In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 07-0810V
(Not for publication)

PETER and CHERIE NUTTALL, as the legal representatives of their minor son, N.N.,	*	
Petitioners,	*	Filed: January 20, 2015
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES	*	Vaccine Act Entitlement; Table Injury; MMR, DTaP/ Encephalitis.
Respondent.	*	

Curtis R. Webb, Twin Falls, ID, for Petitioners.
Voris E. Johnson, U.S. Department of Justice, Washington, DC, for Respondent.

DECISION

HASTINGS, Special Master

This is an action in which Petitioners, Peter and Cherie Nuttall, seek an award under the National Vaccine Injury Compensation Program (hereinafter "the Program[1]), on account of their son N.N.'s severe neurologic disorder, which they believe was caused by "encephalitis" triggered by either a measles, mumps, rubella ("MMR") vaccination or a diphtheria-tetanus-acellular pertussis ("DTaP") vaccination, each administered to their son on November 22, 2004. For the reasons set forth below, I conclude that Petitioners are not entitled to an award.

I

THE APPLICABLE STATUTORY SCHEME

Under the National Vaccine Injury Compensation Program, compensation awards are made to individuals who have suffered injuries after receiving vaccines. In general, to gain an

[1] The applicable statutory provisions defining the Program are found at 42 U.S.C. § 300aa-10 *et seq.* (2006 ed.). Hereinafter, for ease of citation, all "§" references will be to 42 U.S.C. (2006 ed.).

award, a petitioner must make a number of factual demonstrations, including showing that an individual received a vaccination covered by the statute; received it in the United States; suffered a serious, long-standing injury; and has received no previous award or settlement on account of the injury. Finally – and the key question in most cases under the Program – the petitioner must also establish a *causal link* between the vaccination and the injury. In some cases, such as the present one, the petitioner may seek simply to demonstrate the occurrence of what has been called a "Table Injury." That is, it may be shown that the vaccine recipient suffered an injury of the type enumerated in the "Vaccine Injury Table," corresponding to the vaccination in question, within an applicable time period following the vaccination also specified in the Table. If so, the Table Injury is *presumed* to have been caused by the vaccination, and the petitioner is automatically entitled to compensation, unless it is affirmatively shown by the Respondent that the injury was caused by some factor other than the vaccination. (§ 300aa-13(a)(1)(A); § 300 aa-11(c)(1)(C)(i); § 300aa-14(a); § 300aa-13(a)(1)(B).)

As relevant here, the applicable Vaccine Injury Table lists "encephalitis" as a compensable injury, if the first symptoms thereof occur within 5 to 15 days of a measles, mumps and rubella ("MMR") vaccination, or within 72 hours of a diphtheria-tetanus-acellular pertussis ("DTaP") vaccination. (§ 300aa-14(a), as amended by 42 CFR § 100.3.)

Alternatively, if no injury falling within the Table can be shown, the petitioner may gain an award by instead showing that the vaccine recipient's injury or death was caused by the vaccination in question.[2] (§ 300aa-13(a)(1)(A); § 300aa-11(c)(1)(C)(ii).)

II

THE OMNIBUS AUTISM PROCEEDING ("OAP")

A. *General*

This case is one of more than 5,400 cases filed under the Program in which petitioners alleged that conditions known as "autism" or "autism spectrum disorders" ["ASD"] were caused by one or more vaccinations. A special proceeding known as the Omnibus Autism Proceeding ("OAP") was developed to manage these cases within the Office of Special Masters ("OSM"). A detailed history of the controversy regarding vaccines and autism, along with a history of the development of the OAP, was set forth in the six entitlement decisions issued by three special masters as "test cases" for two theories of causation litigated in the OAP (see cases cited below), and will only be summarized here.

A group called the Petitioners' Steering Committee was formed in 2002 by the many attorneys who represented Vaccine Act petitioners who raised autism-related claims. Their responsibility was to develop any available evidence indicating that vaccines could contribute to

[2] In this case Petitioners have opted *not* to pursue this alternative "cause-in-fact" method of demonstrating causation. Petitioners indicated at the expert hearing that they were not attempting to prove, and were thereby waiving, any "off-Table" injury claim. (Tr. 8.) I note, however, that even if this case were evaluated under a cause-in-fact analysis, for the reasons discussed in Section VIII of this decision, the outcome would be the same.

causing autism, and eventually present that evidence in a series of "test cases," exploring the issue of whether vaccines could cause autism, and, if so, in what circumstances. Ultimately, the PSC selected a group of attorneys to present evidence in two different groups of "test cases" during many weeks of trial in 2007 and 2008. In the six test cases, the PSC presented two separate theories on the causation of ASDs. The first theory alleged that the *measles* portion of the measles, mumps, rubella (MMR) vaccine could cause ASDs. The second theory alleged that the mercury contained in *thimerosal-containing vaccines* could directly affect an infant's brain, thereby substantially contributing to the causation of ASD.

Decisions in each of the three test cases pertaining to the PSC's *first* theory rejected the petitioners' causation theories. *Cedillo v. HHS*, No. 98-916V, 2009 WL 331968 (Fed. Cl. Spec. Mstr. Feb. 12, 2009), *aff'd*, 89 Fed. Cl. 158 (2009), *aff'd*, 617 F.3d 1328 (Fed. Cir. 2010); *Hazlehurst v. HHS*, No. 03-654V, 2009 WL 332306 (Fed. Cl. Spec. Mstr. Feb. 12, 2009), *aff'd* 88 Fed. Cl. 473 (2009), *aff'd*, 604 F.3d 1343 (Fed. Cir. 2010); *Synder v. HHS*, No. 01-162V, 2009 WL 332044 (Fed. Cl. Spec. Mstr. Feb. 12, 2009), *aff'd*, 88 Fed. Cl. 706 (2009).[3] Decisions in each of the three "test cases" pertaining to the PSC's *second* theory also rejected the petitioners' causation theories, and the petitioners in each of those three cases chose not to appeal. *Dwyer v. HHS*, No. 03-1202V, 2010 WL 892250 (Fed. Cl. Spec. Mstr. Mar. 12, 2010); *King v. HHS*, No. 03-584V, 2010 WL 892296 (Fed. Cl. Spec. Mstr. Mar 12, 2010); *Mead v. HHS*, No. 03-215V, 2010 WL 892248 (Fed. Cl. Spec. Mstr. Mar. 12, 2010).

Thus, the proceedings in the six "test cases" concluded in 2010. Thereafter, the Petitioners in this case, and the petitioners in other cases within the OAP, were instructed to decide how to proceed with their own claims. The vast majority of those autism petitioners elected either to withdraw their claims or, more commonly, to request that the special master presiding over their case decide their case on the written record, uniformly resulting in a decision rejecting the petitioner's claim for lack of support. However, a small minority of the autism petitioners have elected to continue to pursue their cases, seeking other causation theories and/or other expert witnesses. A few such cases have gone to trial before a special master, and in the cases of this type decided thus far, all have resulted in rejection of petitioners' claims that vaccines played a role in causing their child's autism. In none of the post-test case rulings has a special master or judge found any merit in an allegation that any vaccine can contribute to causing autism.

B. Relevance of OAP to this case

This case, however, is quite *different* from the cases cited in Section II(A) of this Decision. The issue here is *not* whether vaccines *caused* N.N.'s severe neurological disorder, which has been characterized as a form of autism. In this case, as noted above, the question is whether N.N. suffered a *Table Injury*, namely "encephalitis," with the first symptoms of that encephalitis occurring within a Table time period after vaccination. I ultimately conclude below that N.N. did *not* suffer an "encephalitis" at all, so that he did not suffer a Table Encephalitis. But it should be stressed that the evidence upon which I have relied in making my decision is limited to the evidence set forth in *this case*, on the issue of whether N.N.'s *MRIs show the existence of a previous encephalitis*. I include this description of the OAP *only* to show why this

[3] The petitioners in *Snyder* did not appeal the decision of the U.S. Court of Federal Claims.

case, filed in 2007, was not processed in the usual manner of non-autism Program cases. Because this case involved a child who had been diagnosed with a form of autism, the processing of this case was *delayed*, at Petitioners' request, along with the other 5,000 autism cases, to await the final outcome of the autism "test cases". When Petitioners filed this case in 2007, it should be noted, they entitled their petition "Petition for Vaccine Compensation--Omnibus Autism Proceeding--MMR Vaccine Causation" (Pet. filed Nov. 19, 2007, p. 1). Their petition did *not* allege a Table Injury at all, but alleged that N.N.'s autism was vaccine-caused. Then, when the "test cases" were finalized in 2010, individual petitioners such as the Nuttals were given a generous period of time to decide whether to abandon their claims or to develop a theory of their own case.[4]

Only after Petitioners filed their Amended Petition on November 28, 2011, did the focus of this case change to a *Table Injury*, namely an alleged Table Injury Encephalitis associated with N.N.'s vaccinations of November 22, 2004.

Thus, the *sole* issue that I address in this case does *not* concern whether autism can be *caused* by the vaccination that N.N. received, but *only* whether N.N. suffered an *encephalitis*, with the first symptoms of that encephalitis arising within a Table time period after his vaccinations.

III

FACTS AND PROCEDURAL HISTORY OF THIS CASE

A. Facts appearing in medical records

N.N. was born on September 11, 2000. (Ex. 3, p. 27.)[5] He was delivered via caesarean section, weighing 8 pounds at birth. (*Id*.)

Between September 22, 2000, and June 27, 2003, N.N. was seen by Dr. John Wynn, a pediatrician, for well-child visits and a variety of ailments, including cough, nasal congestion, runny nose, wheezing, ear pain, loose stool, diarrhea, rash, eczema, fever, bronchiolitis, otitis media, atopic dermatitis, rhinitis, concern that he might be hyperactive, concern that he was not sleeping or eating well, and upper respiratory infections. (Ex. 2, pp. 1-13.) Dr. Wynn also saw N.N. on February 7, 2001, for possible seizure activity while he was falling asleep. (Ex. 2, p. 7.) Dr. Wynn's impression was "probable mild clonic jerking with transition to sleep phase." (*Id*.)

As a result of the family's move from Salt Lake City to Las Vegas, N.N. began seeing a new pediatrician, Dr. Ivana Winkler. N.N. saw Dr. Winkler on November 5, 2004, with complaints of a sore throat, runny nose, sneezing, low fever, cough and decreased appetite. (Ex.

[4] Thus, after the initially assigned special master, Special Master Moran, decided an initial factual issue in his Findings of Fact filed on March 6, 2009, the case was then reassigned to Special Master Campbell-Smith, because she was one of the three special masters handling the autism cases.

[5] Exhibits filed by Petitioners are identified by number, while exhibits filed by Respondent are identified by letter. In addition, certain exhibits presented for the first time at the expert hearing in this case – referred to as "trial exhibits" – are also identified by number.

3, p. 1.) Dr. Winkler diagnosed N.N. as having, among other conditions, an upper respiratory infection. (*Id*.)

On November 22, 2004, N.N. saw Dr. Winkler for a well-child visit. (Ex. 3, p. 2.) At this visit, N.N. received DTaP and MMR vaccinations, as well as an inactivated poliovirus vaccination ("IPV"). (*Id*.) There is no evidence in the contemporaneous medical records indicating that N.N. had an adverse reaction to these vaccines, or that medical attention was sought in the weeks following the vaccinations.

N.N. continued to see Dr. Winkler for ailments during January of 2005. (Ex. 3, pp. 3-5.) He was diagnosed with an upper respiratory infection and prescribed amoxicillin on January 11, 2005. (Ex. 3, p. 4.)

On March 1, 2005, N.N. saw Dr. Winkler for complaints including frequent urination, loss of appetite, lethargy, watery diarrhea, and a concern about a habit of holding his jaw. (Ex. 3, p. 6.) N.N. saw Dr. Winkler again on May 26, 2005, for complaints of lethargy, loss of appetite, sneezing, and sniffling. (*Id*. at 7) At this visit, it was noted that N.N. was exhibiting head-banging episodes, and that he experienced mood-swings and self-biting. (*Id*.)

On June 14, 2005, Dr. Winkler saw N.N. for complaints of regression and lack of speech. (Ex. 3, p. 8.) A history taken at that visit indicates that N.N. "used to go weeks without saying much," "never talked like a normal kid," and "wasn't communicating normally earlier but [was] much worse [during the] last two weeks." (*Id*.) Dr. Winkler diagnosed N.N. with speech and developmental delay with acute regression, and indicated the possibility of an ASD. (*Id*.)

On June 8, 2005, and June 15, 2005, N.N. was seen by Dr. Joan Carapucci, a child psychiatrist, for an evaluation of possible bipolar disorder. (Ex. 5, p. 6.) Dr. Carapucci concluded that N.N. "certainly meets the criteria for Autism Spectrum Disorder." (*Id*., p. 5.) Dr. Carapucci diagnosed N.N. with Childhood Disintegrative Disorder ("CDD"), a form of autism, and recommended a referral to a neurologist. (*Id*.)

On June 24, 2005, N.N. was seen by Dr. Winkler for a follow-up regarding his lab work. (Ex. 3, p. 9.) Notes from that visit indicate that N.N. made minimal eye contact and talked little. (*Id*.) His family history was noted to include a maternal cousin with autism. (*Id*.) Dr. Winkler referred N.N. for audiology and neurology consultations. (*Id*.)

A CT scan of N.N.'s brain performed on July 18, 2005, was interpreted as normal. (Ex. 6, p. 11.)

On August 16, 2005, Dr. Alfreda Maller, a neurologist, evaluated N.N. for global developmental regression. (Ex. 6, p. 1.) Dr. Maller's impression was that N.N. had "severe developmental regression, highly consistent with childhood disintegrative disorder." (Ex. 6, p. 2.) Dr. Maller ordered an EEG and a metabolic work-up. (*Id*.)

On September 1, 2005, N.N. was seen by Dr. Howard Baron, a gastroenterologist, for regressive behavior and large, foul-smelling stools. (Ex. 7, p. 1.) Dr. Baron noted that "the mother recalls that [N.N.] was nearly potty trained prior to his regressive symptoms," but that he had reverted to being diapered. (*Id*., p. 1-2.) Dr. Baron indicated that N.N. might have a central

nervous system disorder or metabolic disorder. (*Id*., pp. 4-5.) He recommended metabolic and chemistry panels. (*Id*. at 4.)

On November 9, 2005, an MRI study was conducted. (Ex. 6, p. 25.) It was interpreted as normal by Dr. Brett Hewell on December 19, 2005. (*Id*.)

From that point forward, N.N. visited multiple specialists, including a geneticist (Ex. 12, p. 1), an autism clinic (Ex. 13, p. 1), and an adolescent psychiatrist (Ex. 15, p. 3). At various points he was prescribed Seroquel (Ex. 14, p. 1) and Depakote (Ex. 14, p. 8), as well as Adderall, Risperdal and Clonidine (Ex. 15, p. 3). He had multiple diagnoses in addition to the previously diagnosed CDD, including "child psychosis" (Ex. 11, p. 11), mental retardation (Ex. 13, p. 3), ADD (Ex. 14, pp. 8-9), and cognitive disorder – not otherwise specified ("NOS") (*Id*.). He was hospitalized for self-injurious behavior from August 12, 2006, to August 18, 2006. (Ex. 14, pp. 1-3.) A second CT scan was conducted on August 3, 2009. (Ex. 40.) Like his 2005 CT scan, it was read as normal. (*Id*.)

At some point, N.N.'s family moved to Georgia and he began seeing Dr. Asma Fischer. (Ex. 23, p. 1.) N.N. was referred to Dr. Fischer by Dr. Shuman, Petitioners' expert in this case. (*Id*.) On September 27, 2011, Dr. Fischer recorded an impression including "encephalopathy status post MMR," and scheduled an MRI for the following month. (*Id*., p. 2.) Dr. Fischer's September 27 record indicates that petitioners' expert in this case, Dr. Shuman, had been in contact with her and advised her that a previous MRI "revealed abnormalities."[6] (*Id.*, p. 1.)

Ultimately, a second MRI study was conducted on October 24, 2011, and interpreted by Dr. Jose Bauza. (Ex. 25, p. 1.) Dr. Bauza reported bilateral hyperintensities within the hippocampus and the fornices. (*Id*.) His report indicated that "the findings described for the hippocampal is in keeping with the patient's history of previous encephalitis, as well as the hyperintensity within the petrigonal region." (*Id*.) Dr. Fischer took note of Dr. Bauza's report on October 25, 2011, and included "post limbic encephalitis" to her "impression" of N.N.'s case for the first time. (Ex. 23, p. 3.)

B. Procedural history

On November 19, 2007, Mr. and Mrs. Nuttall filed a petition on behalf of their minor son, N.N., under the Vaccine Act. (§§ 300aa-1 to 300aa-34.) The petition, accompanied by medical records, affidavits, and photographs marked as Exhibits 1 to 16, alleged that N.N. developed Child Disintegrative Disorder caused by his MMR vaccination on November 22, 2004. (Pet. at ¶¶ 2-4.) After reviewing the petition and accompanying documents, representatives of the Secretary of the Health and Human Services ("HHS") concluded that this case was not appropriate for compensation under the Vaccine Act, issuing a "Rule 4 report" on February 27, 2008. (ECF No. 6.)

A fact hearing was held by Special Master Christian Moran on June 24, 2008, in Las Vegas, Nevada. (ECF No. 17.) The purpose of the hearing was to resolve factual disputes regarding the onset of N.N.'s condition in light of conflicts between the medical records and Petitioners' claims. Several witnesses testified, including N.N.'s parents, two grandparents, and

[6] Based on the record of this case, it appears that the only previous MRI was the study of November 9, 2005, interpreted by Dr. Hewell as normal.

a babysitter. (ECF No. 24, pp. 1-5.) On March 6, 2009, Special Master Moran issued a document entitled Unpublished Findings of Fact, resolving the inconsistencies and addressing the timing and nature of N.N.'s regression relative to his vaccinations of November 22, 2004. (ECF No. 24.) Special Master Moran's findings of fact answered a series of seven specific questions posed by the parties. (ECF No. 24, p. 2.)

Special Master Moran's fact findings concluded that certain of N.N.'s symptoms, though not mentioned in the contemporary medical records, did arise for the first time in the time period following soon after his vaccinations of November 22, 2004. In particular, Special Master Moran found that N.N.'s "ability to use language started to decrease in November 2004." (Findings, p. 7), and that his loss of speech skills also began in November 2004 (*id.* at 8). Special Master Moran also found that N.N. "experienced an episode of separation anxiety" on November 30, 2004. (*Id*. at 8.)

Subsequently, the case was stayed pending the outcome of the Omnibus Autism Proceeding ("OAP") "test cases" addressing the theory that the MMR vaccination can contribute to causing autism. (ECF No. 25.) On November 7, 2011, the case was reassigned to Chief Special Master Patricia Campbell-Smith (ECF No. 41), one of the three special masters handling the autism cases (the undersigned and Special Master Vowell were the other two), and Petitioners filed an amended petition on November 28, 2011 (ECF No. 45). The amended petition alleged that N.N.'s CDD was a result ("sequela") of an encephalitis, a Table Injury, attributable to his November 22, 2004, MMR vaccine. (ECF No. 45, ¶ 11.)

Petitioners' and Respondent's expert reports were filed on December 8, 2011, and April 23, 2012, respectively (Ex 26; Ex A), and the parties submitted briefing regarding the standard to be applied in terms of what constitutes proof of an "encephalitis" for purposes of demonstrating a Table Injury under the Vaccine Act. (ECF Nos. 58, 61, and 62.) Special Master Campbell-Smith issued a pre-hearing ruling regarding, *inter alia*, the definition of "encephalitis" for Program purposes on January 18, 2013. (ECF No. 66.)

The expert hearing was conducted on January 25, 2013, at the Office of Special Masters in Washington, D.C, with testimony from Drs. Shuman and Wiznitzer. (*See* Transcript of Proceedings, ECF No. 71 ("Tr.").) Following a post-hearing status conference, Special Master Campbell-Smith issued an order on February 11, 2013, addressing objections raised at the hearing, regarding the timeliness and clarity of exhibits presented by Respondent's expert at the hearing, and setting a schedule for the filing of annotated exhibits and a rebuttal report from Petitioners' expert. (ECF No. 67.)

The case was reassigned to me on March 8, 2013 (ECF No. 72),[7] after which I issued an order instructing the parties to adhere to the filing schedule set forth in the post-hearing order of Special Master Campbell-Smith (ECF No. 73.) Ultimately, annotated trial exhibits were filed on April 12, 2013 (ECF No. 79), and Petitioners' rebuttal expert report (Ex. 49) was filed on May 17, 2003 (ECF No. 84).

[7] This reassignment resulted from the fact that Special Master Campbell-Smith had been nominated by the President to become a Judge of this Court. She subsequently, after being confirmed by the U.S. Senate, became a Judge of this Court, and later became Chief Judge of this Court.

Petitioners' initial post-hearing brief was filed on August 8, 2013. (ECF No. 92.) A responsive brief was filed by Respondent on September 27, 2013 (ECF No. 93), and a reply brief by Petitioners was filed on October 15, 2013 (ECF No. 94).

IV

ISSUE TO BE DECIDED

In this case, Petitioners seek a Program award, contending that the severe neurologic disorder from which N.N. suffers was caused by a "Table Injury Encephalitis" resulting from either the MMR or DTaP vaccinations administered on November 22, 2004. Specifically, Petitioners contend that N.N.'s disorder was caused by "limbic encephalitis," the first symptoms of which took place shortly after those vaccinations. Petitioners seek to prove that N.N. experienced "encephalitis" after his vaccinations on November 22, 2004, and that the onset of that injury falls within the timeframes set forth in the Vaccine Injury Table. They are relying *exclusively* on the Table Injury presumption of causation, and are *not* attempting to establish a "cause-in-fact" basis linking N.N.'s injury to his vaccinations. (*See* footnote 2, above.)

Respondent contends that N.N. suffers from CDD unrelated to his vaccinations. Respondent's expert argues that N.N. did *not* suffer encephalitis. Respondent argues that N.N.'s MRI scans are devoid of proof that N.N. suffers from limbic encephalitis, and that he is missing certain key symptoms of encephalitis.

After careful consideration, I find, for all the reasons discussed below, that Petitioners have *failed* to meet their burden. Although I accept Special Master Moran's findings concerning the onset of certain symptoms in N.N. soon after the vaccinations of November 22, 2004, I find that Petitioners have *failed* to establish by preponderant evidence that what N.N. experienced at that time was, in fact, a "Table Encephalitis."[8]

V

SUMMARY OF EXPERT WITNESSES' QUALIFICATIONS AND OPINIONS

In this case, each side relies upon the expert reports and hearing testimony of one medical expert. At this point, I will briefly summarize both the qualifications and the opinions of those expert witnesses.

A. Petitioners' expert, Dr. Robert M. Shuman

1. Qualifications

Petitioners rely primarily on the expert reports and testimony of Dr. Robert M. Shuman. Dr. Shuman studied experimental psychology at Cornell University from 1959 to 1963 before attending Stanford Medical School from 1964 to 1968. (Ex. 27, p. 1.) From 1969 to 1970 he completed a residency in pediatrics at the University of Colorado Medical Center. (*Id*.) He later

[8] Petitioners have the burden of demonstrating the facts necessary to show entitlement to an award by a "preponderance of the evidence." § 300aa-12(a)(1)(A). Under that standard, the existence of a fact must be shown to be "more probable than its nonexistence." *In re Winship*, 397 U.S. 358, 371 (1970) (Harlan, J., concurring).

completed a residency in pathology at the University of Washington from 1970 to 1972. (*Id*.) He is licensed to practice medicine in several states, and has been Board-certified in neurology and neuropathology. (Ex. 27, p. 2.) He has also been certified by the American Society of Neuroimaging. (*Id*.)

Dr. Shuman was an instructor in neuropathology for the University of Washington from 1974 to 1975. (*Id*., p. 1) He was an assistant professor of Neuropathology at the University of Pittsburgh School of Medicine from 1975 to 1976, and a professor of Neurology and Pathology at the University of Nebraska from 1977 to 1983. (*Id*.) From 1985 to 1988 he served as Vice Chairman of the department of Pathology at the University of Oklahoma, and later as Interim Chairman of Neurology there from 1989 to 1990. (*Id*.) In 1991 he left academia to set up a private practice in pediatric neurology, which he maintained until retiring in 2006. (Tr. 16-17.) Since that time he has served as a legal consultant. (Tr. 17.)

Dr. Shuman lists numerous grants and awards on his curriculum vitae. (Ex. 27, pp. 2-3.) In addition, he lists many publications, including 49 journal articles, two textbooks, and 38 abstracts. (Ex. 27, pp. 6-11.) He has also held editorial positions with the *Journal of Child Neurology*. (*Id*., p. 5.)

2. Summary of Dr. Shuman's opinion

Dr. Shuman's report in this case indicates that he believes that N.N.'s *MMR* vaccination of November 22, 2004, caused him to suffer a viral encephalitis that damaged the limbic system of his brain.[9] Dr. Shuman interprets both N.N.'s 2005 and 2011 MRIs as abnormal, and argues that they demonstrate a pattern of abnormalities consistent with scarring from past encephalitis. (Ex. 26, p. 7.) (These alleged abnormalities are discussed in detail in Section VIII below.) Dr. Shuman asserts that N.N.'s neurologic damage in the limbic system correlates to his regression and behavior problems. (*Id*., pp. 8-9.) He contends that the pattern of limbic encephalitis is "consistent with the literature description of Measles Encephalitis in the wild." (*Id*., p. 7.) At the hearing in this case, Dr. Shuman acknowledged that absent evidence that N.N. had received an MMR vaccine, he would agree that N.N.'s clinical course could be considered consistent with the onset of CDD. (Tr. 125.) He also indicated that he could not offer an opinion that N.N. experienced encephalitis absent evidence of abnormality in N.N.'s MRI scans. (Tr. 109.)

B. Respondent's expert, Dr. Max Wiznitzer

1. Qualifications

Respondent relies on the expert reports and testimony of Dr. Max Wiznitzer. Dr. Wiznitzer attended the Northwestern University Honors Program and specialized in Medical Education, earning a Bachelor of Science degree in Medicine in 1975 before entering medical school. (Ex. B, p. 1.) He attended Northwestern University Medical School and graduated in 1977 with a degree in medicine. (*Id*.) During his postgraduate training, Dr. Wiznitzer was a resident in pediatrics at the Children's Hospital Medical Center in Cincinnati, Ohio, from 1977 to1980. (*Id*.) He also was a fellow in developmental disorders at the Cincinnati Center for

[9] Although Petitioners also assert involvement of N.N.'s DTaP vaccination of the same date, Dr. Shuman opined that he *did not* believe that the DTaP vaccination contributed to N.N.'s condition. (Tr. 264.)

Developmental Disorders from 1980 to 1981. (*Id*.) He thereafter became a fellow in pediatric neurology at the Children's Hospital of Pediatric Neurology from 1981 to 1984. (*Id*.) He received the NIH National Research Service Award fellowship in Higher Cortical Functions from 1984 to1986. (*Id*., p. 2.) From 1986 to the present, Dr. Wiznitzer has served as an Assistant Professor of Pediatrics, Neurology, and International Health at Case Western Reserve University. (Ex. B, p. 2; Tr. 132.)

Dr. Wiznitzer has additionally won the NIH National Research Service Award from the Albert Einstein College of Medicine in 1986, and was recognized as the Professional of the Year from the Autism Society of Ohio in 1991. (Ex. B, p.4.) He was certified by the American Board of Pediatrics in 1982, the American Board of Psychiatry and Neurology in Child Neurology in 1986, and the National Board of Medical Examiners in 1978. (Ex. B, p. 5; Tr. 134.) He has been licensed to practice in three states. (Ex. B, p. 5.) Dr. Wiznitzer served on the Editorial Board of many journals, including *Pediatric Neurology*, *Journal of Child Neurology*, and *Lancet Neurology*. (*Id*., p. 6.) He has helped author 47 original articles, 9 book chapters, and 52 abstracts, which are listed on his CV. (*Id*., pp. 12-22.)

2. Summary of Dr. Wiznitzer's opinion

Dr. Wiznitzer believes that N.N.'s clinical course is consistent with CDD, and argues that neither N.N.'s MRIs nor his clinical symptoms indicates that N.N. suffered from encephalitis. (Ex. A, pp. 4-5; Tr. 161.) Dr. Wiznitzer stresses that individuals who suffer an episode of encephalitis have a significant change in mental status such as lethargy, stupor, or coma, as well as movement disorders, symptoms which he argues are absent in N.N.'s case. (Ex. A, p. 5; Tr. 161.) Dr. Wiznitzer also disagrees with Dr. Shuman's interpretation of N.N.'s 2005 and 2011 MRIs, arguing that both show a normal brain. (Ex. A, p. 4; Tr. 169.) Thus, he concludes that N.N.'s condition is a typical case of CDD, rather than a result of encephalitis. (Ex. A, p. 4; Tr. 215.)

VI

SUMMARY OF MY OPINON

In this case, the first major factor is that, like Chief Special Master Campbell-Smith, I conclude that it is only fair that I accept the factual findings made by Special Master Moran. I *accept* those findings as accurate.

Further, when instructed to accept those findings as accurate, both testifying experts testified that N.N.'s *clinical course* is at least generally consistent with *either* a finding of limbic encephalitis as Petitioners contend, or with an ordinary course of CDD, as respondent contends. (Ex. A, p. 4; Tr. 125.)

Therefore, I conclude that *if* Petitioners were able to show, by analysis of the MRIs, that N.N. did suffer an "encephalitis" sometime in the past, then I would find that the November 2004 symptoms *did constitute* the first symptoms of that encephalitis, and that those first symptoms likely appeared either within 72 hours of N.N.'s DTaP vaccination of November 22, 2004, *or* within 5 to 15 days after N.N.'s MMR vaccination of that same date. Thus, if the MRI analysis were favorable to Petitioners, I *would* find that N.N. suffered a Table Injury Encephalitis.

Accordingly, the outcome of this case boils down to which party's interpretation of the *MRI results* is more persuasive.

For the reasons set forth below in part VIII of this Decision, I found the testimony of Dr. Wiznitzer concerning the MRIs to be substantially more persuasive than that of Dr. Shuman, and thus I conclude that Petitioners have *failed* to show that it is "more probable than not" that N.N. suffered from a limbic encephalitis, or *any* encephalitis. I will set forth my detailed analysis concerning the MRIs below.

VII

LEGAL STANDARD: "TABLE ENCEPHALITIS"

For petitions, such as this one, filed since March 24, 1997, "encephalitis" exists as a Table Injury for MMR and DTaP vaccinations. I will set forth the relevant Table Injury sections below.[10]

§ 100.3 Vaccine injury table.

(a) In accordance with section 312(b) of the National Childhood Vaccine Injury Act of 1986, * * * the following is a table of vaccines, the injuries, disabilities, illnesses, conditions, and deaths resulting from the administration of such vaccines, and the time period in which the first symptom or manifestation of onset or of the significant aggravation of such injuries, disabilities, illnesses, conditions, and deaths is to occur after vaccine administration for purposes of receiving compensation under the program:

VACCINE INJURY TABLE

	Vaccine	Illness, disability, injury or condition covered	Time period for first symptom or manifestion of onset or of significant aggravation after vaccine administration
	*	*	*
I.	Vaccines containing whole cell pertussis bacteria, extracted or partial cell pertussis	A. Anaphylaxis or anaphylactic shock	4 hours
		B. Encephalopathy (or encephalitis)	72 hours

[10] The statute itself contains a version of the Vaccine Injury Table that applied to vaccinations administered prior to the enactment of the Program and for several years after that enactment. See § 300aa-14(a). However, the Vaccine Injury Table was administratively modified with respect to Program petitions, such as this one, that were filed after March 24, 1997. See 62 Fed. Reg. 7685, 7688 (1997); *O'Connell v. Shalala*, 79 F.3d 170 (1st Cir. 1996). That Table modification, along with an earlier administrative modification of the Table in 1995 (see 60 Fed. Reg. 7678 (1995)), significantly altered the "Table Injury" categories with respect to the MMR and DTaP vaccinations from the version of the Table contained in the statute. The portion of the new Table applicable to this case, listing "encephalitis" as a Table Injury for the MMR and DTaP vaccinations, appears at 42 C.F.R. § 100.3(a)(II)(B) and (III)(B) (10-1-97 edition of C.F.R.--all C.F.R. references in this Decision will be to the 10-1-97 edition of the C.F.R.).

	bacteria, or specific pertussis antigens (*e.g.*, DTP, DTaP, P, DTP-Hib)	C. Any acute complication or sequela (including death) of an illness, disability, injury, or condition referred to above which illness, disability, injury, or condition arose within the time period prescribed	Not Applicable
	*	*	*
III.	Measles, mumps, rubella, or any of its components (*e.g.*, MMR, MR, M, R)	A. Anaphylaxis or anaphylactic shock B. Encephalopathy (or encephalitis) C. Any acute complication or sequela (including death) of an illness, disability, injury, or condition referred to above which illness, disability, injury, or condition arose within the time period prescribed	4 hours 5-15 days (not less than 5 days and not more than 15 days.) Not applicable
	*	*	*

Thus, as described above, this decision addresses Petitioners' "Table Injury" claim alleging that N.N. suffered an "encephalitis" corresponding either to his MMR or DTaP vaccinations of November 22, 2004. As indicated in the chart, the applicable Vaccine Injury Table lists "encephalopathy (or encephalitis)" as a compensable injury if the first symptoms thereof occurred within 5 to 15 days after an MMR vaccination, or within 72 hours of a DTaP vaccination. (§ 300aa-14(a), as amended by 42 CFR § 100.3.) This simplifies Petitioners' burden in that they have the benefit of a *presumption* of causation. That is, as described in Section I above, if Petitioners demonstrated that it was more likely than not that N.N. experienced the first symptoms of an encephalitis within 72 hours of receiving his DTaP vaccination, or within 5 to 15 days after receiving his MMR vaccination, his encephalitis would be a Table Injury, *presumed* to have been caused by the vaccination, and the Petitioners would automatically be entitled to compensation for any complication of that encephalitis (unless it was affirmatively shown by the Respondent that the encephalitis was caused by some factor other than the vaccination.) (§ 300aa-13(a)(1)(A); § 300 aa-11(c)(1)(C)(i); § 300aa-14(a); § 300aa-13(a)(1)(B).)

Of course, this still leaves open the question of what constitutes "encephalitis" for Program purposes. In most instances, Table Injury cases are guided by the statutory or

regulatory "Qualifications and aids in interpretation" ("QAI"), which provide more detailed explanation of what should be considered when determining whether a vaccinee has actually suffered an injury listed on the Vaccine Injury Table. (§300aa-14(b).) Significant to this case, however, while "encephalopathy" is carefully and minutely defined in the QAI, "encephalitis" is not. (*Id*.) The parties agree that, although the two terms are listed together on the table--*i.e.* "encephalopathy (or encephalitis)"--they refer to two distinct conditions. (*See* ECF No. 58, p. 2; ECF No. 61, p. 4.) The parties differ greatly, however, in their preferred definitions of "encephalitis." (*Id*.)

This issue was previously briefed before Special Master Campbell-Smith prior to the expert hearing. (ECF Nos. 61 and 62.) Petitioners argued that "because the law governing the Program does not define encephalitis, you should use 'the common, ordinary, and accepted meaning' of encephalitis." (ECF No. 61, p. 5.) Citing to *Taber's Cyclopedic Medical Dictionary*, Petitioners then urged that encephalitis is simply "inflammation of the brain." (*Id*.) Respondent, on the other hand, argued that the court should apply Respondent's own draft proposal for a revision to the QAI, setting forth detailed criteria for demonstrating encephalitis. (ECF No. 62, pp. 2-5.) Significantly, Respondent admitted that, far from being a binding addition to the QAI, the draft was only an *anticipated* proposal, and, "at this time, respondent does not know when a new version of the QAI will be proposed through rulemaking." (*Id*.)

Special Master Campbell-Smith concluded that although Petitioners' definition was accurate, it was "too broad for Program purposes." (ECF No. 66, p. 1.) The Special Master also declined to adopt Respondent's proposed criteria "because the proposed definition of encephalitis is not yet part of the QAI." (*Id*.) Rather, Special Master Campbell-Smith ruled that "among the factors to be considered are: (1) whether [N.N.] in fact did experience demonstrable brain inflammation, (2) whether the impact of the claimed inflammation on [N.N.]'s brain was severe enough to result in the injuries he experienced, (3) whether the location of the inflammation in [N.N.]'s brain could have caused the symptoms he experienced, and (4) whether there is evidence of the appropriate temporal relationship between the onset of [N.N]'s inflammation and his vaccinations." (*Id*., p. 2.)

First, I agree with Petitioners' contention that, according to principles of statutory construction, the appropriate definition to use in this context is the "common, ordinary, and accepted meaning." (*See, e.g., Waddell v. HHS*, No. 10-316V, 2012 WL 4829291, at *8 (Fed. Cl. Spec. Mstr. Sept. 19, 2012) ("In the absence of a specific indication to the contrary, words used in the statute will be given their common, ordinary and accepted meaning, and the plain language of the statute will be afforded its plain meaning.").) This necessitates rejecting Respondent's proposed definition.

Citing "encephalopathy" as an example, Respondent argues that QAI criteria can be narrower than the commonly accepted medical definition. (ECF No. 93, p. 6.) That is, Respondent implicitly acknowledges that QAI definitions are not necessarily commonly used definitions, but rather specialized for Program purposes. Yet, the Respondent also admits that her proposed definition has not yet been vetted by the rulemaking process. (ECF No. 62, p. 2.) Thus, Respondent's proposed definition is neither the commonly used definition, nor a statutorily prescribed definition. Obviously, if Respondent's definition of encephalitis is ultimately adopted as a result of the completed rulemaking process, then it will control in the future. In the meantime, however, to apply Respondent's *anticipated* proposal would be to preempt the

rulemaking process, abrogate principles of statutory construction, and impermissibly heighten the Petitioners' burden.

Therefore, I accept Petitioners' argument that an "encephalitis" is simply any "inflammation of the brain." However, that does not mean that the rest of Special Master Campbell-Smith's four-part discussion of "encephalitis" in this case is not important. The Vaccine Injury Table, as set forth above, prescribes that "encephalitis" is a Table Injury and therefore presumed to be vaccine-caused, and *also* that "any acute complication or sequela" *of the encephalitis* is also presumed to be vaccine-caused. Thus, Special Master Campbell-Smith was correct in stating that Petitioners would need to prove not only that N.N. (1) "in fact did experience demonstrable brain inflammation," but also must show "(2) whether the impact of the claimed inflammation on [N.N.'s] brain was severe enough to result in the injuries he experienced, [and] (3) whether the location of the inflammation in N.N.'s brain could have caused the symptoms he experienced." In other words, Special Master Campbell-Smith's Factors (2) and (3) are inherent in showing that N.N.'s conditions are an "acute complication or sequela" of his encephalitis. And, of course, Special Master Campbell-Smith was correct that Petitioners also need to demonstrate "(4) whether there is evidence of the appropriate temporal relationship between the onset of N.N.'s inflammation and his vaccinations"--that is, that the first symptoms of the inflammation arose within 72 hours of N.N.'s DTaP vaccination, or within 5 to 15 days after his MMR vaccination (both vaccinations occurred on November 22, 2004).

VIII

ANALYSIS OF MRI STUDIES

The correct interpretation of N.N.'s MRI studies is clearly the key issue in this case. That is because, though the two experts in this case differ on the correct interpretation of N.N.'s MRI images, both experts agree that N.N.'s *clinical course* is basically consistent with either limbic encephalitis *or* Childhood Disintegrative Disorder (CDD). (Ex. A, p. 4; Tr. p. 125.) In fact, Dr. Shuman acknowledged at the expert hearing that absent an abnormal finding in N.N.'s MRI, he would *not* be able to opine that N.N. experienced encephalitis. (Tr. 109.) Thus, in her post-hearing Order of February 11, 2013, Special Master Campbell-Smith characterized the interpretation of N.N.'s MRI imaging as the "dispositive" issue in this case. (ECF No. 67, p. 2.) I agree.

For the reasons discussed below, however, I find that Petitioners have *failed* to demonstrate that N.N.'s MRI images show any abnormalities, and have therefore *failed* to carry their burden concerning this key issue. Dr. Shuman presented a series of MRI images from two MRI studies of N.N.'s brain alleging the presence of several different abnormalities indicative of past inflammation. In each instance, I found Respondent's interpretation of the MRI images more persuasive.[11]

A. Dr. Wiznitzer's MRI interpretation is more convincing than Dr. Shuman's

[11] Obviously, because I have found that Petitioners have failed to demonstrate that N.N. experienced any inflammation of the brain at all, it is unnecessary to address prongs two and three of Special Master Campbell-Smith's test, which go to the location and severity of the inflammation. These prongs are mooted by my finding.

Dr. Shuman opined that MRI studies conducted in November of 2005 and October of 2011 demonstrate that N.N. had scarring of the brain consistent with past encephalitis. During the evidentiary hearing, Dr. Shuman walked the special master through eight MRI images from these studies (Exs. 42-A through 45-B). (Tr. 33-77.) Dr. Shuman selected these eight images in particular, because they are the "most illustrative" of the abnormalities he alleges to be present. (Tr. 75-76.) Specifically, Dr. Shuman argued that Exhibits 42-A through 45-B show the following abnormalities which evidence scarring of the brain consistent with past encephalitic inflammation: Trigonal hyperintensities; hyperintensity of the hippocampi; hyperintensity of the ventricle lining; enlarged ventricles; and hyperintensity of the fornices. Dr. Wiznitzer, however, provided a contrary view on each of these five points. Dr. Shuman's and Dr. Wiznitzer's arguments relative to each of these five alleged abnormalities are addressed in turn below.

1. Trigonal hyperintensities

Exhibits 42-A and 42-B are images of the same portion of the brain (described as the "coronal cut") from N.N.'s 2011 and 2005 MRI studies, respectively. Both of these images, according to Dr. Shuman, show an abnormal brain, illustrating "linear radiant striped zones of T2 hyperintensity in the same region of the peritrigonal white matter." (Tr. 48-49.) According to Dr. Shuman, the white matter of the brain is represented on the MRI image as "black signal" and therefore, normal myelination appears as black. (Tr. 45.) Describing Ex. 42-A from N.N.'s 2011 MRI, Dr. Shuman noted that "dense, black signal void, that is, normal white matter, in the deep centrum semiovale, that is the core of the white matter, in the human cerebrum should be equally black and it's not. It's Swiss cheese. It's lighter than it ought to be, it has holes in it, it has lines in it. It is rattled. It is damaged." (Tr. 45.) In Dr. Shuman's view, this represents old scarring consistent with "an old, established inflammatory encephalitis." (Tr. 46-47.)[12]

Dr. Wiznitzer, however, describing the same area that Dr. Shuman characterized as "Swiss cheese," observed that Exhibit 42-A shows "a band of normal myelinated white matter sitting between these linear intensities, and the ventricle, the ventricular wall is basically smooth." (Tr. 208.) According to Dr. Wiznitzer, "bottom line, these are known as terminal zones, a normal finding in individuals anywhere between 16 months up through the second decade of life." (Tr. 208-09.) Dr. Wiznitzer indicated that terminal zones are believed to be areas of immature myelin, where vascular pathways are beginning to form and cerebrospinal fluid is captured. (Tr. 209.) This, Dr. Wiznitzer explained, is why the hyperintensities appear as linear. (*Id*.)

In support of his contention, Dr. Wiznitzer presented MRI images appearing in the medical literature that are considered "normal" and which feature the type of linear hyperintensities identified by Dr. Shuman as abnormal. (Tr. 209-11; Trial Ex. 4, *Assessment of Normal Myelination with Magnetic Resonance Imaging*, p. 27, Figure B; Trial Ex. 5, *Pediatric Neuroimaging*, Fourth Ed., p. 39, Figure C.)[13] Dr. Wiznitzer also indicated that his opinion is

[12] Ex. 42A is marked with black circles noting the location of the hyperintensity described by Dr. Shuman. (Tr. 33.)

[13] At the hearing, Petitioners raised an objection to the quality of the copies being presented by Dr. Wiznitzer. Special Master Campbell-Smith resolved this objection in an order of February 11, 2013, (ECF No. 67) which allowed for the filing of clearer annotated copies of the hearing exhibits. I note that it is these later-filed exhibits which I have reviewed in reaching my decision. (See Respondent's Trial Exhibits 4 and 5, ECF No. 79-6 and 79-7,

supported by the lack of any indication, such as "puckering of the brain," that N.N. experienced tissue loss in conjunction with these hyperintensities. According to Dr. Wiznitzer, this feature differentiates terminal zones from scarring. (Tr. 209.)

Importantly, Dr. Shuman did not dispute Dr. Wiznitzer's description of terminal myelination, but merely argued in rebuttal that N.N. was too old at the time of his MRI to have exhibited immature myelination, and that the spaces around N.N.'s vessels do, contrary to Dr. Wiznitzer's opinion, indicate tissue loss. (Tr. 250-51; *see also* ECF No. 84-1, pp. 17-19.) In particular, Dr. Shuman characterized the image presented by Dr. Wiznitzer in Trial Ex. 5 as "a smooth anatomic phenomenon of age, age-limited, age-dependent in the first year of life." (Tr. 250.) N.N., however, was eleven years old at the time Ex. 42-A was imaged, and according to Dr. Shuman, N.N.'s MRI "is not a smooth band or zone of terminal myelination. It is instead increased size of spaces around the vessels." (*Id*.) Thus, Dr. Shuman contended that N.N.'s imaging is not age-appropriate and that it shows tissue loss, because of the size of the "Virchow-Robin" spaces around the vessels. (Tr. 250-51.) Dr. Shuman also stressed that the asymmetry of the perivascular spaces indicates that it is more likely to be pathologic than developmental.[14] (Tr. 251.)

However, Dr. Shuman's assertion that terminal myelination is limited to the first year of life is not supported by the medical literature submitted in this case. *Assessment of Normal Myelination with Magnetic Resonance Imaging*, submitted by Respondent as Trial Exhibit 4, indicates, just as Dr. Wiznitzer testified, that terminal zones may remain hyperintense under T2 imaging into the second decade of life.[15] (Trial Ex. 4, p. 3 (Table 1).) Moreover, the image relied on by Dr. Wiznitzer within Trial Exhibit 4 shows the brain of a six-year-old girl rather than an infant in the first year of life, and, consistent with Dr. Wiznitzer's opinion in this case, Trial Exhibit 4 indicates that "small areas of hyperintensity are considered to be *a normal developmental variant* in children and at times are even identifiable *in the young adult population*." (Trial Ex. 4, p. 11 (emphasis added).) To the extent that Dr. Shuman's later supplemental report takes issue, not with the presence of *any* hyperintensity in an eleven-year-old, but with the *greater prominence* of the hyperintensities found in N.N.'s MRI compared to the six-year-old girl presented in Exhibit 4, he does not support this part of his argument with any citation to any medical literature indicating what degree of hyperintensity would be considered age-appropriate for an eleven-year-old. (ECF No. 84-1, p. 17.)

Additionally, Dr. Shuman contends that N.N.'s MRI does not show terminal myelination, but, rather, shows enlarged perivascular spaces. He notes that "to see any perivascular spaces in this age is remarkable, worthy of further comment." (Tr. 251.) Trial Exhibit 4, however,

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filed on April 12, 2013.) Dr. Shuman additionally submitted his own reproduction of Trial Exhibit 5 (ECF No. 85-2), which I have also reviewed.

[14] Dr. Shuman does not provide any citation for his assertion that asymmetry is necessarily pathologic. I note, further, that Dr. Shuman and Dr. Bauza are not in agreement on this point – at least as regards asymmetry in the size of the hippocampi. (Tr. 117.)

[15] I note that while Dr. Shuman clearly takes issue with Dr. Wiznitzer's comparison of N.N.'s MRI studies to those depicted in *Assessment of Normal Myelination with Magnetic Resonance Imaging*, he does not appear to challenge the article's authority as a general proposition, characterizing it as "a state-of-the-art perspective on the current status of understanding MRI imagery in early childhood." (ECF No. 84-1, p. 14.)

indicates that although perivascular spaces may contribute to signal hyperintensity, they can be distinguished from terminal zones "by looking for small bands of low signal, normally myelinated brain separating the high signal regions from the ventricles." (Trial Ex. 4, p. 11.) This pattern, which according to the article is present with terminal zones but not perivascular spaces, is illustrated in figure 9(B) and appears to closely match the pattern displayed in N.N.'s own MRI image at Ex. 42-A. Moreover, it is exactly what Dr. Wiznitzer indicated was present when he described N.N.'s MRI image, noting that "if we look closely at the imaging study, there is a band of normal myelinated white matter sitting between these linear intensities, and the ventricle, the ventricular wall is basically smooth." (Tr. 208.)

Thus, on the whole, I find Dr. Wiznitzer's explanation for the presence of the trigonal hyperintensities more persuasive than Dr. Shuman's. Although it is undisputed that Exhibits 42 A and B illustrate linear hyperintensities within N.N.'s white matter, Dr. Wiznitzer offered a coherent explanation for their presence that is supported by medical literature, arguing that these hyperintensities are a normal developmental variant known as terminal myelination. Dr. Shuman's response to this explanation, however, seemed, if not completely at odds with the medical literature in the record, at the very least less consistent with it. Dr. Shuman appears to have conceded the point that terminal myelination can appear as hyperintense as a normal developmental variant, but argued that it is not an age-appropriate finding for N.N. and that the hyperintensity should be interpreted as perivascular spaces, which would not be normal. For the reasons discussed above, however, I do not find these arguments to be in accord with the medical literature submitted in this case.

2. Bilateral hyperintensity of the hippocampi

Exhibit 43-A and 43-B are axial images from N.N.'s 2011 and 2005 MRI studies, respectively. (Tr. 50-52; 61-62.) Dr. Shuman argued that these images show bilateral hyperintensity of the *posterior* portions of the hippocampi. (Tr. 57; 63.) He suggested that the level of T2 signal may be at the edge of normal limits, but noted that signal from a normal brain 'would not be a globular, irregular, intense signal as you see here."[16] (Tr. 57.) According to Dr. Shuman, this is significant because "the hippocampi is a very prominent part of the limbic system. It's a very prominent site of attack in encephalitis, especially limbic encephalitis." (Tr. 57-58.)

Citing to "Limbic Encephalitis in Children and Adolescents," an article by Haberlandt published in *Archives of Disease in Childhood* (Ex. D), Dr. Wiznitzer disagreed. He indicated that limbic encephalitis causes inflammation of the *anterior* part of the hippocampal region, rather than the *posterior* portions of the hippocampi as Dr. Shuman suggested. (Tr. 170-75.) Describing the hyperintensity that Dr. Shuman pointed out in Exhibit 43-A, Dr. Wiznitzer testified that "we're in a different territory than where the imaging classically tells us we should see abnormalities with limbic encephalitis."[17] (Tr. 182.) For example, Dr. Wiznitzer pointed out

[16] Ex. 43-A is marked with white arrows indicating areas of hyperintensity Dr. Shuman described. (Tr. 56.)

[17] Petitioners argue in their post-hearing briefing that they are not obligated to prove "limbic encephalitis," but only "encephalitis" in a non-specified form. (ECF No. 94, pp. 16-17.) That is true, as a matter of law. However, Dr. Shuman has consistently and specifically opined that N.N.'s MRI studies show and N.N.'s clinical symptoms are explained by "limbic encephalitis." (*See, e.g.*, Ex. 26, p. 11 ("An MRI of his brain 1 year into his post-vaccinal course illustrates demyelinated lesions of his limbic system. This pattern of damage is seen after a viral encephalitis

that the MRI image of "three weeks" appearing as part of Figure 1 of the Haberlandt article displays hyperintensity in this anterior location. (Tr. 171-72; Ex. D, p. 187.)

Dr. Shuman rejected Dr. Wiznitzer's reading of the Haberlandt article, arguing that "there's not enough information [in the three-week image] to tell you whether or not Dr. Wiznitzer is right that this is confined to the anterior one-third of the hippocampus. I will tell you that Dr. Wiznitzer is not right that limbic encephalitis is confined to the anterior one-third of the hippocampus." (Tr. 241.) Dr. Shuman asserts that the Haberlandt article speaks of inflammation *anywhere* in the mediotemporal lobe as evidence of limbic encephalitis, and does not at any point limit its discussion by use of the modifying "anterior" descriptor. (ECF No. 84-1, p. 2.)

Dr. Shuman is correct in that I do not see any text in the Haberlandt article limiting evidence of limbic encephalitis to the anterior portion of the hippocampus. (Ex. D.) However, Dr. Wiznitzer relied on the images from the Haberlandt article as only one example of what he described as a "classic" pattern that he has observed in his own clinical practice (Tr. 182-83), and Dr. Shuman has not produced any literature supporting his position that hyperintensity of the *posterior* hippocampus in particular is evidence of limbic encephalitis.[18]

Nonetheless, even assuming *arguendo* that Dr. Shuman's reliance on a *posterior* hippocampal abnormality as a sign of limbic encephalitis was sufficient, Dr. Wiznitzer additionally casts significant doubt on Dr. Shuman's interpretation of that hyperintensity as abnormal. With regard to the posterior hyperintensity pointed out by Dr. Shuman, Dr. Wiznitzer argued that this was an "artifact" of the MRI process and was *not* an abnormality at all. (Tr. 182-83.) Dr. Wiznitzer pointed out that when viewed from a coronal plane, the hyperintensity identified in Exhibit 43-A is rectangular in shape. (Tr. 184.) According to Dr. Wiznitzer, a true finding of abnormality would look "fluffy" or have an "irregular contour" whereas this image is "like a little peg." (Tr. 185.)

Dr. Shuman acknowledged the rectangular shape (Tr. 240), but argued that the hyperintensity in N.N.'s MRI cannot be an artifact, because it is visible in different planes (ECF No. 84-1, p. 6-7). In particular, Dr. Shuman stresses that both N.N.'s 2005 and 2011 MRI studies show the same hyperintensity, arguing that this makes it highly unlikely that the image is an artifact, which should be difficult to reproduce. (*Id*.) However, Dr. Shuman, does not provide any supporting citations for his arguments, and I am left without the requisite background information regarding the causes and nature of MRI artifacts to find it persuasive. That is, Dr. Shuman has not substantiated his assertion that MRI artifacts are transient, unreproducible, or

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in which the tissues of the limbic system have been affected. The clinical pattern of his "Childhood Disintegrative Disorder exactly fits the neuroradiologic pattern of his viral (limbic) encephalitis.").) With Dr. Shuman's opinion explicitly limited in that way, even though Petitioners are not *obligated* to demonstrate "limbic encephalitis" in particular, they have not presented any medical evidence in this case establishing any *other* form of encephalitis.

[18] Dr. Shuman asserts in his post-hearing supplemental report that the hyperintensity in N.N.'s 2011 MRI is not limited to the posterior hippocampi, but is actually found throughout the entire hippocampi, including the anterior portions. (ECF No. 84-1, p. 5.) This is not consistent with his hearing testimony, however, in which he clearly characterized the hyperintensity as being posterior. (Tr. 57.) Moreover, at no point has Dr. Shuman, in his testimony or his written reports, presented any image from either of N.N.'s MRI studies which he would contend illustrates hyperintensity of the anterior hippocampi.

limited to a single plane. Dr. Wiznitzer, on the other hand, supported his argument with examples from the medical literature, showing the court "normal" MRI images featuring similar artifacts.[19]

For example, Respondent's Trial Exhibit 2 is an MRI image of a normal 24-year-old male.[20] (Tr. 189.) According to Dr. Wiznitzer, it, like Exhibit 43-A, shows "the exact same artifact, a bright, somewhat rectangle, located on either side of the fluid-filled space in the exact same location in fact." (Tr. 190.) Dr. Wiznitzer also produced an article from the *American Journal of Neuroradiology*, September 1999, titled "Normal Myelination of the Pediatric Brain Imaged with FLAIR Magnetic Resonance Imaging." (Tr. 191; Trial Ex. 3.) Looking at image "I" from that article, an MRI scan of a normal three-year-old, Dr. Wiznitzer indicated that this is "a slice of the brain that is very similar, in a similar location to the one listed on Exhibit 43-A. Both of these are FLAIR studies. And I think we can appreciate a bright linear artifact on both sides of the fluid-filled space. It looks more like, I would call it like a straw, a straw shape." (Tr. 191-92, 193.) Trial Exhibit 4, presented by Dr. Wiznitzer, is an article titled "Assessment of Normal Myelination with Magnetic Resonance Imaging" from *Seminars in Neurology*. (Trial Ex. 4.) Looking at image A on page 23 of Trial Ex. 4, the scan of a normal 28-month-old, Dr. Wiznitzer argued that this image likewise shares the same brightness on either side of the CSF fluid-filled space. [21] (Tr. 194.)

Again I find that Dr. Wiznitzer presents the more compelling explanation with regard to this particular point. There is disagreement among these two experts on whether hyperintensity only of the posterior hippocampi, without involvement of anterior portions, represents a marker of limbic encephalitis. This ambiguity alone might be indication enough that Petitioners have failed to meet their burden on this issue. But in any event, it would appear that the anterior hyperintensity shown on N.N.'s MRI may be no indication of any abnormality at all. Dr. Wiznitzer's opinion that it is nothing more than an artifact of the MRI process is compelling, particularly in light of the multiple examples he provides from medical literature.

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[19] Moreover, for the reasons discussed in subsection B below, I find Dr. Wiznitzer, as a *currently* practicing clinician, to be more qualified to speak about the quality of *contemporary* MRI technology.

[20] Dr. Shuman argues in his supplemental report that Dr. Wiznitzer is wrong to claim that this 24-year-old male is "normal" with no history of central nervous system problems, since this individual suffered from multiple sclerosis. Dr. Shuman stops short, however, of explicitly arguing that a diagnosis of multiple sclerosis would explain the artifact Dr. Wiznitzer indicated was present. (ECF No. 84-1, pp. 5-6.) In any event, this is only one of multiple examples presented by Dr. Wiznitzer.

[21] These images were not previously produced to Petitioners' counsel prior to the hearing. At the hearing, Petitioners' counsel raised objections to the images on the basis of foundation, image quality, and lack of notice. (Tr. 186-96.) I note, however, that *Special Master Campbell-Smith* allowed Petitioners an opportunity to submit a supplemental report rebutting these submissions, which they filed on May 17, 2013. (*See* ECF No. 84-1.) She concluded that such procedure would give Petitioners a full and fair opportunity to address the substance and quality of these exhibits. I agree, and therefore I am considering them despite the objection noted above. This is consistent with Program rules, which state that "in receiving evidence, the Special Master will not be bound by common law or statutory rules of evidence but must consider all relevant and reliable evidence governed by principles of fundamental fairness to both parties." (Rule 8 of the "Vaccine Rules," which make up Appendix B of the Rules of this Court.)

3. Hyperintensity in the ventricle lining

Still addressing Exhibit 43-A, Dr. Shuman additionally noted that "there is signal intensity in the subependymal, ependymal." (Tr. 53-54.) That is, "there is signal intensity around the lining of the lateral ventricle." (Tr. 54.) This is significant, argued Dr. Shuman, because "this is a favored spot for scarring in any kind of inflammatory process." (*Id*.) Although Dr. Shuman acknowledged that it does not reveal the nature of the inflammation, he argued that subependymal T2 signal intensity is evidence of past inflammation. (*Id*.)

Dr. Wiznitzer, however, argued that bright signal along the sides of the ventricles on FLAIR images are normal findings. (Tr. 199.) Turning to Trial Exhibit 3, p. 1409, Figure I, Dr. Wiznitzer pointed out that this image shows "bright signal capping on top of the ventricle" which Dr. Shuman would characterize as abnormal. (Tr. 200.) Dr. Wiznitzer explained that this type of brightness along the ventricular wall is known as "anterior cap," and is a well-known radiologic finding on T2 FLAIR images. (*Id*.) According to Dr. Wiznitzer, it is considered normal. (*Id*.) Dr. Wiznitzer additionally quoted text from *Normal Myelination in the Pediatric Brain*, underscoring the point that it is considered a normal finding after about eight months of age. (Tr. 201-02.) He further pointed out that it is a finding caused by the FLAIR technique and is not found on the normal T2 study. (Tr. 202-03.)

In response, Dr. Shuman characterized the issue as "a throw away," and noted that he only pointed it out "because [he] was fascinated by it." (Tr. 247.) He acknowledged that hyperintensity of the ventricle lining is a normal finding "to a degree," but argued that the thickness in N.N.'s case (which Dr. Shuman placed at over a millimeter) is pathologic for a child of N.N.'s age. (Tr. 245-47.) In other words, Dr. Shuman argued that "this is a pathological variant of a normal finding." (Tr. 247-48.) Significantly, although Dr. Shuman raised an issue with regard to the thickness of the hyperintensity shown on N.N.'s MRI, at no point did he offer any objective or quantifiable description of what would be a *normal* thickness. Dr. Shuman's argument was couched completely in the relative terms of "thick" versus "thin," without any context against which to measure.

Dr. Wiznitzer made a strong case, supported by the medical literature, that this type of finding is not necessarily abnormal. Dr. Shuman, on the other hand, did not substantiate his counter-argument that N.N.'s case represents a pathological variant of what he admitted was a normal finding. But in any event, given that Dr. Shuman acknowledged at the outset that hyperintensity of the ventricle lining is a non-specific indication of inflammation, that the finding is normal "to a degree," and ultimately dismissed the finding as a "throw away," I find that, even if this type of hyperintensity were abnormal, there is an insufficient basis to conclude that it is evidence of encephalitis in particular.

4. Enlarged ventricles

Exhibit 44-A is an axial cut just above ear level from N.N.'s 2011 MRI study. (Tr. 64.) Exhibit 44-B is a similar image from N.N.'s 2005 MRI study. (Tr. 69.) Dr. Shuman argued that a comparison of these two images shows that N.N. lost tissue between 2005 and 2011, because the ventricles appear larger in the 2011 image than in the 2005 image. (Tr. 69-70.) In particular, Dr. Shuman argues that while the 2005 MRI study shows appropriately-sized ventricles, the

2011 image shows ventricles that are "huge" for a child of N.N.'s age. (Tr. 64, 69.) According to Dr. Shuman, tissue loss is the only explanation for this. (Tr. 64.)

Dr. Wiznitzer, on the other hand, argued that "if you look at ventricular size over time, and this is also well-known, that in the very young child, ventricles tend to be a little bit narrower and smaller, but as you get older, as an older child, they tend to get a little bit wider, and if you don't account for that fact, you may misinterpret or over-read the information." (Tr. 212.) In rebuttal, Dr. Shuman acknowledged that "the lateral ventricles do have normative values," but reiterated his opinion that N.N.'s ventricles are "pathologically enlarged" in the 2011 MRI. (Tr. 243-44.) Neither party, however, has submitted any evidence establishing those normative values.

Here, there is a conflict on this point between these two experts unresolvable on this record. I find that Petitioners have not established that N.N.'s ventricles were pathologically enlarged, because while they have shown a change over time, they have failed to come forward with any persuasive evidence demonstrating the significance of that change, or that the resulting size of N.N.'s ventricles is abnormal.

5. Hyperintensity in the fornices

Finally, according to Dr. Shuman, Exhibits 44-A and 44-B also show hyperintensity indicative of scarring in the body of the fornix.[22] (Tr. 65, 69-70.) Dr. Shuman argues that in a normal brain "the deeply myelinated structures should give you the norm for the appearance of the columns in the body of the fornix." (Tr. 66.) He testified that Exhibit 44-A, however, "is consistent with either a direct encephalitic attack on the myelinated tissue of the fornix or with secondary gliosis to the columns of the fornix from extensive attack on the hippocampus of origin of the fornix." (Tr. 66-67.)

Additionally, Exhibit 45-A is an axial image from N.N.'s 2011 MRI cut lower than Exhibit 44-A. (Tr. 70-71.) According to Dr. Shuman, this provides another view of the scarring of the fornix. (Tr. 71-72.) That is, according to Dr. Shuman "these columns of the fornices, have too much T2 signal intensity and are therefore too white and not black enough." (Tr. 72.) Exhibit 45-B is a similar image from N.N.'s 2005 MRI. Dr. Shuman indicated that this image shows signal intensity in the same region, but does not have sufficient resolution to identify the columns of the fornix. (Tr. 74-75.)

Dr. Wiznitzer argued, however, that the hyperintensity that Dr. Shuman pointed out in the fornix is actually a further example of the "anterior cap" phenomenon discussed above with regard to the ventricle lining. (Tr. 197-200.) That is, Dr. Wiznitzer explains that "what we have is the brightness of the fornix in this area is actually this thin rim of the ventricular wall going by it and lighting it and basically contributing to that. Remember we said it's thought to be due to the packing of the axons? What's a fornix but a group of axons that are going by? So that's all this is." (Tr. 202-03.)

Again, as with the ventricle lining, Dr. Shuman does not appear to dispute that this type of hyperintensity *can* be a normal finding, but rather disputes that it is an age-appropriate finding for N.N. (ECF No. 84-1, pp. 12-14.) Specifically, Dr. Shuman argues that "a normal

[22] The fornix is marked on both Exhibits 44-A and 44-B by a white circle. (Tr. 66, 69-70.)

periventricular zone at birth is invisible. A normal periventricular subependymal zone at 1 year of age is thin. Normally the zone is still thin until the 4th decade, but it clearly becomes thicker and tougher with each decade." (*Id*., p. 13.) Thus, Dr. Shuman acknowledges that N.N.'s MRI *should* demonstrate some level of hyperintensity in the region of the fornix. He simply contends that what appears is too "thick" for an eleven year old. In this regard, however, as with the hyperintensity of the ventricle lining, Dr. Shuman has failed to offer any normative values from which to judge the appropriate thickness. He has therefore failed to substantiate his assertion that N.N.'s fornix, which he seems to concede should show *some* hyperintensity, is *pathologically* hyperintense.

B. There is no qualifications gap between these experts regarding MRI interpretation

Dr. Shuman has an impressive *curriculum vitae*, and there is no question that as a pediatric neuropathologist he is well qualified to interpret N.N.'s MRI studies. (Ex. 27.) In fact, Petitioners argue that Dr. Shuman's certification from the American Society of Neuroimaging is particularly significant, making him *more qualified* than Dr. Wiznitzer to interpret N.N.'s MRI images. (Tr. 146-47.) In effect, Petitioners argue that while Dr. Wiznitzer may have sufficient experience as a pediatric neurologist to be considered an expert in neuroimaging for this case, he lacks the additional specialization in neuroanatomy held by Dr. Shuman. (*Id*.)

Dr. Wiznitzer, however, is also well qualified to review these MRI studies. Although Dr. Shuman maintains certification from the American Society of Neuroimaging, Dr. Wiznitzer does have significant clinical and teaching experience devoted to pediatric neurology, which requires the use of MRI and other imaging techniques for diagnostic purposes. (Tr. 132-41.) Moreover, in seeking to argue that Dr. Wiznitzer has no more experience with MRIs than does the typical practicing neurologist, Petitioners overlook Dr. Wiznitzer's particular history. Dr. Wiznitzer testified that he has extensive background with neuroimaging, having written papers on the use of MRI in pediatric neurology and participated in studies on the subject. (Tr. 143-44.) Dr. Wiznitzer also participates in developing the board-certification exam for pediatric neurology, in which he is *specifically* involved in the testing concerning *neuroimaging*. (Tr. 135-36.) Dr. Wiznitzer has also been an invited lecturer for both the National Institutes of Health and the American Society of Neuroimaging, speaking to pediatric issues in neuroimaging. (Tr. 144.)

It is also worth noting that Dr. Wiznitzer challenges the weight of Dr. Shuman's "certification" from the American Society of Neuroimaging, pointing out that it is not a *board certification*, but simply a societal designation which results from a less rigorous process than would a board-certification. (Tr. 147-48.)

In addition, as Respondent points out, Dr. Shuman has not been a practicing clinician since 2006, but has instead spent that time working as a legal consultant. (Tr. 108.) While being a legal consultant is not in itself disqualifying, this criticism is not without significance. For example, in his supplemental report rebutting Dr. Wiznitzer's hearing presentation, Dr. Shuman argued--without supporting citation and therefore relying on his own expertise--that Trial Exhibit 2, an image captured in 1996, cannot be compared to N.N.'s 2005 and 2011 images because "the technology is old, clumsy, and imprecise. It produced factitious signal where 2005 and 2011 techniques do not." (ECF No. 84-1, p. 5.) Yet Dr. Shuman testified that he stopped his clinical practice in 2006, and acknowledged that he has not read an MRI in a clinical setting since that time. (Tr. 108-10.) His CV lists his most recent academic appointment as ending in 1991, his

most recent medical licensure and publication as 2003, and his most recent continuing medical education seminar as 2008. (Ex. 27, pp. 3, 8, 25.) Further, there is nothing in the record to indicate Dr. Shuman's exposure through legal consulting to contemporary MRI technology. Dr. Wiznitzer, on the other hand, remains a practicing neurologist who makes current use of MRI technology. (Ex. B; Tr. 132-44.) Thus, to the extent that Dr. Shuman himself would seek to make an issue of the efficacy of contemporary MRI technology, Dr. Wiznitzer would seem to be the more qualified expert to speak to the practical limitations of MRI technology as it existed in 2011 when N.N.'s most recent MRI study was conducted.

Ultimately, I am faced with two qualified experts – both pediatric neurologists – with different strengths. While Dr. Shuman is a pathologist with a certification in neuroimaging, Dr. Wiznitzer is a current practitioner with *up-to-date* clinical skills in utilizing neuroimaging not demonstrated by Dr. Shuman. I cannot say that one is inherently more qualified than the other. In any event, even assuming *arguendo* there was any qualifications gap between these two experts as Petitioners suggest, I find that Dr. Wiznitzer more than closed that gap by presenting coherent and detailed testimony that was supported by *specific references to medical literature*. (*See, e.g., Caves v. HHS*, 100 Fed. Cl. 119, 134 (2011), *aff'd,* 463 Fed. Appx. 932 (2012) (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136 (1997)) for the proposition that "*Daubert* does not require a trial court 'to admit opinion evidence that is connected to existing data only by the *ipse dixit* of the expert.'"); *see also Hennessey v. HHS*, No. 01-190V, 2009 WL 1709053, at *42 (Fed. Cl. Spec. Mstr. May 29, 2009) ("When experts disagree, many factors influence a fact-finder to accept some testimony and reject other contrary testimony. Objective factors, including the qualifications, training, and experience of the expert witnesses and the extent to which their proffered opinions are supported by reliable medical research, other testimony, and the factual basis for their opinions, are all significant in determining what testimony to credit and what to reject.").)

C. The reports of Drs. Bauza and Hewell are not of strong import in resolving this case.

Of course, Drs. Shuman and Wiznitzer are not the only two physicians to have offered opinions on N.N.'s MRI studies. N.N.'s 2005 MRI was initially interpreted by Dr. Brett Hewell, a radiologist. (Ex. 6, p. 25.) His later 2011 MRI was initially reviewed by Dr. Jose Bauza, a neuroradiologist. (Ex. 25, p. 1.) Dr. Hewell concluded that N.N.'s 2005 MRI was normal, while Dr. Bauza found abnormality in the 2011 MRI. (Ex. 6, p. 25; Ex. 25, p. 1.) Not surprisingly, each party favors the report which supports that party's theory, while discounting the other. Ultimately, I conclude that neither report is of much weight in resolving this case.

At the expert hearing, Dr. Shuman questioned the validity of Dr. Hewell's conclusion by raising concerns about the state of radiology in general, referring to it as being in "crisis" and claiming that radiologists do not take sufficient time or care in their interpretations. (Tr. 28-30.) He was also critical of the fact that Dr. Hewell does not specialize in *neuroradiology*. (*Id*.) When asked about the basis for his criticisms, however, Dr. Shuman acknowledged that he did not know Dr. Hewell and had never spoken to him about this case. (Tr. 111.) Asked what he knew about Dr. Hewell's handling of N.N.'s case, the only detail Dr. Shuman gleaned from the record was that Dr. Hewell did not review the images until 40 days after the study was completed. (*Id*.) Cross-examination indicated, however, that the delay had no impact on the quality of the images available for interpretation. (Tr. 111-13.) Dr. Shuman was also critical of the quality of the

images generated in the 2005 MRI study (Tr. 26-27), though he nonetheless testified at the hearing that each of the abnormalities he alleged to be present were visible in both the 2011 study and the 2005 study. [23] (*See* Section A above.)

Petitioners argue that Dr. Bauza's findings, on the other hand, are significant confirmation of Dr. Shuman's own opinion. In fact, Petitioners go so far as to claim that Dr. Bauza's conclusions are *superior* to that of either Dr. Shuman or Dr. Wiznitzer, because Dr. Bauza specializes in *neuroradiology*. (ECF No. 94, p. 8.) I note, however, that Petitioners' characterization of Dr. Bauza as a "treating physician" is somewhat misleading. (ECF No. 94, p. 8.) Petitioners argue that Dr. Bauza "was not hired for the purpose of testifying for either party" (ECF No. 92, p. 8), and that N.N. was referred to Dr. Bauza by his treating physician, Dr. Fischer (*Id*, p. 7.). Yet Dr. Shuman acknowledged at the hearing that the decision to do another MRI in 2011 was made at Dr. *Shuman's* behest, after he had begun working with Petitioners' counsel on this case. (Tr. 26.) Moreover, Dr. Shuman indicated that he believed Mr. Webb was in direct contact with Dr. Bauza. (Tr. 116.) Thus, even if Dr. Bauza was not hired for the particular purpose of *testifying*, and even if the referral technically came from N.N.'s treating physician, Dr. Fischer,[24] Dr. Shuman's testimony indicates that Dr. Bauza's MRI study arose not for treatment purposes, but for furtherance of the instant claim. This is enough to cast doubt, not on Dr. Bauza's integrity, but on his neutrality and on his purpose relative to this case.

In any event, Respondent points out that Dr. Bauza's report does not completely support Dr. Shuman's viewpoint. (ECF No. 93, pp. 15-16.) Whereas Dr. Shuman believes that N.N.'s MRI shows enlarged ventricles, Dr. Bauza did not make that finding. (Tr. 116-17.) Dr. Shuman also disagreed with Dr. Bauza's opinion that the asymmetrical size of N.N.'s hippocampi is a normal variant. (Tr. 117.)

In the final analysis, I conclude that neither Dr. Hewell's nor Dr. Bauza's report is entitled to great weight in resolving this case. I reach this conclusion chiefly because of the issues discussed in detail above at pp. 14-22. Although the notations of treating physicians will often be accorded significant weight,[25] in this instance, I find that neither Dr. Bauza's nor Dr. Hewell's reports change my view of this issue.

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[23] Dr. Shuman's criticism of Dr. Hewell and the 2005 image quality may be somewhat blunted, however, in that N.N.'s *CT scan* of July 2005, conducted by a different organization, was also interpreted as normal, which would seem to offer some corroboration for Dr. Hewell's interpretation of the 2005 MRI. (Ex. 6, p. 11.) In his expert report, Dr. Shuman admitted that he has not reviewed the CT scan or the accompanying report, but speculated based on the subsequent MRI that the CT scan must have been abnormal. (Ex. 26, p. 2.) At the hearing, however, Dr. Shuman acknowledged that he had no basis to make that claim. (Tr. 110.)

[24] Furthermore, I note that Dr. Fischer's records also indicate that Dr. Shuman referred N.N. *to Dr. Fischer*, and that Dr. Shuman wrote a note advising Dr. Fischer of his interpretation of N.N.'s first MRI as abnormal. (Ex. 23, p. 1.) Because it appears from these records that Dr. Shuman was attempting to exert influence over Dr. Fischer from the very beginning of her treatment of N.N., I am disinclined to rely on the conclusions of either Dr. Fischer or Dr. Bauza as corroboration of his opinions. To do so would be somewhat circular reasoning, since it appears that those conclusions may have been reached based, at least in part, on Dr. Shuman's own input.

[25] See, *e.g.*, *Capizzano v. HHS,* 440 F.3d 1317, 1326 (Fed. Cir. 2006)(noting that "medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect shows that the vaccination was the reason for the injury.'")

In the first place, the two reports *contradict each other*, one finding evidence of encephalitis in an MRI image, the other not. But *far more important* is the fact that their reports, neither of which exceeds a single paragraph, do not provide any insight into the *reasoning* behind the conflicting interpretations of Drs. Hewell and Bauza. As Respondent points out, it is quite clear from this case that qualified experts can and do differ on questions of MRI interpretation. (ECF No. 93, p. 16.) That much was clear from the reports and testimony of Drs. Shuman and Wiznitzer. That two additional physicians have similarly produced conflicting findings is not in itself enlightening with regard to the question of whether Dr. Shuman or Dr. Wiznitzer has presented superior evidence explaining why one interpretation is correct while another is wrong. Dr. Wiznitzer and Dr. Shuman have not simply reported their ultimate findings, but have submitted their *underlying reasoning* to the scrutiny of the court, through extensive reports and testimony. The same cannot be said of either Dr. Hewell or Dr. Bauza.

Thus, these two additional reports do little more than further highlight the disagreement between Drs. Shuman and Wiznitzer, without providing any further elucidation of the issues involved in MRI interpretation as explained by the competing experts in this case. Ultimately, I find that the conflicting reports of Drs. Hewell and Bauza are substantially outweighed by the testimony of those experts who testified fully, Drs. Wiznitzer and Shuman.

D. Conclusion

Dr. Shuman identified five possible abnormalities visible in N.N.'s MRI scans. Dr. Wiznitzer, however, offered a competing interpretation in each instance, supported not merely by his own experience and expertise, but also by corroborating medical literature. In contrast, many of Dr. Shuman's key assertions regarding N.N.'s MRIs, though undoubtedly made by an expert in the field, were left otherwise unsubstantiated. For this reason, and for all the reasons above, I find that Dr. Wiznitzer was substantially more persuasive than Dr. Shuman with regard to the interpretation of N.N.'s MRI images. I also found that the conflicting opinions of N.N.'s treating physicians, Drs. Howell and Bauza, were outweighed by the experts in this case. I therefore find that Petitioners have *not* established that it is more likely than not that N.N. experienced inflammation of the brain.

IX

CLINICAL SIGNS AND STYMPTOMS

Dr. Wiznitzer also argued that another reason that I should reject Petitioners' Table Encephalitis claim is that, even accepting Special Master Moran's findings concerning N.N.'s symptoms displayed in late November of 2004, N.N. was still not displaying at that time *other* clinical indicators of encephalitis, including seizures, respiratory regulators, or persistent movement disorder. (Tr. 228-29.) Dr. Wiznitzer relied on certain medical articles in this regard.

But it is unnecessary for me to address Dr. Wiznitzer's analysis in this regard, since Petitioners, as explained above, have failed to demonstrate that the *MRI's* showed the existence of a prior encephalitis.

On the other hand, Petitioners argue that the symptoms found by Special Master Moran were the first symptoms of an encephalitis. But their own expert, Dr. Shuman, acknowledged that without *MRI evidence* showing the existence of a prior limbic encephalitis, he could *not*

offer his opinion that N.N. suffered an encephalitis in 2004. (Tr. 109.) And, I have already rejected his analysis of the MRI evidence.

Accordingly, on this record it is unnecessary for me to further address the clinical symptoms that Special Master Moran found to have occurred in 2004. The analysis of the MRI evidence alone fully decides this case.

X

CONCLUSION

The record of this case demonstrates plainly that N.N. and his family have been through a tragic medical ordeal. They are certainly deserving of great sympathy. Congress, however, designed the Program to compensate only those individuals whose injuries or deaths can be linked causally, either by a Table Injury presumption or "causation-in-fact" evidence, to a listed vaccine. In this case, as described above, no such link has been demonstrated. Accordingly, I conclude that Petitioners in this case are *not* entitled to a Program award.[26]

s/ George L. Hastings, Jr.
George L. Hastings, Jr.
Special Master

[26] In the absence of a timely-filed motion for review of this Decision, the Clerk of the Court shall enter judgment accordingly.