemorrhages, and 36% had skeletal injuries. Based on the small percentage of cases in which there was any evidence of skull fractures and external injuries, Minns concluded that most cases involved shaking alone. Res. Ex. HH at 6. He also noted a well-documented case report involving shaking alone (*id*. at 7), and two cases in which adults who were violently shaken had retinal hemorrhages and subdural hematomas. One of the adults, who also had evidence of diffuse axonal injury, died. Minns, Res. Ex. HH at 7. While these adult cases likely included more force exerted for a longer time period, they support a finding that shaking is a mechanism that can cause these injuries.

(3) Animal Studies and Computer Models.

The hypothesis that diffuse axonal injury is caused by shaking is supported by autopsy findings. Case 2008b, Res. Ex. KK at 576; *see also* Tr.3 at 381-82. It is also supported by animal studies. The Case 2008b paper described several animal studies of acceleration and deceleration injuries producing axonal or neuronal damage and hypoxia. In the Bonnier study, mouse pups shaken once for 15 seconds demonstrated both axonal damage and retinal hemorrhages. Newborn rats in the Bittigau study were subjected to focal impact and shaking on three separate occasions, and showed extensive neuronal death in the gray matter. Newborn rats in the Smith study were subjected to shaking without impact and had neuronal degeneration with evidence of hypoxia during the shaking, as well as retinal hemorrhages. Although the Smith

researchers did not find diffuse axonal injury, they noted that axonal damage would be difficult to observe in neonatal rats because of rapid development of myelination and nerve fiber growth. Case 2008b, Res. Ex. KK at 576.

The Minns paper discussed computer modeling of shaking injuries. The described experiments supported the concept that rotational accelerations such as those produced in shaking were likely to produce sufficient strain on bridging veins to snap them by manual shaking. Unlike the doll model studies of Duhaime and Prange, which focused on the force generated by shaking, the computer models focused on the amount of force actually needed to tear bridging veins. Minns, Res. Ex. HH at 8.

C. Association with Retinal Hemorrhages.

1. Petitioner's Experts' Opinions.

Doctor Uscinski testified that retinal hemorrhages were not causally associated with NAT. Tr.3 at 285-86, 293; *see also* Pet. Ex. 12 at 1 (Dr. Barnes' report indicating that retinal hemorrhages are a nonspecific finding). Doctor Uscinski indicated that an abrupt increase in venous pressure, possibly as the result of an increase in intracranial pressure caused by Austin's cardiopulmonary collapse and resultant edema, could account for his retinal hemorrhages. Tr.3 at 285-90. Doctor Uscinski concurred with Dr. Gardner (*see* Pet. Ex. 13 at 2) that Austin's retinal hemorrhages were probably the combination of increased intracranial pressure and the "prolonged" resuscitation attempts. Tr.3 at 293.

2. Studies Supporting Trauma as Causal.

In spite of these opinions, the weight of the evidence is that <u>diffuse</u> retinal hemorrhages,[79] the type seen in Austin, are causally linked to abusive head trauma in general and shaking in particular. The medical literature filed[80] supported Dr. Reece's testimony that diffuse retinal hemorrhages are a strong marker for NAT, and are not caused by CPR. Tr.3 at 171; *see* Case 2008b, Res. Ex. KK at 575 (explaining that retinal hemorrhages occur rarely with CPR, and when they "occur in these conditions that are unrelated to inflicted neurotrauma, the hemorrhages tend to be few in number

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[79] Doctor Reece testified that one of the leading pediatric ophthalmologists, Dr. Alex Levin, defined diffuse bilateral retinal hemorrhages based on three criteria: (1) multiple hemorrhages in each eye; (2) hemorrhages appearing in multiple layers of the eyes; and (3) hemorrhages not confined to the posterior pole (the point at which the optic nerve enters the globe of the eye) and extending out almost to the area around the lens. According to Dr. Reece, these three criteria are seen almost exclusively in cases of shaking or shaking plus impact. Tr.3 at 170-72. Doctor Levin was an author or co-author of studies filed as Res. Exs. AA, BB, and CC.

[80] Petitioner filed two publications by Dr. Gardner that did not support Dr. Reece's testimony, but both are correspondence. Petitioner's Ex. 65 is a brief letter to the editor criticizing a study, and Pet. Ex. 66 is a short literature review advocating for a link between vaccines and retinal hemorrhages.

and confined to the posterior pole of the retina"). Retinal hemorrhages occur in about 85% of cases of suspected NAT. Case 2008b, Res. Ex. KK at 575; A. Levin and Y. Morad, *Ocular Manifestations of Child Abuse*, *in* CHILD ABUSE: MEDICAL DIAGNOSIS AND MANAGEMENT 211-25 (R. Reece and C. Christian ed., 2009) ["Levin and Morad"] at 211, filed as Res. Ex. AA; Y. Morad, et al., *Correlation Between Retinal Abnormalities and Intracranial Abnormalities in the Shaken Baby Syndrome*, AM. J. OPHTHALMOL. 134(3): 354-59, 357 (2002) ["Morad 2002"], filed as Res. Ex. CC.

The mechanism by which the retina is injured in shaking is similar to the hypothesis of how shaking causes diffuse axonal brain injury. The ten layers of the retina move differently under acceleration and deceleration, causing microvascular shearing and thus producing hemorrhages. Tr.3 at 177.

Significantly, diffuse retinal hemorrhages are not found in cases of mild accidental trauma.[81] Case 2008b, Res. Ex. KK at 575; Levin and Morad, Res. Ex. AA at 216. In the rare cases when retinal hemorrhages occur outside the context of suspected inflicted injury, they are few in number and are found only in the posterior pole of the retina. Case 2008b, Res. Ex. KK at 575; Levin and Morad, Res. Ex. AA at 216. A pattern of multiple hemorrhages throughout the retina accompanied by a vitreous hemorrhage (the pattern found in Austin) would not have any differential diagnosis other than that of child abuse. *See* Levin, Res. Ex. BB at 159, 179.

In a case study of all children with apparent NAT presenting to a specific hospital over a period of six years, the severity of head trauma and retinal hemorrhages was each scored. Morad 2002, Res. Ex. CC at 355. The study found a high correlation between severe head trauma and severe retinal hemorrhage. There was no correlation between signs of impact and retinal hemorrhage, a finding that the authors considered as supporting the hypothesis that shaking alone could account for both brain and eye injuries. Morad 2002, Res. Ex. CC at 357-58.

3. Refuting CPR as a Cause.

Doctor Reece rejected Dr. Uscinski's testimony that CPR can cause retinal hemorrhages. He explained that retinal hemorrhages may be seen in children who have received CPR, but they are not found unless the CPR is required because of the brain injury caused by abuse. Tr.3 at 387; *see also* Tr.3 at 118-19 (similar testimony by Dr. Wiznitzer).

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[81] Retinal hemorrhages can result from bleeding disorders, sepsis, and meningitis, and they sometimes occur in very severe accidental trauma, such as in motor vehicle accidents. Case 2008b, Res. Ex. KK at 575; *see also* Levin and Morad, Res. Ex. AA at 216. Retinal hemorrhages are found in about 3% of cases of accidental head trauma, and most of those involve life-threatening events that raise no suspicion of abuse, such as motor vehicle accidents. A. Levin, *Retinal haemorrhages and child abuse*, *in* RECENT ADVANCES IN PAEDIATRICS 151-219 (T. David ed., 2000) ["Levin"] at 179, filed as Res. Ex. BB.

Respondent's experts were supported by a 1993 study which convincingly demonstrated that diffuse retinal hemorrhages rarely occur in association with CPR.[82] None of the 49 children in the study who died after prolonged vigorous resuscitation attempts from conditions not associated with NAT had retinal hemorrhages. The skill of the person performing the CPR with chest compressions did not affect these results. Gilliland & Luckenbach, Res. Ex. MM at 190-91; *see also* Levin, Res. Ex. BB at 182-85 (chronicling similar studies and case reports).

4. Refuting Increased Intracranial Pressure as Causal.

The studies filed by respondent and the testimony of Drs. Reece and Wiznitzer also convincingly countered Dr. Uscinski's testimony and Dr. Gardner's report that increased intracranial pressure causes retinal hemorrhages. Doctor Wiznitzer testified that there was not enough bleeding to cause an immediate increase in intracranial pressure and only a rapid increase in intracranial pressure produces retinal hemorrhages. Austin's anoxic encephalopathy did not result in enough swelling to cause a marked increase in his intracranial pressure for 12-24 hours after the event. Tr.3 at 77-78. Doctor Reece also testified that elevations in intracranial pressure alone and in conjunction with anoxia do not cause significant retinal hemorrhages. Tr.3 at 189-90, 198-99, 223. He was supported in this testimony by several medical journal articles and textbook chapters.[83] In the Morad 2002 study, the patients who showed signs of increased intracranial pressure "did not differ in type, extent, or frequency of retinal findings when compared with the rest of the patients." Res. Ex. CC at 357. There were similar negative findings for seizures, severe cough, and vomiting in other studies.[84]

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[82] M. Gilliland and M. Luckenbach, *Are Retinal Hemorrhages Found After Resuscitation Attempts?* AM. J. FORENSIC MED. PATHOL. 14(3): 187-92 (1993) ["Gilliland and Luckenbach"], filed as Res. EX. MM. Postmortem ocular examinations were performed on a sample of 169 children who had died from a variety of causes, including intentional injury, suspected child abuse, accidental trauma, and apparent natural death. Prolonged CPR (lasting longer than 30 minutes) had been performed on 131 of the children studied. Gilliland and Luckenbach, Res. Ex. MM at 188. The authors concluded that retinal hemorrhages were not found in the absence of injuries or diseases known to cause such hemorrhages, and that prolonged vigorous resuscitative attempts did not produce such hemorrhages, in the absence of other causes for them. This included eight children who died as the result of extreme force to the trunk in unskilled attempts at CPR. They also concluded that retinal hemorrhages were important markers for abuse. *Id*. at 190-91.

[83] One study noted that massive retinal hemorrhages throughout the entire retina are rarely reported in conditions other than shaking, and when they are (in the first few days after birth or after severe motor vehicle accidents involving multiple impacts), there is clear evidence of a specific cause other than abuse. Levin and Morad, Res. Ex. AA at 213; *see also* Morad 2002, Res. Ex. CC at 357 (finding no correlation between increased intracranial pressure and severity of retinal hemorrhages in a six year study).

[84] *See* S. Sandramouli, et al., *Retinal haemorrhages and convulsions*, ARCH. DIS. CHILD. 76:449-51 (1997), filed as Res. Ex. NN. This study concluded: "Convulsions rarely (if ever) give rise to retinal haemorrhages. The finding of retinal haemorrhages should stimulate a detailed assessment to exclude non-accidental injury, whatever the nature of the associated or antecedent events." *Id*. at abstract; *see also* A. Curcoy, et al., *Do retinal haemorrhages occur in infants with convulsions?* ARCH. DIS. CHILD. 94:

D. The Presence of Other Injuries.

In addition to the association of subarachnoid or subdural bleeding with diffuse retinal hemorrhages, this type of bleeding is also associated with other nonaccidental injuries. The co-occurrence of subarachnoid or subdural bleeding with long bone fractures in infants with no explanation for either injury was first noted in 1946 by Caffey, who is also credited with coining the term "whiplash shaken baby." *See* Minns, Res. Ex. HH at 5, 6; *see also* Case 2008b, Res. Ex. KK at 571; Duhaime, Pet. Ex. 56 at 409. In one autopsy study of deaths due to suspected NAT, over half of the cases presented with additional injuries, and over half of those included recent or old fractures. Case 2008b, Res. Ex. KK at 577. Absent any medical reason for an infant being prone to fractures or any history of accidental trauma sufficient to cause fractures, these cases are very suggestive of nonaccidental trauma. As Dr. Rust testified, infants like Austin, who cannot walk, are very unlikely to fracture bones, and such fractures are "virtually pathognomonic" of abuse. Tr.2 at 59-60, 99.

The spinal hematomas observed in Austin are also consistent with a trauma diagnosis. His spinal hematomas were on both the front and back of the spinal column and blood was found in several separate collections. Dr. Reece testified that because the blood was "spotty," it was unlikely that this was blood from Austin's head that had traveled down the spinal column. Tr.3 at 177. He noted that autopsies of infants who died from NAT had found tiny hemorrhages from blood vessels where they pass through the spinal vertebrae. During shaking, the spinal cord moves up and down within the spinal column, and shears off tiny blood vessels at the nerve roots. Tr.3 at 177-78. Doctor Wiznitzer analogized the presence of blood on both the back and front of the spinal cord and in several separate locations to finding four discrete bruises along the spinal column. Tr.3 at 29-30.

Doctor Uscinski agreed that Austin had an ankle fracture and isolated bleeding along the spinal cord. He could not explain Austin's ankle fracture. While he described the bleeding along the spinal cord as "not surprising," he did not explain whether the bleeding was consistent with his theory. Tr.3 at 329-30.

E. Doctor Uscincki's Explanation for Austin's Subdural Bleeding.

Unlike any of the other physicians skilled in reading CT scans, Dr. Uscinski identified two areas of bleeding, rather than just one, on the initial CT scan performed at

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873-75 (2009), filed as Res. Ex. OO. Severe coughing does not produce retinal hemorrhages. M. Goldman, et al., *Severe Cough and Retinal Hemorrhage in Infants and Young Children*, J. PEDIATR., 148: 835-36 (2006), filed as Res. Ex. PP. Forceful vomiting caused by pyloric stenosis also does not cause retinal hemorrhages. S. Herr, et al., *Does Valsalva Retinopathy Occur in Infants? An Initial Investigation in Infants with Vomiting Caused by Pyloric Stenosis*, PEDIATR. 113(6): 1658-61 (2004), filed as Res. Ex. QQ. In this study, the eyes of 89 infants who had experienced projectile vomiting were dilated and examined. None had hemorrhages detected. *Id.* at abstract.

RMH. He agreed that there was subdural bleeding in the interhemispheric space in the same location identified by the RMH and UVA radiologists, the treating physicians, respondent's experts, and Dr. Barnes. Tr.3 at 279, 283. He and Dr. Barnes disagreed with the rest of these physicians that this bleeding represented new blood. Tr.3 at 279-280.

However, Dr. Uscinski alone identified another area of bleeding in the tentorium (Tr. 3 at 259, 262),[85] identified as a bright white area on the scan, which he claimed represented new blood.[86] He acknowledged that he was the only physician who interpreted this bright white area on the June 15, 2004 RMH CT scan as representing active bleeding. Tr.3 at 335. He attributed the bleeding seen in the intrahemispheric space to the same mechanism that produced what he claimed was blood below and above the tentorium. He testified that the blood in the intrahemispheric space must have traveled from the tentorium to that location. Tr.3 at 346-50.

Because the second area of bleeding was below the tentorium and blood present below the tentorium was most likely from birth trauma,[87] he concluded that Austin had experienced a birth injury resulting in a chronic subdural hematoma. Little or no force is required to cause re-bleeding of a chronic hematoma; thus, this bleeding was not consistent with abuse occurring just prior to Austin's cardiopulmonary collapse on June 15, 2004. He believed that the bleeding caused by birth trauma continued, albeit at a minimal level, over the four months following Austin's birth, and cited to an unfiled Japanese study in support. Tr.3 at 255-56, 262-65, 334-35.[88]

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[85] Doctor Uscinski did not define the tentorium, but Dr. Wiznitzer testified that it is the part of the dura that separates the cerebral hemisphere from the posterior fossa (the brainstem and the cerebellum). Tr.3 at 132.

[86] He testified that fresh blood on a CT scan looks white because it contains calcium and iron, both of which are opaque to x-rays. As time passes, calcium and iron both leach out, and the blood looks darker. Tr.3 at 268.

[87] He initially testified that blood below the tentorium could occur only as the result of the stretching of the head and spine during birth (Tr.3 at 259, 263), but later appeared to agree with Drs. Wiznitzer and Reece (Tr.3 at 132, 210-11), that it might result from post-birth trauma as well, such as a blow to the back of the head (Tr.3 at 271).

[88] At least initially, Dr. Uscinski attributed Austin's injuries to bleeding caused by birth trauma and resulting chronic subdural hematoma. *See* Tr.3 at 255, 259, 271-72. Later in his testimony, Dr. Uscinski appeared to retreat from the position that Austin's injuries were the result of a chronic subdural hematoma:

Dr. Uscinski: The presence of old blood and fresher blood and blood below the tentorium makes this just about certain a chronic subdural hematoma.

Mr. McLaren: Okay.

Dr. Uscinski: I think Dr. - - well, yeah, it's a chronic subdural dating as far back as birth.

Doctor Wiznitzer summarized the numerous reasons for disagreeing with Dr. Uscinski's analysis and interpretations. His opinions are buttressed by the other scans performed on Austin. Additionally, there are other problems with Dr. Uscinski's assertions about a chronic subdural hematoma.

Asymptomatic subdural hematomas are present in a substantial number of newborns. Doctor Uscinski relied on the Rooks study[89] for this point (Tr.3 at 264), but even though he acknowledged the study's finding that the hematomas resolved by three months of age (Tr.3 at 332), he did not explain how this would affect his opinion in Austin's case. Thus, the Rooks study provided little support for Austin having an asymptomatic chronic subdural hematoma. *See* Tr.3 at 208; *see also* Case 2008b, Res. Ex. KK at 573 (few cases of acute subdural hemorrhage become chronic subdural hematomas). Doctor Uscinski could only point to facial bruising, overriding sutures, and head molding as signs of birth trauma in Austin, but he acknowledged that these are frequently present at birth. Tr.3 at 331-32. There is no indication in Austin's medical records that he had sustained any injury at birth.

The most significant reason for rejecting Dr. Uscinski's assertions that Austin had a chronic subdural hematoma in the area of the tentorium is that none of the other scans showed any blood at all in that area. The June 16, 2004 MRI had no trace of any blood in the tentorial area. Doctor Uscinski agreed that this MRI showed fluid below the tentorium, but that the fluid did not have a density consistent with blood (Tr.3 at 302-03). Doctor Wiznitzer testified that chronic subdural hematomas cannot disappear overnight. Tr.3 at 359-60. He commented that an MRI is very good at defining old and new subdural hematomas, and will actually show layering of the blood, old and new, within the same hematoma. The June 16 initial MRI scan of Austin's brain showed nothing in the tentorium, but showed the same bleeding in the interhemispheric fissure that Dr. Uscinski agreed was present on the CT scan. Tr.3 at 362. As Dr. Wiznitzer testified, "[t]he real finding persists from one day to the next. The misinterpretation is not present because it wasn't an accurate identification to start." Tr.3 at 362. He also noted that the June 20, 2004 CT scan failed to show any tentorial bleeding. Tr.3 at 363.

Mr. McLaren: Okay. And it's not your position that this is the cause of Austin's injuries because you're not giving an opinion with respect to the vaccine and the immediate encephalopathy following the vaccine. Is that right?

Dr. Uscinski: Right.

Tr.3 at 283-84.

[89] V. Rooks, et al., *Prevalence and Evolution of Intracranial Hemorrhage in Asymptomatic Term Infants*, AM. J. NEURORADIOL. e-published with no further citation provided (2008) ["Rooks"], filed as Res. Ex. Z. This study examined 101 asymptomatic term infants for intracranial bleeding using MRI and ultrasound within 72 hours of birth. Small subdural hematomas were found in 46 infants. In most of these infants, the hematomas resolved by one month of age, and all were resolved by three months of age *Id.* at abstract.

Doctor Wiznitzer relied more heavily on the MRI scan on June 16, 2004 rather than the CT scan performed the day before because an MRI provides a clearer picture. Tr.3 at 358; *see also* Case 2008b, Res. Ex. KK at 573. Another reason for relying more heavily on the MRI results is that, in children, a CT scan might sometimes show a bony artifact that appears to be blood but is not. Tr.3 at 358-59, 364. However, Dr. Wiznitzer testified that in this case, the bright signal in the tentorium to which Dr. Uscinski pointed was actually blood in the venous sinus, where blood would be expected. Tr.3 at 361.

Austin's appearance and behavior were not consistent with a chronic subdural hematoma present since birth. There was nothing in Austin's medical records that would suggest he experienced any birth injury. Tr.3 at 185. Children with true chronic subdural hematomas have a significant build up of fluid in their brains. At well baby checks, these children present with bulging fontanels and are irritable. Tr.3 at 370-71. The amount of blood shown on any of the scans was small and could not have accounted for the increases in Austin's head circumference. Tr.3 at 369-70.

Doctor Willson testified that the blood on the CT scan from June 15, 2004 was bright, representing new blood, consistent with an injury occurring on June 15, 2004. *See* Tr.2 at 162-63; Res. Ex. A at 426. Doctor Wiznitzer relied on the MRI results from June 16, 2004 to opine that Austin's injury was very recent. Tr.3 at 23, 46-47, 151. The timing of all of the other brain injuries observed is consistent with an inflicted injury occurring on June 15, 2004. Tr.3 at 67-68.

F. Conclusions Regarding the Expert Testimony and the Validity of the Trauma Diagnosis.

Respondent successfully demonstrated that Austin's trauma diagnosis was correct. Petitioner's attempts to rebut this diagnosis, and demonstrate that trauma was not the preponderant cause of Austin's injuries, were unconvincing. Doctor Uscinski's opinion that shaking alone could not cause Austin's injuries, and that Austin's injuries precluded a diagnosis of shaking plus impact, were persuasively rebutted by respondent's experts and the medical literature.

Additionally, Dr. Uscinski's opinion on the cause of Austin's injuries requires at least three separate explanations, and yet still does not fully explain Austin's clinical picture. To explain the subdural hematoma, he opined that Austin had an asymptomatic birth injury that periodically began to bleed again at around the same time as his unrelated cardiopulmonary collapse. Second, something unknown caused Austin to stop breathing. He experienced brain swelling, probably as a result of his cardiopulmonary collapse. Third, the retinal hemorrhages were caused by a combination of the brain swelling and the CPR he required. These opinions fail to address at least two of the physical injuries Austin suffered: the four separate areas of bleeding in his spine and his fractured ankle.

In contrast, trauma occurring shortly before Austin's cardiopulmonary collapse provides a unifying explanation for all of Austin's injuries. While a theory that unifies all of the evidence is not necessary to demonstrate entitlement (*see Knudsen v. Sec'y, HHS*, 35 F.3d 543, 550 (Fed. Cir. 1994)), petitioner's theory fails to account for several of Austin's injuries, and provides different and implausible explanations for each of the injuries it does attempt to explain. Respondent has explained the cause of each of Austin's injuries, and it happens to be the same cause. Regardless of the exact mechanism of injury, Austin's injuries are trauma injuries. Tr.3 at 27-31, 227. While Dr. Uscinski agreed that the subdural hematoma was a trauma injury, his explanation that it occurred at birth finds no support in the record or literature.

IV. Essential Factual Findings.

I find that Austin received a pertussis-containing vaccine on June 14, 2004. On June 15, 2004, within 72 hours of receipt of the vaccine, Austin was in a coma which initially met the diagnostic criteria to be considered a Table encephalopathy. However, I find by preponderant evidence that Austin's coma was the result of nonaccidental trauma.

In making these findings, I necessarily reject Mr. Huffman's account of leaving Austin alone for minutes, and returning to finding him blue and not breathing. I accept Dr. Willson's testimony that this was not biologically plausible. Mr. Huffman's testimony in this proceeding was consistent with his prior denials. His demeanor was calm, cool, and rehearsed. He failed to persuade me that his version of events was more likely than not what happened. His denials are insufficient, alone or in combination with petitioner's other evidence, to refute respondent's case.

I make no factual finding that Mr. Huffman shook, strangled, or otherwise injured Austin. As discussed below, I do find that Austin was the victim of NAT based on the other evidence before me: the medical records, the opinions of the treating physicians, and the expert testimony. Whether Mr. Huffman was responsible is not a necessary conclusion in this case.

V. Law Pertaining to Table Injuries and Alternate Cause.

A. Standards for Evaluating Table Causation Claims.

In every case under the Vaccine Act, a petitioner bears the burden to prove a vaccine-caused injury.[90] A petitioner can demonstrate causation in two ways, either as

[90] Other prerequisites for entitlement, found in § 300aa-11(c), are not at issue in this case.

a Table case or as an off-Table case[91]. The first requires that petitioner show: (1) receipt of a vaccine listed on the Table; (2) an injury listed on the Table for that vaccine; and (3) that the injury occurred within the time period specified for that injury and vaccine. § 300aa-11(c)(1)(c)(i); § 300aa-14, as revised by 42 C.F.R. § 100.3; *see also Walther v. Sec'y, HHS*, 485 F.3d 1146, 1149 (Fed. Cir. 2007). Although opinions interpreting the Vaccine Injury Table commonly say that proof of these elements entitles a petitioner to a presumption of causation, it may be more accurate to say that proof of these elements excuses petitioner from producing evidence of vaccine causation of the claimed injury. A special master finds the causal link as a matter of law when preponderant evidence establishes the Table requirements. *See Grant v. Sec'y, HHS*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). Causation is presumed, but some Table injuries such as an encephalopathy have exceptions that, when established by preponderant evidence, rebut the presumption.

Once a petitioner has established a *prima facie* case of a Table injury, respondent may prevail only when she demonstrates some other cause is more likely than not responsible for the vaccinee's injury. When the respondent seeks to prove the existence of a "factor unrelated" in a causation in fact case (*see* § 300aa-13(a)(1)(B)), respondent has the burden to do so by preponderant evidence. The Federal Circuit has determined that, once a petitioner has produced preponderant evidence of causation, the burden to show a factor unrelated to the administration of the vaccine under § 300aa-13(a)(1)(B) lies with respondent, and that respondent's burden for the factor unrelated mirrors the *prima facie* causation-in-fact case under the Act. *See Walther*, 485 F.3d at 1150; *Knudsen v. Sec'y, HHS*, 35 F.3d 543, 549 (Fed. Cir. 1994).

In a Table encephalopathy claim[92] based on a pertussis vaccination, a petitioner is entitled to compensation when the vaccinee experiences an encephalopathy meeting certain criteria within 72 hours of receipt of the vaccination. However, if there is preponderant evidence for a non-vaccine cause for the encephalopathy, the condition is not considered to be a Table encephalopathy. *See* 42 C.F.R. § 100.3(b)(2)(iii) ("An encephalopathy shall not be considered to be a condition set forth in the Table if . . . it is shown . . . that the encephalopathy was caused by . . . trauma.").[93] The same subsection of the Vaccine Injury Table that defines "encephalopathy" also enumerates the exceptions. *See* 42 C.F.R. § 100.3(b)(2).

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[91] This second method requires that petitioner show that the vaccine in fact caused the injury. A petitioner does this by establishing the three *Althen* factors by a preponderance of the evidence. *Althen v. Sec'y, HHS*, 418 F.3d 1274, 1278 (Fed. Cir. 2005). *See* §§ 300aa-11(c)(1)(c)(ii); § 300aa-13(a)(1). This method is no longer in issue in this case.

[92] The QAI define encephalopathy for purposes of the Vaccine Injury Table's applicability. *Terran,* 195 F.3d at 1307. The terms used in the Table are interpreted narrowly according to their Table definition, and not by a broader dictionary definition.

[93] I refer to a non-vaccine cause under the QAI as a "Table exception" herein to distinguish it from a "factor unrelated" under section 13.

This case presents the issue of the effect of the Table's trauma exception (42 C.F.R. § 100.3(b)(2)(iii)) on a Table encephalopathy claim. It is uncontested that Austin experienced a coma that otherwise met the requirements for a Table encephalopathy and that he experienced the encephalopathy within the requisite time frame. However, there is also preponderant evidence that trauma was the actual cause of the encephalopathy. What is unclear is how to evaluate the effect of that preponderant evidence. Does preponderant evidence of trauma act to prevent petitioner from establishing a *prima facie* Table injury in the first instance?[94] Or, does it simply shift the scales, which were tipped in favor of causation by evidence of a pertussis containing vaccine followed by a coma within 72 hours, to the opposite conclusion, by demonstrating that the vaccine was not responsible?

Some case law suggests the first formulation. In *Vant Erve v. Sec'y, HHS*, the special master found that, despite the fact that petitioners had demonstrated receipt of an appropriate vaccine, onset during the requisite time period, and evidence of an encephalopathy, respondent submitted preponderant evidence that a metabolic disturbance caused the encephalopathy. He concluded that this meant that petitioners were not entitled to a Table presumption in accordance with § 300aa-14(b)(3)(B). No. 92-341V, 1998 WL 887126, at *8 (Fed. Cl. Spec. Mstr. Dec. 3, 1998), *aff'd*, 43 Fed. Cl. 338 (1999), *aff'd*, 232 F.3d 914 (Fed. Cir. 2000). The special master characterized § 300aa-14(b)(3)(B) as part of the *prima facie* case, stating: "part (B) of 300aa-14(b)(3) provides another requirement for a 'Table Injury encephalopathy.'" *Id.* (emphasis omitted). He concluded that evidence of a metabolic disturbance as the cause of the encephalopathy indicated that the vaccinee had "not suffered an encephalopathy falling into the 'Table Injury' category."[95] *Id.* (emphasis omitted).

In *Leary v. Sec'y, HHS*, petitioners argued that their daughter had experienced significant aggravation of a Table encephalopathy, which would entitle them to a Table presumption of causation.[96] Petitioners put forth sufficient evidence of (i) receipt of a Table vaccine for which an encephalopathy was listed as a Table injury, (ii) evidence of an encephalopathy and of significant aggravation of the encephalopathy, and (iii) evidence of appropriate timing. The special master found petitioners were not entitled

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[94] This is the position respondent took earlier in this case. *See* Respondent's Status Report, filed June 2, 2008, at 2 ("If respondent proves, by a preponderance, that Austin suffered from non-accidental head trauma, … petitioner cannot prevail on a Table theory as she cannot make out a prima facie case. Petitioner would then have to show that the vaccination(s) actually caused Austin's injury.").

[95] He added, in a footnote, that "even if part B of 300aa-14(b)(3) did not exist, the ultimate result here would be no different" because respondent's evidence of a metabolic disturbance would successfully defeat the *prima facie* case when analyzed under § 300aa-13. *See* 1998 WL 887126 at *8 n.9.

[96] To demonstrate significant aggravation of a Table claim, petitioners must demonstrate receipt of a Table vaccine, significant aggravation of an injury listed on the Table, and onset within the time period required by the Table. *See Leary*, 2003 WL 1699450 at *23 (citing § 300aa-11(c)(1)(C)(i) and § 300aa-13(a)(1)(A)).

to a Table presumption because respondent presented preponderant evidence that the vaccinee's encephalopathy was caused by a metabolic disturbance. No. 94-626V, 2003 WL 1699450, at *26 (Fed. Cl. Spec. Mstr. Mar. 11, 2003). "[T]he special master must refer to § 300aa-14(b)(3) to determine at the outset if a petitioner presents with either the initial onset of a Table encephalopathy or the significant aggravation of a Table encephalopathy." *Leary*, 2003 WL 1699450, at *25.

In other cases, it is less clear which provision of the Vaccine Act was used to evaluate the Table injury. In *Lord v. Sec'y, HHS*, petitioners alleged that a DPT vaccination caused the vaccinee to sustain an encephalopathy within a time frame that would qualify the event as a Table encephalopathy. Respondent argued that a metabolic disorder was the cause of the vaccinee's encephalopathy, and that petitioner was not entitled to a Table presumption. No. 90-1630V, 1997 WL 588999, at *1 (Fed. Cl. Spec. Mstr. Aug. 21, 1997). The special master set forth the standard for proving a factor unrelated as both the standard in § 300aa-13(a)(1)(B), and also the "related provision" in § 300aa-14(b)(3)(B), the predecessor to the QAI. *Lord*, 1997 WL 588999, at *2. She explained that she cited to both "because they are relevant to the allegation that a metabolic condition is the more likely cause" of the encephalopathy at issue. *Id.* She then concluded that petitioner was "entitled to a statutory presumption of a vaccine-related cause of his injury" and shifted the burden to respondent to prove a factor unrelated, stating: "[R]espondent's statutory burden of proof is to demonstrate its claim by a preponderance of evidence and is subject to 13(a)(2)[97] and 14(b)(3)(B) relating to proof of factors unrelated." *Id.* at 2-3. She also noted that "[s]ections 13(a)(2) and 14(b)(3)(B) require no more than a showing that a metabolic disorder exists and that more likely than not it caused the encephalopathy."[98] *Id.* at 7. The special master did not discuss how § 300aa-13(a)(1)(B) and § 300aa-14(b)(3)(B) are distinguishable from each other, nor whether it was more appropriate to apply one before the other. This analysis suggests she regarded the provisions as identical, at least as they applied in that case.

Schirmer-Guzman v. Sec'y, HHS, presented a factual scenario similar to the one at issue here: receipt of a pertussis-containing vaccine, symptoms of encephalopathy, onset within 72 hours, and evidence of trauma as the cause. No. 99-998V, 2005 WL 6122530 (Fed. Cl. Spec. Mstr. July 20, 2005). The decision did not directly address whether applying one provision over the other to analyze alternative cause evidence

[97] Section 300aa-13(a)(2) provides a definition of "factors unrelated to the administration of the vaccine" that mirrors, in part, the provisions of § 300aa-14(b)(3)(B).

[98] The special master here cited *Dieudonne v. Sec'y, HHS*, No. 90-1695V, 1996 WL 718286 (Fed. Cl. Spec. Mstr. Nov. 27, 1996), and *Lassiter v. Sec'y, HHS*, No. 90-2036V, 1996 WL 749708 (Fed. Cl. Spec. Mstr. Dec. 17, 1996). The analysis of the interplay of § 300aa-13(a)(2) and § 300aa-14(b)(3)(B) in these two cases was nearly identical to that in *Lord*.

was appropriate, other than to opine that § 300aa-14(b)(3)(B)[99] "merely restates in specific terms the proposition" set forth in § 300aa-13(a)(2). *Id.* at *3. Because the special master viewed § 300aa-14(b)(3)(B) and the QAI as a restatement of § 300aa-13(a)(2), he analyzed the evidence under *Knudsen*, 35 F.3d 543, and § 300aa-13(a)(2). *Schirmer-Guzman,* 2005 WL 6122530, at *16-17.

B. Analyzing Causation in This Case.

When preponderant evidence of trauma is present in a Table encephalopathy claim, the controversy over which provision of the Act applies could be a distinction without a difference.[100] Indeed, several of the cases cited above seem to treat the analysis under § 300aa-13 and the QAI as the same, because the ultimate outcome is the same. In this case, regardless of whether I conclude that the preponderant evidence of trauma as the cause of Austin's injuries prevents petitioner from establishing a *prima facie* case of a Table injury, or that it establishes a factor unrelated under § 300aa-13, petitioner's claim for compensation fails.

However, the basis for my conclusion could influence the evaluation of petitioner's attempt to undercut the application of the trauma exception with Dr. Uscinski's testimony. If I conclude that the trauma diagnosis and evidence in support of that diagnosis prevents petitioner from establishing a *prima facie* Table case, petitioner's attempts to undercut respondent's evidence of trauma may be insufficient. One could argue that petitioner must also produce evidence of vaccine causation in order to prevail. That is, if one views the Table as excusing petitioner from the requirement to produce evidence of vaccine causation, one could view evidence of the trauma exception as requiring petitioner to produce actual causation evidence, not just evidence negating trauma, in order to prevail. On the other hand, if I conclude that petitioner presented a *prima facie* Table case, followed by respondent producing preponderant evidence of trauma, petitioner's efforts to undercut that diagnosis and the evidence in support of the diagnosis could, if successful, restore the Table presumption of causation. This would excuse petitioner from presenting evidence of vaccine causation, as, once again, it would be presumed. In this case, I need not resolve this esoteric issue.

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[99] The special master cites § 300aa-14(b)(3)(B) in his opinion, but at the time of the decision, the QAI superseded the text of § 300aa-14(b)(3)(B). *See* 42 C.F.R. § 100.3(c)(1). The trauma exception appeared in both Act provisions.

[100] It is a distinction that might have collateral consequences, however. Whether petitioner earns the presumption could be particularly relevant in a case where the evidence of alternative cause is so overwhelming that it suggests the petitioner lacked good faith or a reasonable basis to bring or sustain the case. This might impact on whether fees and costs could be awarded to an unsuccessful litigant. *See* § 300aa-15(e)(1). For example, consider the case of a Vaccine Act petitioner who, on returning home from receiving a DPT vaccination, is struck by a car and develops an encephalopathy. If a special master holds that petitioner demonstrated a *prima facie* Table case (legal cause), petitioner would have a strong argument that his case meets the good faith and reasonable basis standard for attorney fees.

In *Leary*, the special master analyzed respondent's evidence of a metabolic disturbance under both the Table's exception and the "factor unrelated" standard of § 300aa-13, finding that even if he awarded petitioners a Table presumption, their claim would still fail. *Leary*, 2003 WL 1699450, at *26, 33. I do likewise. I have considered the evidence in this case under both the standards set forth in the QAI and under § 300aa-13. Respondent produced preponderant evidence that the encephalopathy was due to trauma. The presence of preponderant evidence of trauma defeats the Table injury claim, either by preventing petitioner from establishing a *prima facie* case in the first instance, or by demonstrating that the vaccinee's injury was caused by a factor unrelated.

For purposes of evaluating Dr. Uscinski's testimony and the evidence upon which he relied, I will assume that petitioner established a *prima facie* Table encephalopathy and apply the factor unrelated analysis under § 300aa-13(a)(2). Nevertheless, respondent successfully established trauma as a factor unrelated by preponderant evidence. Therefore, I treat Dr. Uscinski's efforts to demonstrate that shaking was unlikely as the mechanism of injury in this case as an attempt to undercut the trauma diagnosis. Thus, I do not require petitioner to counter the trauma exception evidence with evidence that the vaccine actually caused Austin's injuries.

This approach is consistent with the evaluation of off-Table injuries. In many cause in fact cases, respondent does not advance her own evidence of alternate cause. Rather, she produces evidence that tends to undercut one or more of the *Althen* prongs upon which petitioner relies. Regardless of the method of evaluation, the burden of establishing the trauma exception falls on respondent. *See Hanlon v. Sec'y, HHS,* 191 F.3d 1344, 1348 (Fed. Cir. 1999) (applying the § 300aa-13 factor unrelated analysis to rebut a Table injury in the absence of the listing of that particular factor (a genetic cause) as an exception to the Table at that time).

Petitioner alleges that Austin's encephalopathy qualifies as a Table injury because she demonstrated: (1) receipt of a vaccine containing pertussis; (2) development of an encephalopathy; and (3) onset of that condition within 72 hours. *See* Pet. Post-Hearing Br. at 1. Though she did not cite it, petitioner was assuming the applicability of § 300aa-14, as revised by 42 C.F.R. § 100.3, for the Table encephalopathy portion of her initial claim. By demonstrating Austin's receipt of a pertussis-containing vaccine within 72 hours of his cardiopulmonary collapse and resultant coma, Ms. Huffman ostensibly established that Austin sustained a Table injury. However, the presumption of causation contained in the Vaccine Injury Table is a rebuttable one, and the same exhibit Ms. Huffman filed to demonstrate a Table encephalopathy within the requisite time frame also established that Austin's encephalopathy was, more likely than not, caused by trauma. *See* Pet. Ex. 8.

Although the facts of Austin's vaccination and physical condition were not contested, the treating physicians' trauma diagnosis was. One could argue that as petitioner's own evidence both raised and refuted a Table injury claim, respondent had no burden to produce any additional evidence. Given the Federal Circuit's admonition

to give weight to the opinions of treating physicians (*see Andreu*, 569 F.3d at 1375) this argument has considerable allure. But the evidence petitioner produced when proceeding *pro se* included statements from several physicians who attributed Austin's injury to causes other than trauma. Although petitioner ultimately did not rely on several of them, and I have found that the others were unreliable or unpersuasive, by filing them, petitioner produced some evidence to question the trauma diagnosis.

This in turn led respondent to produce Investigator Fetterman, and Drs. Rust, Willson, Wiznitzer, and Reece to support the trauma diagnosis. They produced preponderant evidence of trauma as the cause of Austin's injuries. Respondent agreed with petitioner that this evidence of alternate (non-vaccine) cause for Austin's condition had to be evaluated under the standard set forth in § 300aa-13(a). *See* Joint Status Report field Dec. 29, 2008. As indicated earlier, I am not satisfied that the parties' analysis of which portion of the Vaccine Act governs respondent's burden is correct, but I will evaluate respondent's evidence this way in light of her concession.

Whether respondent must comply with the *Althen* standard when producing evidence of a Table exception or a factor unrelated is not clear. As Special Master Moran has observed, *Knudsen* requires that "'the standards that apply to a petitioner's proof of actual causation in fact in off-table cases should be the same as those that apply to the government's proof of alternative actual causation in fact.'" *Heinzelman v. Sec'y, HHS*, No. 07-01V, 2008 WL 5479123, at *17 (Fed. Cl. Spec. Mstr. Dec. 11, 2008), *mot. for rev. docketed* (Fed. Cl. Jan. 6, 2011) (quoting *Knudsen*, 35 F. 3d at 549). *Knudsen* thus likely requires respondent to satisfy the *Althen* framework for analyzing and evaluating causation evidence. *See id.*

Respondent produced preponderant evidence under *Althen* that Austin's injuries were caused by trauma. *See* 418 F.3d at 1278. The treating and non-treating experts provided a medical theory by which nonaccidental trauma could have caused the injuries present (the "can cause" question), successfully linked Austin's presentation to nonaccidental trauma by a plethora of circumstantial evidence (the "did cause" question), and demonstrated that Austin's injuries likely occurred shortly before his cardiopulmonary collapse, which is consistent with the time frame expected for collapse from nonaccidental trauma. Thus respondent's evidence passes muster under both the trauma exception of § 300aa-14(b)(3)(B), as revised by 42 C.F.R. § 100.3(b)(2)(iii), and under the factor unrelated analysis of § 300aa-13(a)(1)(B).

This finding does not end the causation inquiry, because petitioner sought to undercut the evidence supporting the trauma exception. There are at least two ways she could have done so. First, she could have produced evidence of vaccine causation of Austin's injuries to establish a cause in fact case under § 300aa-13(a)(1) and *Althen,* but evidence of a vaccine injury was virtually non-existent.[101] What petitioner attempted

[101] I am not ignoring Dr. Barnes' suggestion of a vaccine-linked infection (*see* Pet. Ex. 12 at 1), but as no evidence of any infection was developed during Austin's hospitalization, the factual predicate for this opinion was lacking. That leaves Dr. Buttram's "smoldering encephalitis." *See* Pet. Ex. 9 at 14. Aside

to do was to undercut the theory on which the trauma diagnosis was based. By analogy, what petitioner attempted to do in this case was what respondent often attempts to do in cause in fact Vaccine Act cases—challenge the evidence supporting one or more of the *Althen* factors to ensure that the quantum of evidence does not reach the preponderance standard. *See, e.g.*, *de Bazan v. Sec'y, HHS*, 539 F.3d 1347, 1353 (Fed. Cir. 2008).

Petitioner failed.[102] Doctor Uscinski's testimony, the scientific studies he cited in support, and the evidence provided by Drs. Barnes and Gardner were insufficient for three reasons. First, I find ample support in the strong circumstantial evidence presented for the majority medical opinion that infants presenting with Austin's constellation of injuries—subdural or subarachnoid bleeding, diffuse bilateral retinal hemorrhages, brain injury, and fractures—are the victims of trauma. When no history consistent with accidental trauma is provided, a diagnosis of NAT is the most likely explanation for the injuries Austin displayed.

Second, even if the Duhaime and Prange studies were stronger evidence than I found them to be that shaking alone could not account for Austin's injuries, there was still preponderant evidence that Austin's injuries were the result of trauma. Furthermore, the Duhaime and Prange studies, as well as several others, indicated that shaking plus an impact could produce sufficient deceleration to cause subdural hematomas and concussive brain injury. The absence of external evidence of impact is not, as the Duhaime study (Pet. Ex. 56 at 410) and the Case 2008b paper (Res. Ex. KK at 571-72) indicated, evidence that no impact occurred. The evidence of injury to both the front and the back of Austin's brain (*see* Tr.3 at 59-60) suggests that there was a physical injury to his brain. To defeat the presumption of vaccine causation, evidence of trauma does not have to include the precise mechanism of injury. 42 C.F.R. 100.3(b)(2)(iii); *Knudsen*, 35 F.3d at 549 (interpreting § 300aa-13(a)(2)). Third, Dr. Uscinski himself attributed Austin's injuries to birth trauma, coupled with vigorous CPR and edema induced by anoxia. Section 300aa-13(a)(2)(B) specifically includes "birth trauma" as a factor unrelated. If I believe Dr. Uscinski, the trauma exception still applies.

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from the fact that petitioner did not argue this evidence, there is no evidence that Austin's scans were consistent with encephalitis, and Dr. Wiznitzer provided unrebutted evidence that a "smoldering encephalitis" is not accepted as a medical diagnosis. Tr.3 at 68-69; *see also id.* at 186 (Dr. Reece opining that he had never heard the term or read it in the medical literature).

[102] The somewhat muddled nature of petitioner's attempts to undercut the NAT diagnosis stem from the fact that much of her evidence was developed in an effort to prevent Chris Huffman's conviction for injuring Austin. Thus the evidence focused on showing that anything other than an inflicted injury could be responsible—birth trauma, vaccines, Vitamin K deficiency, infection, etc.—as well as on indicating that shaking could not produce the injuries Austin sustained. This has been Dr. Uscinski's focus in the many criminal trials in which he has testified as well. He clearly attributed Austin's brain bleeding to trauma, and only in response to leading questions much later in his testimony did he indicate otherwise. *See supra* note 88.

VI. Conclusion.

While petitioner demonstrated that Austin experienced an encephalopathy within 72 hours of receipt of a pertussis-containing vaccine, her claim fails. Respondent successfully proved by preponderant evidence that Austin's injuries were caused by trauma. Petitioner has failed to establish entitlement to compensation and the petition is **DENIED**. In the absence of a motion for review filed pursuant to RCRC, Appendix B, the clerk is directed to enter judgment accordingly.[103]

IT IS SO ORDERED.

s/Denise K. Vowell
Denise K. Vowell
Special Master

[103] Pursuant to Vaccine Rule 11(a), entry of judgment can be expedited by each party's filing a notice renouncing the right to seek review.