

In the United States Court of Federal Claims  
OFFICE OF SPECIAL MASTERS

No. 08-0311V

Filed: 30 November 2010

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JASON and BRIDGET BYERS, parents \*  
and legal representatives of a minor child, \*  
BROOKE BYERS, \*

Petitioners, \*

v. \*

SECRETARY OF HEALTH AND \*  
HUMAN SERVICES, \*

Respondent. \*

\* \* \* \* \*

*William A. Miller, Jr., Esq.*, Hummel, Coan, Miller & Sage, Louisville, Kentucky, for Petitioner;  
*Ryan Daniel Pyles, Esq.*, United States Department of Justice, Washington, District of Columbia,  
for Respondent.

**UNPUBLISHED<sup>1</sup>**

Actual Causation; Trigger; Proximate Cause;  
MMR; *Status Epilepticus*; Stroke;  
Patent Foramen Ovale; MTHFR Mutation

**ENTITLEMENT RULING**

**ABELL**, Special Master:

On 25 April 2008, Petitioners filed this Petition for compensation under the National Childhood Vaccine Injury Act of 1986 (Vaccine Act or Act)<sup>2</sup> alleging that, as a result of the Measles-Mumps-Rubella (MMR) vaccine administered to their daughter Brooke on 9 May 2005, Brooke

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<sup>1</sup> Petitioners are reminded that, pursuant to 42 U.S.C. § 300aa-12(d)(4) and Vaccine Rule 18(b), a petitioner has 14 days from the date of this ruling within which to request redaction “of any information furnished by that party (1) that is trade secret or commercial or financial information and is privileged or confidential, or (2) that are medical files and similar files the disclosure of which would constitute a clearly unwarranted invasion of privacy.” Vaccine Rule 18(b). Otherwise, “the entire decision” may be made available to the public per the E-Government Act of 2002, Pub. L. No. 107-347, 116 Stat. 2899, 2913 (Dec. 17, 2002).

<sup>2</sup> The statutory provisions governing the Vaccine Act are found in 42 U.S.C. §§300aa-10 et seq. (West 1991 & Supp. 1997). Hereinafter, reference will be to the relevant subsection of 42 U.S.C. §300aa.

suffered from *status epilepticus*,<sup>3</sup> which was followed by a hemiplegic stroke (infarction), and ushered in a long-term seizure disorder.

Eventually, a telephonic evidentiary hearing on the ultimate issue of vaccine causation was convened by the Court *in vitro* (telephonically) from the Court's Chambers on 19 February 2009. Hearing Transcript ("Tr.") at 1. Wherein, the Court heard from medical expert witnesses for both parties, neurologists both: Dr. Marcel Kinsbourne for the Petitioner, and Dr. Gerald Raymond for the Respondent. Following those hearings, the parties filed closing briefs with the Court, and the case is now ripe for a ruling.

As a preliminary matter, the Court notes that Petitioners have satisfied the pleading requisites found in § 300aa-11(b) and (c) of the statute, by showing that: (1) they are the real party at interest as legal representatives of their daughter Brooke, the injured party; (2) the vaccine at issue is set forth in the Vaccine Injury Table (42 C.F.R. § 100.3); (3) the vaccine was administered in the United States or one of its territories; (4) no one has previously collected an award or settlement of a civil action for damages arising from the alleged vaccine-related injury; and, (5) no previous civil action has been filed in this matter. Additionally, the § 16 requirement that the Petition be timely filed have been met. On these matters, Respondent tenders no dispute.

The Vaccine Act authorizes the Office of Special Masters to make rulings and decisions on petitions for compensation from the Vaccine Program, to include findings of fact and conclusions of law. §12(d)(3)(A)(I). In order to prevail on a petition for compensation under the Vaccine Act, a petitioner must show by preponderant evidence that a vaccination listed on the Vaccine Injury Table either caused an injury specified on that Table within the period designated therein, or else that such a vaccine *actually caused* an injury not so specified. § 11(c)(1)(c).

## I. FACTUAL RECORD

Despite their accord on certain factual predicates contained in the filed medical records, there is, unsurprisingly, a pronounced conflict between the parties on certain factual issues of viewing understood scientific mechanisms of vaccine injury within the context of the expert witness testimony and the medical records. Considering these disputes and the Court's commission to resolve them, it behooves the Court to explain the legal standard by which factual findings are made.

It is axiomatic to say that a petitioner bears the burden of proving, by a preponderance of the evidence—which this Court has likened to fifty percent and a feather—that a particular fact occurred or circumstance obtains. Put another way, it is required that a special master, "believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the [special master] of the fact's existence." *In re Winship*, 397 U.S. 358, 371-72 (1970) (Harlan, J., concurring). Moreover, mere conjecture or speculation does

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<sup>3</sup> *Status epilepticus* is "1. a continuous series of generalized tonic-clonic seizures without return to consciousness, a life-threatening emergency... 2. any prolonged series of similar seizures without return to full consciousness between them." DORLAND'S ILLUSTRATED MEDICAL DICTIONARY (30th ed. 2003) (SAUNDERS) at 1756.

not meet the preponderance standard. *Snowbank Enterprises v. United States*, 6 Cl. Ct. 476, 486 (1984).

This Court may not rule in favor of a petitioner based on his asseverations alone. This Court is authorized by statute to render findings of fact and conclusions of law, and to grant compensation upon petitions that are substantiated by medical records and/or by medical opinion. §§ 12(d)(3)(A)(i) and 13(a)(1).

Contemporaneous medical records are afforded substantial weight, as has been elucidated by this Court and by the Federal Circuit:

Medical records, in general, warrant consideration as trustworthy evidence. The records contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium. These records are also generally contemporaneous to the medical events.

*Cucuras v. Sec’y of HHS*, 993 F. 2d 1525, 1528 (Fed. Cir.1993).

Medical records are more useful to the Court’s analysis when considered in reference to what they include, rather than what they omit:

[I]t must be recognized that the absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance. Since medical records typically record only a fraction of all that occurs, the fact that reference to an event is omitted from the medical records may not be very significant.

*Murphy v. Sec’y of HHS*, 23 Cl. Ct. 726, 733 (1991), *aff’d*, 968 F. 2d 1226 (Fed. Cir. 1992), *cert. denied sub nom. Murphy v. Sullivan*, 113 S. Ct. 263 (1992) (citations omitted), citing *Clark v. Sec’y of HHS*, No. 90-45V, slip op. at 3 (Cl. Ct. Spec. Mstr. March 28, 1991).

#### A. MEDICAL RECORDS *ET AL.*

There were no issues of circumstantial fact disputed by the parties, and the medical records reveal those circumstances sufficiently. The Court turns first to the recorded facts drawn from the medical records engendered and maintained by those responding to, and treating, Brooke’s condition. The Court gleaned the following from the most pertinent of the medical records:

Brooke was born 15 April 2004. Since birth, she has possessed a “patent foramen ovale” (PFO), which is an abnormal opening between the two atria (upper chambers of the heart), present in everyone at birth, but which is supposed to close up after birth. In a certain percentage of the population, it never closes up, as is the case with Brooke.

On 9 May 2005, Brooke received the MMR, Hep B, Hib, IPV, and PCV vaccines, but the discussion herein focuses primarily on the MMR vaccination. Brooke underwent a well-child visit just 5 days later, on 14 May 2005, at which nothing out of the ordinary was apparent. However, on

18 May 2005, Brooke was taken to the Emergency Room in the middle of a seizure and a temperature of 102.8°F, after being discovered at 9:50 AM that morning with irregular breathing and seizing, after two days of fever. The affidavit of Mrs. Byers noted that Brooke started to run a fever on 17 May 2005, and seemed “clumsy” throughout that day.

That seizure, on the morning of 18 May 2005, started right-sided, but gradually shifted to left-sided over the course of the seizure, which was only halted by a dose of Ativan, and which lasted approximately one hour. After the seizure subsided, Brooke was observed with left eyelid drag, drooping left corner of mouth, and decreased movement of left arm and leg. The medical assessment was complex febrile seizure, with “Todd’s paralysis” remaining. She moved her right side freely, but not the left side: she had slight movement of the left leg, and no movement of the left arm. She had a scattered rash scattered across her torso. The “Todd’s paralysis” did not resolve by 20 May 2005, leading to a neurology consultation. A MRI showed “signal abnormalities involving the right posterior parietal lobe, the right temporal lobe, and the posterior right thalamus.”

The assessment by the Radiologist favored “infarcts over cerebritis” (cerebritis is an infection of the brain, often resulting in an abscess and swelling). Genetic testing for heterozygous C677T mutation was positive, but A1298C mutation tested negative. A brain scan indicated normal intracranial vasculature with no occlusion (blockage) or stenosis (constriction/narrowing of vessels). Also, coagulation studies of Brooke’s blood showed no abnormalities.

A 21 May 2005 EEG rendered abnormal results indicating focal slowing in the right “parieto-occipital region,” which suggests focal cerebral dysfunction. They also observed an electrical seizure, coming from the right parieto-occipital region that lasted 1 minute, 15 seconds. The discharge summary from that visit diagnosed complex febrile seizure with a presumption of “post-infectious cerebritis.”

An EEG on 30 August 2005 was adjudged normal, however, notwithstanding “focal epileptiform discharges emanating from the right hemisphere, especially from the right central parietal region.” Associated with partial seizures. Later that summer, and into the Fall of 2005, Brooke was seen by neurologists for myoclonic jerks. At that time, she had good vocabulary, good comprehension, and good social interaction, although she still evidenced some lasting effects on musculature and walking gait from the prior paralysis. The attending neurologist’s impression was threefold: 1. Right hemisphere stroke from a central nervous system infection, which led to inflammatory vasculitis (swelling of the blood vessels); 2. Focal seizures (apparently of myoclonic type and focal origin) as a sequela of #1; and 3. hemiparetic cerebral palsy.

On 16 October 2005, Brooke went to the hospital in *status epilepticus*, where she was treated with drugs and eventually her seizures subsided. Thereafter, she continued to take multiple anti-convulsants and was discharged on 18 October 2005 with a diagnosis of *status epilepticus* and left hemiparesis. An MRI on 23 November 2005 indicated: 1. some volume loss in her posterior right frontal lobe and right parietal lobe, consistent with a prior infarction; and 2. T2 signal abnormality in the white matter of the right cerebral hemisphere, thought to be either gliosis or under-myelinated white matter related to the prior infarction.

In deciding whether Brooke should receive immunizations in the future, Brooke was seen by an infectious disease specialist who believed it was “very possible that febrile illness was related to immunization,” as fever following measles vaccine occurs in five to fifteen percent of recipients, and typically occurs in the first or second weeks following the vaccination. However, said the infectious disease specialist, in this case, “the absence of pleocytosis is inconsistent with usual description of measles-related CNS disease, as are the stroke-like MRI findings.” The doctor speculated that the symptoms might be due to genetic mutation, but remained unsure. She remained skeptical concerning whether the problems were “truly due to immunization.”

In February 2008, Brooke’s speech rehabilitation therapist noted that she had not had a seizure since 2006, although the affidavits in this case say that she continues to experience myoclonic seizures up to the point of their composition.

The most recent neurologic consultation in the record, on 4 March 2008, diagnosed “post-immunization encephalopathy after immunizations in May 2005,” adding that Brooke then “sustained a stroke and subsequent left-sided hemiparesis.”

#### B. TESTIMONY BEARING ON ENTITLEMENT

The Court greatly appreciated hearing from two eminent scholars on the question presented in this case. Both experts’ testimony is accepted as admissible *in toto*, and the Court reiterates its gratitude for sharing their credible, professional expertise.

##### 1. Marcel Kinsbourne, M.D.

In his filed expert opinion report, Dr. Kinsbourne opined that the MMR vaccine (perhaps in conjunction with other vaccines) caused a fever in reaction, which triggered a febrile *status epilepticus*, during which occurred an acute right hemisphere stroke. He diagnosed the hemiplegia<sup>4</sup> as “neocortical epilepsy, subtype hemiconvulsion-hemiplegia-epilepsy syndrome.”

Dr. Kinsbourne relied upon a published report linking MMR to seizures and/or *status epilepticus* through Measles encephalopathy. Using a diagnosis of exclusion, Dr. Kinsbourne pointed out that there are no other identifiable causes within the medical record for the seizures or the stroke. He opined that genetic mutation was not really an active factor, as it is common and not associated with “detrimental consequences.” A coagulation study was negative, as were viral and bacterial tests, which led him to contradict the postulation of Respondent’s expert’s theory of embolism from a thrombus that broke free from elsewhere in Brooke’s body and lodged in her brain. Dr. Kinsbourne pointed out that there is no direct evidence in the record in support for that, in findings from a coagulation study or clinical symptoms of swelling in the extremities.

Dr. Kinsbourne did not believe that the PFO between Brooke’s cardiac atria was more likely than not the cause of the stroke, based on a filed Mayo Clinic epidemiology study stating that such a condition is common, and is not a substantial cause or heightened risk factor for stroke.

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<sup>4</sup> Hemiplegia is total paralysis of one side, distinguished from hemiparesis, where one side is weakened, but not completely paralyzed.

At the entitlement hearing, Dr. Kinsbourne pinpointed the MMR vaccination as having “either singly or in combination with her other one-year vaccinations caused Brooke to suffer a sustained fever,” which fever “caused her to start seizing, to have seizures,” and that “those seizures caused her stroke,” which “in turn caused her hemiplegia, hemiparesis.” Tr. at 9-10. He explained:

The vaccinations, and particularly MMR, that Brooke received are well-known to be apt to cause a fever after a few days, and that’s an incontrovertible fact. The timing of the fever is consistent with what happens after MMR, and no other cause for that fever was determined by investigations or by anyone.

Tr. at 10.

Dr. Kinsbourne summarized the course of events as follows:

So we have an event, which was a fever seizure with jerking of limbs on both sides of the body, which was described as lasting about an hour. This was detected by the parents. They called the EMS. The ambulance noted the ongoing seizure activity, and it was noted when she arrived at the hospital where it was arrested after about an hour by an injection of Ativan.

So far, all we have is seizure activity. There’s no indication of the stroke. The movements of both sides of the body were in fact very forceful. It was noted that they were more forceful on the left side than on the right side. Now, after the seizure was arrested, we have the first appearance of muscular weakness, which I have already referred to, which was a placid or a hypertonic state late of the left-sided limbs.

That then introduces the stroke aspect of the case. Now, I note that on the 21st of May was an EEG, and the EEG showed the expected slowing of activity on the right side of the cerebral cortex, the right hemisphere, expected because that’s opposite to the side of the paralysis. Already, they noticed epileptic discharges, spikes, and in fact they noticed one that is called epileptic fever.

There was an event noticed on the EEG, which represents seizure activity, although at that point abnormal movements were not observed. In fact, the report mentions that the child was lying on her left side, so any jerking on the left, which would be expected from the seizure focus on the right might have been completed by gravity as it were.

So ... the seizure activity was not one event, but [] there was now a seizure tendency certainly along a seizure threshold, and in fact subsequently, as the record reflects, Brooke had multiple so-called myoclonic seizures, which sometimes originated on one side of the brain and affected the other side of the body, and sometimes they’re reversed. In other words, she had a myoclonic epilepsy. That was treated with the appropriate antiepileptic agent and I think was treated successfully for the time being.

THE COURT: Dr. Kinsbourne, would it be your analysis or not that this first infamous seizure started as a generalized seizure and then over time became more focal?

THE WITNESS: Well, the description is of a generalized seizure. The observation that the activity was more marked on the left was made sometime during the course of the seizure. I don't know whether it was the case or whether a few just observed them in what they happened when. It's not exactly focal because there was jerking on both sides, but one would say there were focal features in that the jerking was somewhat asymmetrical.

THE COURT: Okay. Would this suggest to you that the presumed stroke occurred about this time or later, or what does it suggest to you?

THE WITNESS: I don't believe that the child had the stroke during the seizure. I think the child had the stroke as a result of the seizure. During the seizure, in fact her limbs were very active on both sides. And there was an interesting feature which is pertinent to your question, Special Master, in the EMS report, and the ambulance report, it appears that although Brooke was having seizures on both sides of the body, it's usually related to an unconscious individual. She was actually responsive, and they even make the comment that her grip strength was preserved, normal, which is certainly quite inconsistent with her having a stroke or having had the stroke at that time.

Tr. at 15-18. Dr. Kinsbourne did agree that a seizure event at the level of *status epilepticus* can cause or trigger a stroke, although it is uncommon. Tr. at 19.

Regarding whether the febrile seizure preceded the stroke, or vice versa, Dr. Kinsbourne pointed out the absence of any mention of a stroke or stroke-associated symptoms within the contemporaneous medical records. Tr. at 11. He explained:

[N]obody saw any stroke of any kind before the child's admission. There was no paralysis on one side of the body, and nothing was noted about that being the case. The second is that in the medical record nobody, not any of the treating physicians, refers to the stroke as having occurred before the seizure. In fact, repeatedly the description is that there was a seizure and then there was the hemiplegia, which in fact is what's recorded in the records. Dr. Raymond [in his expert report] mentioned a remark by Brooke's mother that she was clumsy on the 16th, and this was when the mother was asked at the admission to think back on any event that might at all be pertinent, and that was a day on which the child was healthy. She'd only just begun to learn to walk, and being clumsy has no resemblance whatever to having a stroke.

Tr. at 12-13. He countered Dr. Raymond's opinion, stating:

Dr. Raymond points out that there are several aspects of the MRI, the one that he finds critical is called the T-2 weighted films. Now, in the report, it is said that both the diffusion weighted and the T-2 weighted aspects were clearly abnormal, hyperintense is I think the description, and this was done two days after the event that we've just discussed. Now, in Dr. Raymond's opinion, two days would be too short for the T weighted films to show hyperintensity, and therefore he feels as he put it that the stroke must have occurred several days earlier. In my opinion, that is not the case, that it is true that in the first 24 hours the T-2 weighted evidence is going to be

spotty and unclear, whereas the diffusion weighted evidence becomes clear within hours. However, by the second day already one sees T-2 hyperintensity, and one certainly does, one certainly sees that.

Tr. at 18-19.

Dr. Kinsbourne also addressed Respondent's contention that genetic predisposition is a superseding cause of the seizure disorder, inasmuch as Brooke possesses a heterozygous C677T genetic mutation:

[This] is a mutation which if it were homozygous, meaning basically coming from both parents, it would render an individual [susceptible to] having strokes. It wouldn't mean that the individual would necessarily have a stroke. It would be a so-called risk factor. On the other hand, if it's heterozygous as it is in Brooke's case, it has not been shown to be a risk factor. Now, from my point of view, neither of these scenarios [bears upon] my opinion. Obviously, it's not risk factor. It's irrelevant. If for some reason it were a risk factor, or we really can't accept that, so that might explain why Brooke was at risk. After all, not every child who gets MMR has a *status epilepticus* and a stroke, so undoubtedly there has been something about the child, which made her vulnerable to this, which obviously the vast majority are not. In other words, determining risk factor really does not contradict the kind of causation opinion that I'm giving.

Tr. at 20-21.

Dr. Kinsbourne had a similar opinion regarding Brooke's patent foramen ovale:

I see no evidence that it has any impact. The PFO is quite a common condition. Some articles think that is a risk factor for embolic stroke, and others deny that, and the fact is that many, many children have PFOs, and they don't have stroke, and in order to support the view that there was an embolism, in other words a clot, going through the opening and upper carotid artery and blocking say the middle three. Roughly, if that is the [putative] mechanism of the stroke, which this child had, then there needs to be some evidence for that, but I haven't seen any, and so far as I know, this is speculation.

Tr. at 21.

Next, Dr. Kinsbourne reviewed the medical literature Petitioners filed in support of his expert opinion, and upon which his opinion relied. The first such resource was from a website,<sup>5</sup> and set forth the clinical manifestations and aetiology of so-called "hemiconvulsion-hemiplegia syndrome," which Dr. Kinsbourne believed described a course very similar to Brooke's:

The first sign of the syndrome is a sudden, prolonged hemiconvulsion in the form of status. It occurs in a child without antecedents, between 5 months and 4 years old, with a peak incidence during the first 2 years of life. A febrile episode is almost

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<sup>5</sup> Available at: <http://www.ilae-epilepsy.org/Visitors/Centre/ctf/hemiconvulsion.html>.

always associated, but in many cases, no cause is obvious. The onset of convulsions may pass unnoticed, the child being discovered convulsing in bed.

If untreated, hemic convulsions may last for several hours. They present as predominantly unilateral clonic jerks. The rhythm is variable, and the jerks can be asynchronous. Impairment of consciousness is not a permanent or even constant feature. In long-lasting convulsions, jerks may diffuse to the opposite side or change sides. Aversion of head and eyes may be observed, sometimes even before the occurrence of jerks. Autonomic symptoms (hypersalivation, cyanosis, etc.) may be associated.

Massive flaccid hemiplegia immediately follows the unilateral seizure. When jerks change sides, it is usually the side involved last that remains hemiplegic. Evolution of hemiplegia is variable. It can either remain as a permanent neurologic deficit with signs of spasticity or decrease progressively, leaving behind a slight hemiparesis. To differentiate hemiplegia from Todd paralysis, a minimum duration of 7 days is arbitrarily set. In more than 80% of the cases, the hemiplegia is permanent. Rarely, it may disappear completely, although some degree of spasticity and pyramidal signs usually persist. In contrast with congenital hemiplegia, the face is constantly involved, and aphasia is present in left-sided cases.

In almost 80% of the cases, partial epilepsy will develop 1 year to 3 years later (hemic convulsion-hemiplegia-epilepsy syndrome). Partial seizures with secondary generalization and episodes of status are not uncommon. In addition to partial epilepsy, most of the children having experienced a hemic convulsion-hemiplegia-epilepsy syndrome present with some degree of mental impairment....

...Prolonged clonic convulsions with a marked unilateral predominance usually occur in the course of a febrile disease. Causes of the initial convulsions are multiple. A number of acute cerebral disorders have been occasionally related to the occurrence of the syndrome (meningitis, subdural effusions, head trauma, etc.). In many cases, no cause is obvious (idiopathic hemic convulsion-hemiplegia-episode syndrome), and such cases may represent only prolonged febrile convulsions that do not otherwise differ from common febrile convulsions. In such cases the seizure activity itself could be responsible for the appearance of new lesions occurring in a previously normal brain. Alternatively, the presence of a preexisting asymptomatic lesion of perinatal or prenatal origin may be responsible in a number of cases (symptomatic hemic convulsion-hemiplegia-episode syndrome) for the initiation or localization of the seizure. The prolonged seizure would then produce or contribute to the development of irreversible brain damage with resultant partial epilepsy.

The role of long-lasting febrile convulsions, as part of a hemic convulsion-hemiplegia-episode, in the genesis of hippocampal sclerosis and consequent mesial temporal lobe epilepsy remain disputed. A statistical association is well demonstrated, and there are strong arguments in favor of an etiological

relationship; however, the presence or absence of other contributing factors is not clearly established. Furthermore, partial epilepsy in hemiconvulsion-hemiplegia-episode syndrome can be temporal, extratemporal, or multifocal.

Pet. Ex. 16, Tab B, at 2-3. Dr. Kinsbourne summarized that “the important point is in such cases, the seizure activity itself could be responsible for the appearance of new lesions occurring in a previously normal brain, and that in my opinion happened in the case of Brooke Byers.” Tr. at 24.

Another article of significance in Dr. Kinsbourne’s mind was Pet. Ex. 16, Tab D, W. Allen Hauser, *Status Epilepticus: Frequency, Etiology, and Neurological Sequelae*, 34 *ADVANCES IN NEUROLOGY* 3-14 (1983). He cited it “in support of the proposition that *status epilepticus* is known to be capable of inflicting acute brain damage.” Tr. at 25. That source states, in relevant part:

*Status epilepticus* alone has been implicated in the production of brain damage in several neuropathological studies. Also, there have been individual case reports of cerebral atrophy following *status epilepticus*, and in two instances serial documentation of cerebral atrophy following *status epilepticus* was provided.... Most studies have reported a positive association between adverse outcome and duration of *status epilepticus*. The proportion of cases with adverse outcomes of all types tends to increase with increasing duration of *status epilepticus*.

Pet. Ex. 16, Tab D, at 6-7. Dr. Kinsbourne also briefly discussed the classic Menkes textbook, *CHILD NEUROLOGY*, to reiterate the penultimate sentence of this passage, which represents the medical consensus that “the more prolonged the status, the worse the outcome.” Pet. Ex. 16, Tab F, at 2; Tr. at 27. Dr. Kinsbourne also relied on that source for a description of “the mechanism by which the status can damage the cortex, and that is the mechanism that I believe occurred in the case of Brooke Byers.” Tr. at 27, discussing Pet. Ex. 16, Tab F, at 2.

The next study discussed was Pet. Ex. 16, Tab G, R. K. Mondal *et al.*, *Hemiconvulsion, Hemiplegia, Epilepsy Syndrome and Inherited Protein S Deficiency*, 73 *INDIAN JOURNAL OF PEDIATRICS* 157-159 (2006). Dr. Kinsbourne stated that he relied upon that study for the proposition that *status epilepticus* can cause permanent sequela, including hemiparesis and persistent epilepsy. Tr. at 27-28. The first three sentences from the abstract of that study reads:

Prolonged focal *status epilepticus* may be followed by permanent hemiplegia, the hemiconvulsion, hemiplegia, epilepsy syndrome (HHE syndrome) in some children. The HHE syndrome is usually associated with atrophy of involved cerebral hemisphere. The pathogenesis of HHE syndrome is likely to be due to histotoxic epileptic brain damage following prolonged focal convulsion due to any cerebral insult.

Pet. Ex. 16, Tab G, at 1.

Another filed article examined the possibility of a relationship between the Measles component of the MMR vaccine and acute encephalopathy, ultimately concluding that the results “suggest[] that a causal relationship between measles vaccine and encephalopathy may exist as a rare complication of measles immunization.” Pet. Ex. 16, Tab J, Robert E. Weibel *et al.*, *Acute*

*Encephalopathy Followed by Permanent Brain Injury or Death Associated with Further Attenuated Measles Vaccines: A Review of Claims Submitted to the National Vaccine Injury Compensation Program*, 101 (3) PEDIATRICS 383-387 (1998). Dr. Kinsbourne pointed out that, in that study, “34 of them had the onset of the MMR-related encephalopathy with seizures [among which were] 17 with *status epilepticus*.” Tr. at 29.

The remainder of the articles were general supports for the idea that *status epilepticus* can cause neuronal damage in the brain, leading to long-lasting problems, or, as Dr. Kinsbourne put it, “These articles support each other in showing the viability of the idea that the *status epilepticus* in Brooke’s case caused the damage to the hemisphere which caused the hemiparesis.” Tr. at 28-29.

On cross-examination, Respondent asked Dr. Kinsbourne why certain indicia commonly observed with HHE were not witnessed in Brooke’s case, to which Dr. Kinsbourne replied that doctors caught and treated Brooke before too much time had elapsed, and the damage was mitigated. Tr. at 42-43. When asked, in a similar vein, why initial cytotoxic edema was not observed in the MRI taken two days after the seizure, Dr. Kinsbourne replied that “Brooke had only a limited atrophy because she [had] a milder case” of edema. Tr. at 43.

Respondent raised an article filed in support of Dr. Raymond’s opinion, Resp. Ex. A, Tab 11, Jeremy L. Freeman *et al.*, *Hemiconvulsion-Hemiplegia-Epilepsy Syndrome: Characteristic Early Magnetic Resonance Imaging Findings*, 17 (1) Journal of Child Neurology 10-16 (2002), and asked Dr. Kinsbourne to comment on the following passage:

The finding of diffusion-weighted imaging abnormalities indicating cytotoxic edema of the epileptic hemisphere in our patients with [HHE], together with the experimental and clinical reports of restricted water diffusion resulting from *status epilepticus*, gives additional weight to the idea that prolonged focal febrile seizures are injurious to the brains of infants and children. It seems reasonable to conclude that the decline in incidence of hemiconvulsion-hemiplegia-epilepsy syndrome in the developed world may be the result of timely and effective treatment of prolonged seizures.

*Id.* at 16.

Dr. Kinsbourne’s reaction to the passage was to say, “it does refer to what I was saying ... My point is that this is a case in which the seizures were treated relatively timely, so the syndrome is not as severe as described in the peak cases.” Tr. at 45. Moreover, Dr. Kinsbourne wished to draw the Court’s attention to one of the patients studied in that case:

Now, I would like to draw the Court’s attention to the description of Patient No. 3 in this article, which is on the second page of the article. It’s on the bottom of [page] 11.

...I’d like to read that out. “One week prior to her presentation, this previously well in development and normal 12-month-old girl had received the measles, mumps, rubella vaccine. She was found in her room by her mother early one morning having undergone focal convulsions of the left side of her body. Seizures subsequently

became generalized with this duration here of 10 minutes, stopped for five days at the time. She was febrile, and she had a plastic left arm and leg.”

Now, this case is so much like Brooke in some respects. It also referred to when the authors point out that only two of the three patients that they were describing had extensive atrophy. Now, that’s on the first page of the article on the column on the right side. At the head of that, just after it says Figures 1 and 2, it says, “Two patients have shown progressive and extensive atrophy of the involved hemisphere.” I’ve just laid you out the case of the third patient, who didn’t show this progressive -- atrophy, and she’s -- included in a description of the syndrome.

Tr. at 45-46.

Respondent’s following questions queried Dr. Kinsbourne on the differences between Brooke’s case and the classical description of HHE (specifically CT scan findings), to which Dr. Kinsbourne demurred generally:

My comment is that it should be very clear that my opinion is not contingent on the definition of the syndrome. I quoted the syndrome to show that the kinds of mechanism of injury, which I’m proposing for Brooke Byers, has been well-documented in the context of this syndrome. I have no stake in Brooke being included in any future series or publications on this syndrome. The fact is that severe convulsions can cause hemiplegias, and did so in her case.

Q So are you saying that Brooke doesn’t necessarily have HHE?

A I’m saying that in my opinion Brooke has a mild form of HHE, but my opinion isn’t critically bound to the fact of whether Brooke has a mild case of HHE. What Brooke has is so clear, and the connection between events is so obvious that one doesn’t have to, in my opinion, extensively debate on whether she has or has not got HHE.

Q Doctor, if your opinion is not bound to the diagnosis of HHE, then why isn’t it just as likely that Brooke had an ischemic infarction?

A Well, that sentence made no sense to me. I don’t see how the two parts of it fit together. I am pointing out that there is a clear mechanism illustrated by the phenomenon described as HHE of a severe prolonged seizure causing a stroke. That’s pertinent to my opinion because it’s also my opinion that the severe prolonged seizure she had was caused by a fever, and the fever was caused by the MMR. Now, the idea that in spite of all these events occurring, Brooke had a stroke caused by some other unknown or speculative mechanism, I don’t find that helpful to my opinion.

Tr. at 48-49. Dr. Kinsbourne’s point was similar on rebuttal when, after hearing Dr. Raymond’s analysis describing how Brooke Byers did not fit the profile of HHE, he stated that “[his] conclusions regarding the medical causation and chain of events, the mechanism by which this stroke and the subsequent neurological damage occurred [were not] dependent upon whether Brooke [suffered from

the HHE Syndrome],” adding, “My opinion is that the prolonged seizure caused the hemiplegia, and I pointed to HHE as a syndrome in which that sort of thing happens. That’s all.” Tr. at 108.

On redirect examination, Dr. Kinsbourne clarified the chain of causation, in his opinion, that led to Brooke Byers’ injury:

THE COURT: [O]ne question I would ask just for our purposes, how did the vaccine cause or trigger the stroke? Directly, or did it not?

THE WITNESS: Well, it did so by launching a chain of events. The vaccine caused the fever. The fever caused the seizure. The seizure caused the stroke.

Tr. at 53.

## 2. Gerald V. Raymond, M.D.

In his expert report, filed by Respondent in this matter, Dr. Raymond opined that the initial seizure and hemiplegia were secondary to brain lesions (observed after the seizure on MRI scan), as opposed to the seizure causing the lesions and the hemiplegia. Dr. Raymond’s position appears to be based largely, if not entirely, on MRI findings—to wit: lesions were noted to be both bright and hyperintense on the diffusion-weighted imaging, and T-2-weighted imaging indicated that they were at least “several” days old. Dr. Raymond’s position is that if the lesions were ischemic lesions secondary to the prolonged seizure, they would have shown up bright on diffusion-weighted imaging, but absent from the T-2-weighted imaging.

Dr. Raymond perceived Brooke’s abnormal PFO to be a likely source and a significant risk factor for spontaneous stroke, and his expert report provides the biologic mechanism for “paradoxical embolism” arising from a thrombus originating at the PFO and being loosed by “atrial pressure.” He added that even trivial events, such as crying or coughing, could be enough to affect atrial pressure and knock such an embolism loose.

Dr. Raymond theorized a bit less firmly that Brooke’s possession of the heterozygous the C677T mutation could be a risk factor for stroke, but he admitted that there was “no clear evidence” that having only a single copy of the C677T mutation can lead to increased risk of stroke. He added that there is no reported association linking the C677T mutation to fever, adverse vaccine reactions, or febrile convulsions.

At the hearing, Dr. Raymond restated his many credentials of expertise, including his board certification in pediatric neurology and clinical genetics. Tr. at 56. He discussed his experience in treating children with pediatric strokes, which he stated were at least as common—if not more so—than brain tumors. Tr. at 57-58. He described his experience with one case that had been labeled HHE:

Well, it was a child who already had a seizure disorder, and presented with sort of a catastrophic presentation following influenza. And she developed a hemispheric swelling, seizures and was thought even more devastated than she was previously, and then there was hemispheric atrophy that followed that.

Tr. at 58.

Dr. Raymond's opinion on Petitioners' claim of vaccine-related injury was as follows: "It's my opinion that the MMR did not cause her hemiplegia or if we're going to use it interchangeably, hemiparesis, her seizures, or her subsequent course." Tr. at 59. On "the key question ... [of] whether Brooke had a stroke that precipitated her seizure or whether her seizure precipitated her stroke," Dr. Raymond stated, "It's my opinion that the onset of this is the stroke, and that is manifested by the seizure so that if you look at pediatric strokes, the numbers vary, but up to a third of them, unlike adult populations, are reported to have their herald as being a seizure." Tr. at 59-60.

As stated in his expert report, Dr. Raymond reiterated that he relied most upon the post-seizural MRI findings to reach his conclusion, but drew on other sources as well:

Well, I think the things that are most clear is her subsequent MRI, which indicates a focal lesion here in the right parietal occipital area. The other thing, that is the basis of what her seizures come from. It's the basis for her hemiparesis. It is very well formed two days into this and that there is some indication from the record that she may have had some mild motor events a day or two before she presented.

Tr. at 60. Dr. Raymond inferred these "mild motor events" from "the mother's testimony that on the 16th she was more clumsy than usual." *Id.* He elaborated on this point:

Now, granted none of us examined her, and the degree of clumsiness I'm just going to have to take it for what it is. But we do know from when we take a clinical history based on strokes and ischemia infarctions, that certain types of strokes can have a stuttering onset or can have transient ischemic attacks, transient ischemic events prior to the episode. So if you were taking this history and trying to go back and say well, did we get any clues about this, you would note that history.

Tr. at 60-61.

The central piece of evidence upon which Dr. Raymond relied for his opinion was the results of the first MRI, conducted 20 May 2005, two days after Brooke's admission, which state, "There are focal areas of signal abnormality involving the right posterior parietal lobe, the hippocampus of the right to frontal lobe and the posterior right thalamus. These areas are each bright on the diffusion sequence and on T-2 weighted sequences." Tr. at 61. These findings lead Dr. Raymond to the following conclusion:

[I]t's my opinion that contrary to what Dr. Kinsbourne was indicating, already there is evidence of abnormality on the T-2 weighted signal, which is not a sort of postseizure event. If we're saying that she seized two days ago, the fact that she's bright on diffusion, which is sort of the initial ischemia or low blood flow, and then the fact that we already have T-2 bright signal indicates to me that this is most [consistent] with an acute infarction.<sup>6</sup>

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<sup>6</sup> Dr. Raymond explained the MRI results in relation to what they communicate about the physical reality of the brain:

When we do MRIs, we're doing several things, and it's not just one study. You get kind of used to looking at CT scans, which is just using X-ray. What MRI sets out to do is that it uses radio waves

Tr. at 61-62. He next discussed whether “there any indication that she has any global hemispheric issues” apparent in the MRI findings: “[Brooke did not] have a hemisphere of swelling ... She has some swelling in the area of infarction on the T-1 weighted images, but there’s no signal abnormality there. Tr. at 62.

Another test was less supportive of Dr. Raymond’s position, but he explained:

[T]he MRA, or the magnetic resonance angiography, which is a way of looking at the arterial circulation does not show any abnormality, but that’s not unusual in pediatric events. It just means that if this was an embolic event, it occurred. It broke up. It went more distantly, and we’re not picking it up on the MRA. Whether you would have seen it with an angiogram, which is actually where we inject contrast dye or not, I don’t know the answer. The second thing is she did not get an MRV, which is the magnetic resonance venogram. But based on this, she has a very focal lesion on MRI, which is consistent with ischemia infarction and not consistent with HHE.

Tr. at 62-63. This was similar to his explanation of normal findings in the CT scan performed:

There was a CT scan done here, and I believe the initial report was that it was normal.... [That] supports my opinion in the sense that this is consistent with again ischemic infarction, and that this is not consistent with HHE.

Tr. at 64-65. Likewise, regarding the coagulation studies that Brooke underwent, Dr. Raymond stated, “They’re appropriate studies, and they do not show her as having any coagulopathy that would clearly raise her risk.” Tr. at 74.

Dr. Raymond next discussed the findings of the second MRI, performed in August of 2005, some three months after the events in question. Tr. at 65. He described how those findings were consistent with the occurrence of a stroke, which Petitioners do not dispute had occurred in May 2005. Tr. at 66. Describing the results, and the interpretation of the radiologist, he stated:

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in specific pulse sequences to in a sense question the tissue and get answers back, so depending on what the sequences are, you get a series of responses, especially if it’s been injured.

MRI using radio frequencies is mainly looking at the water molecules, and what the specific sequences do is they are pulsing and then receiving. And so when you look at something like diffusion, which is very sensitive to the vibration of water molecules along the myelinated axons, there you’re looking for immediate swelling, which is very sensitive to injury, so bright on diffusion is typically seen within hours of a stroke.

It’s then followed in 24 to 48 hours by brightness on the T-2 weighted images, which is another way of looking at the myelin specifically as well as the other status of the tissue. Now, the T-1 weighted images, which show a little swelling here are not going to then show injury until many days, to weeks, possibly months later in that region if it’s truly been injured in a stroke.

This is in contrast to something like CT which we know very well know you can have an ischemic lesion leading to death of the tissue such as infarction, which is the pathologic basis of a stroke that you’re not going to see anything on CT scan.

Tr. at 63-64.

There's no other focal lesion there, so we're basically seeing a very focal event that has now evolved into a focal scar. And the radiologist's opinion, and what I would concur with, is that it's consistent with the results of the infarction which occurred some time ago in that right frontal parietal region. It's not consistent with what has been reported in HHE in terms of sort of a global event, and it's consistent with an embolic event.

Tr. at 66.

Dr. Raymond contrasted those results with what he would expect with a complex febrile seizure:

When we talk about complex febrile seizures, we're talking about either those that are focal or those that are prolonged as well as some other aspects to them. But if you're asking about where has been the issue in terms of injury to the brain in febrile seizures, people have typically focused on the medial temporal sclerosis or the area around the hippocampus, and that's not an area that's here involved in the permanent scar that Brooke has.

Q Okay. When medial temporal sclerosis is not present, does the medical community agree that complex febrile seizures in and of themselves can cause epilepsy?

A No. I mean, if you're asking for sort of unsymptomatic complex partial seizures, and I'm backing this question out so that I'm happy with my answer, is that when you start to talk about febrile seizures, there are individuals who will break them down into those that are symptomatic and those that are unsymptomatic. By symptomatic, you end up with a whole mixed group of individuals who've asked where their presentation for meningitis was or their presentation for a brain tumor or presentation for a stroke. And so when you remove those, and you just talk about those that are idiopathic, then I think most of the community would say, no, if you don't have medial temporal sclerosis, you don't have an injury just from a prolonged febrile seizure. This would be a typical case where you would go in and say if you were keying this in and trying to do an epidemiologic study of complex febrile seizures, you would remove this case because she's got a stroke. This would never meet your case ascertainment.

Tr. at 67-68.

Last among the tests conducted to assess Brooke were the EEGs, performed 21 May 2005 and 30 August 2005:

[The first is] very consistent with a focal lesion in the right parietal occipital. They're slowing over the right compared to the left. They report it as the right occipital, which is a little bit more posterior, and there are also some spike wave discharges that are coming out of the region also, and this is very consistent with an acute focal injury in the region that we see on MRI.

...And [the second] EEG, the interpretation once again it's abnormal because of the presence of a focal epileptiform discharge emanating from the right hemisphere from the right central parietal region, once again concurring right with the area that was injured as we see it on the MRI, with the MRI reports. This is not consistent with something that would be seen in medial temporal sclerosis.

Tr. at 68-69.

Dr. Raymond does not agree with Dr. Kinsbourne's representation of medical consensus that complex febrile seizures can precipitate a stroke:

My opinion is that complex febrile seizures do not ordinarily precipitate strokes. I mean, it's just something that doesn't happen. The converse is a sudden arterial or vascular occlusion can result in someone's presentation, and someone's presentation can be heralded by seizure.

Tr. at 69. This led the Court to a query:

THE COURT: What, in your opinion, did cause or trigger the stroke or the clot?

THE WITNESS: Special Master, I think if I could answer that definitively, we probably wouldn't be having this conversation. But you have to understand, in pediatric strokes, we often do not find a clear indication, even in adult strokes, but more so in the pediatric field. Many of them are idiopathic. I should be careful.... What I would say though is that when we look at events, I'm torn here between an arterial event with an embolism so that she may have had a clot, and that pulled off to the right parietal or peripheral region, or she may have had a venous event. And I don't have the MRIs in front of me, and frankly because of the limits of the workup, I can't further discern which would weigh more in my opinion.

Tr. at 69-70.

The Court also questioned Dr. Raymond about the circumstances of a PFO-related stroke, and what could have precipitated the stroke in Brooke's case:

THE COURT: If it was a PFO-related stroke, ... what knocked the PFO loose?

THE WITNESS: There's probably shunting that occurs. For instance, if I have a PFO,<sup>7</sup> there's probably mild shunting that occurs throughout the day or throughout

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<sup>7</sup> Dr. Raymond described the PFO (patent foramen ovale) as follows:

Well, a patent foramen ovale is, you have to understand, the embryologic circulation. We all have this opening between our atria so that before our lungs are expanded at the time of birth, that the blood predominantly shunts from right to left and does not enter our forming lungs because it just would serve no purpose. At the time of birth, there is some wonderful physiologic events that go along with that first breath the baby takes, and one of the things is reversal in the pressure so that now the lungs open up, and the blood pressure of pulmonary arteries is such that the blood can now go that way, and this flap so to speak closes down. Now, it then should scar, seal over, and as has been pointed out in this hearing as well as in the articles we are all citing, in probably about 24 percent of individuals even in the adult population, there may be still a potential opening between those two chambers so that

events of the day, so something as simple as bearing down for a bowel movement, crying, I don't know if you have any recent experience with toddlers, ...[but] at any point in time [they] may be fussing, so these are all events, and then there's then the matter of chance is that if there is a little platelet clot or something, that can literally trigger a small cascade that will lead to a clot being carried out. Now, given a healthy circulation, such as Brooke would have, that clot would probably quickly form. It would precipitate a little bit, and then it would probably start to break up, and so the fact that we don't see something on MRA is really not pertinent because the times of sorts would have grabbed that would have been probably soon after the event occurred.

THE COURT: A prolonged seizure, such as *status epilepticus*, could that have triggered it? Could that have knocked it loose?

THE WITNESS: No. I'm not aware of anyone who sort of kind of came back and tried to put that event before the strokes, so we're usually talking about events that have now precipitated a stroke, have injured the brain and it would be a little bit far-fetched for me to then say okay, she had the epilepsy. That propagated an emboli, which we don't typically see, and that was the sequence of events. I just find that just much less satisfactory.

Tr. at 70-72.

While conceding that Brooke's MTHFR genetic mutation was heterozygous, not homozygous, Dr. Raymond still believed that a genetic predisposition could be a causative factor:

I felt like I needed to address it. When you look at pediatric strokes, one is often struck by being handed multiple risk factors. Even pediatric stroke experts often come to the realization that you've got a whole bunch of things lining up on one side, but why do they all line up as in this particular circumstance versus another can't be said. If you look at the MTHFR, I'm not someone who does direct research in that field. I recognize that it is a controversial topic on whether heterozygotes specifically carry a risk. Some papers have said maybe in pediatric stroke they do. Others have said based upon the adult literature, we feel that they don't. It's noted in that aspect that she is heterozygous for that alteration. And when you're looking at pediatric stroke, I think you just have to say I don't know. I can't answer to your satisfaction what role that played in setting it up for this event.

Tr. at 73-74.

Dr. Raymond's position seemed to consist of pooling the conditions that might be, but have not been implicated as risk factors, and viewing them in the aggregate to conclude that they worked in combination to induce a spontaneous or idiopathic stroke:

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periodically shunting can occur not only between the systemic side of the circulation, left to the right, but also if you reverse the pressures from the right to the left.

Tr. at 72-73.

Yes, I think it is significant [that this is a child who has these risk factors] because ... when you're talking about pediatric strokes, they're a much more heterogeneous quality. When we're looking at risk factors in the pediatric population, they're going to be different than the adult population. So, putting the Mayo Clinic article in context, and I guarantee that will not be the final word on PFOs because other groups will have a different take on that, but when you look at this, you're talking about the risk in adults. There, that risk of a PFO may be very small in an adult who's carried it for 70 years of age and now you're trying to implicate that in their onset of an embolic event when cardiac disease is a much higher risk factor from ischemic disease or atrial fibrillation or something along that line. When you talk about the pediatric population, there you have to bring up the fact that we've got individuals with hemoglobin abnormalities, congenital heart disease and sickle cell disease and trauma and infections and all of these sort of raise new and sort issues that are much less common in the adult population. When you actually look at some recent reviews for the role of evaluating pediatric stroke, they bring back to the point that congenital heart disease so that certainly early on it probably still is a significant type of abnormality in the pediatric population setting them up for these events.

Tr. at 74-75.

Dr. Raymond then reviewed the medical literature Respondent filed in support of his position, and upon which he ostensibly relied in forming his opinion. Tr. at 76 *et seq.* The first four of those articles suggested an implication of PFO-associated "paradoxical embolism" in cases of "ischemic stroke without identifiable causes" or "cryptogenic strokes that we see in children." Tr. at 76-78. Those were cases of isolated and unexplainable strokes, and there were no seizures reported in those cases. Tr. at 77. The Court queried further in discussing one of those articles:

THE COURT: ...How about also on [the] Agnetti article on the first page in the right column. Let's see. "The proposed mechanism is a paradoxical embolism through the PFO when the pressure in right atrium increases, i.e., during physical exertion so creating a right to left shunt. This relationship becomes stronger when it is possible to find a source of the venous thromboembolism or to identify genetic coagulation abnormalities predisposing to thrombosis." Do we have either of those situations here?

THE WITNESS: No, we don't. We don't have a documented thrombotic event, and we don't have a documented coagulation abnormality.

Tr. at 77-78.

Dr. Raymond (unpersuasively) attempted to link one article's description with the facts in the instant case:

In the Benedik paper, two-thirds of patients with truly otherwise cryptogenic strokes had PFOs. Some of them were preceded by transient ischemic attacks, which is a

transient, nonpersistent neurologic abnormality such as potentially clumsiness<sup>8</sup> a few days before the event.

Tr. at 78.

The literature was similarly equivocal in its discussion of the heterozygous genetic mutation implicated in this case:

The Lynch paper, which is No. 5, raises the issue of MTHFR and 12 out of 56 in stroke-acquired sefolate, and some of those individuals were just heterozygous. As I said, I think the jury is out. I think that there's going to be probably back and forth on this for quite some time until we understand stroke in the pediatric population better.

Tr. at 79.

This discussion of the medical literature led the Court to ask:

THE COURT: Okay. Now, let me ask in reference to those because I presume this would be *apropos*, in your medical literature, are there any studies that have recorded hard data rather than simply the hypothesis thereto of PFO-related pediatric strokes?

THE WITNESS: Special Master, I do think the literature I pointed to is hard data, but if you're asking is there someone who's addressed specifically the question of the epidemiology of PFO in pediatric stroke, no there is not.

Tr. at 80-81.

Dr. Raymond explained why he did not think Brooke fit the syndromic profile of HHE:

The basis for my opinion is the description of HHE, which is a hemispheric event and does not coincide with the vascular territory, which Brooke had, so that the classic description of HHE, which is an older description, is the onset of either a generalized or focal seizure, which then imaging has a characteristic edematous pattern on CT because that was the original way they looked at it, or MRI involving the entire hemisphere with edema and bright signal and T-2 and possibly abnormal signal. Then, you see the onset of a focal seizure disorder with profound atrophy of the entire hemisphere.

Tr. at 84-85. Dr. Raymond explained the scan results that indicated primary involvement of parietal occipital lobe by stating, "I think it just goes back to the focality of Brooke's lesions versus this sort of global hemispheric pattern that we typically see in HHE." Tr. at 85. He also explained the references to "cytotoxic edema" in the literature discussing HHE syndrome: "We don't completely

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<sup>8</sup> Regarding whether clumsiness could be a symptom of stroke, Dr. Raymond stated:

Absolutely. Absolutely. As I indicated, the clumsiness if she was weak on that side, and it was a transient event, that could be the first symptom or sign of the impending stroke.

Tr. at 82.

understand the etiology of it, but when they're talking about cytotoxic edema, they're talking about all the cells are all in that hemisphere being swollen and edematous, not something you would see with a focal pattern." Tr. at 85. He elaborated further:

The actual etiology of HHE is not well understood. People have broken it down into I think it's the Mondal article. People have broken it down into symptomatic versus again idiopathic, and when you look at the symptomatic causes, it can be everything from again a widespread vascular event, coagulopathy infections, and the hallmark of it is that the entire hemisphere is edematous, and it begins to seize and then atrophies.

Tr. at 86.

On cross-examination, Dr. Raymond stipulated that "a fever can cause seizures," but he was leery of agreeing "that seizures can produce a stroke." Tr. at 90-91. He explained his discomfort:

That's where you begin to lose me, and as I just said prolonged seizures may result in specific scarring of the brain and what the exact role of each of the individual confounders of prolonged seizure needs to be sort of stated out a little bit more. Secondly, I would disagree that a seizure could cause a stroke.

Tr. at 91. Petitioners pressed him further on this subject, asking whether a complex febrile seizure could, under some hypothetical circumstance, "precipitate a stroke," to which he responded:

I'm not aware of such a circumstance. I think we're splitting hairs here, and I'm not trying to be difficult. I'm just trying to be as accurate as I can be. If someone is seizing, and they're having a prolonged seizure secondary to low blood sugar, for instance, I would state that their subsequent injury is secondary to their low blood sugar, but the heralding of that is the seizure. The second thing is, people who have prolonged seizures then run into if you address their prolonged seizures, then run into a variety of metabolic compromises, including poor oxygenation, raising the CO2 level, dropping of their blood sugar as fuel is burned. And if you don't intervene, those subsequent metabolic derangements can injure the brain.

Tr. at 91-92. He did agree, though, "that seizures can either directly or indirectly cause adverse changes in the brain tissue such as scarring." Tr. at 92.

Dr. Raymond agreed with the restatement of his opinion on specific causation in this case, "that the fever may have been caused by the MMR vaccine, but the stroke was caused independently ... probably by the passage of a venous clot, through a PFO into the arterial system, which then went to the brain and then caused a stroke, and then caused the seizures and the neurological damage."

Tr. at 92. Nonetheless, he stipulated to several aspects of the clinical data that militate against the occurrence of a stroke prior to Brooke's seizure on 18 May 2005:

Q [T]here is no evidence in the medical records of a thrombus anywhere in [Brooke's] brain, am I right?

A That's correct.

Q Okay. In other words, there's no evidence in the records that you can point to which could tell us where this postulated venous clot might have originated, correct?

A That's correct.

...

Q [T]here's no evidence that she had any trauma anywhere which caused a bruise, which produced a clot or anything like that, right?

A Not in the record. That's correct.

Q Okay. No swelling in her hands, no swelling in her feet or legs or arms that would indicate that she had any clots anywhere in her body, right?

A That's correct.

Tr. at 92-93.

Dr. Raymond stipulated that the treating neurologist at the time of the events in question made "a differential diagnosis in which he's diagnosing cerebritis with vasculitis as being the cause of this problem, and less likely a thrombotic or embolic event." Tr. at 95. He stipulated that the coagulation studies returned normal results, and that, to the extent those studies could ascertain, there was "no evidence of any clotting problems with her blood." Tr. at 96. He likewise stipulated that the MRA testing on 20 May 2005 "did not reveal any evidence of a clot either." Tr. at 97.

Most interesting from the Court's perspective was Dr. Raymond's abstention from offering a preponderant opinion on a likely cause for Brooke's stroke, believing it to be idiopathic in nature: "Sitting where I am here today no, I cannot go back and figure out what caused Brooke's stroke." Tr. at 98-99.

Next, Petitioners contrasted Dr. Raymond's reliance on the first MRI's results with the normal CT scan which had been performed two days prior, on the day of Brooke's seizure and emergency admission to the hospital:

Q ...Basically, correct me if I'm wrong, in your report you point out this MRI study indicating focal areas of signal abnormality that you refer to as bright areas on the diffusion sequence and the T-2 weighted sequences. Do these findings indicate a stroke? They do indicate that a stroke has occurred, don't they?

A Yes, they do.

Q All right. And the CT scan on May 18, two days previously when she came into the emergency department was normal, was it not?

A That's correct.

Q If she had an old stroke, that probably would have shown up on a CT scan on the 18th, wouldn't it?

A If she had an old lesion, that would have shown up. That's correct.

Q Okay. But there was nothing shown on the CT scan that would indicate that she had a previous stroke, right?

A There's nothing on her MRI either except for what we're presently seeing that would indicate something that occurred weeks or months before.

Q Now, you say the brightness of these lesions indicate that the stroke would have occurred I think you say at least several days old, am I right?

A Yes.

Q That's what you say in your report there. How long is several days at least, Doctor? Are you talking three or four days, or what?

A 24 to 72 hours.

Tr. at 99-100. Interestingly, the initial seizure occurred approximately 48 hours before that MRI reading was taken.

Lastly, Petitioners questioned Dr. Raymond on the EMT records, and challenged him to square those records with the occurrence of a stroke:

Q ...In the findings section of that EMS report of May 18, under Findings, you referred to the Glasgow Coma Score.... And then you go down below that, and it refers to her eyes. The right eye was reactive. The left eye was reactive, and the right eye size was normal. I assume they're referring to pupil size there, is that right?

A I assume so also.

Q Yes, and the left eye was normal pupil size, correct?

A Okay.

Q Okay. And none of that indicates a stroke, correct?

A That's correct.

Q And there was no facial droop, is that right?

A That's correct.

Q And no arm drift.

A Well, that's what they say. I'm a little leery of believing that they actually were looking. They asked her to hold her hands out in front of her when they're actually saying that she's actively seizing, so when you do pronate or drift, if you ask the individual hold your arms out in front of you, palms up, close your eyes, that's how you do arm drift.

Q Okay. And her speech is listed as being normal, and I understand you think that's a mistake?

A Well, when they particularly comment just three lines above that, Verbal 3, inconsistent, consolable moaning, so they have an inconsistency there.

Q And you think the arm drift is also a mistake then? The arm drift is a mistake?

A Based upon my ability to examine one-year-olds, asking them to do the things that I just stated is how you do arm drift, I'm betting that they didn't do it.

Q Then, the last item they say there under Findings is grip strong-bilaterally.

A That may be so because they go on to say seizures.

Q Yes.

A Under seizure.

Q Seizures and shaking, right?

A Yes.

Q Okay. So at least the only way if her grip strength is strong bilaterally, that is not an indication of a stroke either, is it?

A Well, she seems to be actively seizing here, so I think volitional movement is still a debateable point.

Q Okay. If we assume that the findings stated in the EMS report are correct and accurate, there's no indication in that report that she had a stroke at that point, is that correct? Do you agree with me on that?

A If I ignore the first couple of lines in the EMS report, and then also ignore the fact that she's seizing, and then I accept only the lines that you wish to highlight, then I would agree that there is no indication that she has had a stroke.

Q The indication to you that she might have had a stroke is the fact that she is noted as being seizing and shaking, right?

A That's correct.

Q Okay. But you can have a seizure and be shaking without having had a stroke, isn't that correct?

A That's correct.

Q Okay. So this EMS report would be consistent with the conclusion that she had a set of seizures without a stroke at that point?

A It would also be consistent with my opinion which would be that this is a herald or the onset of her stroke.

Tr. at 101-104.

## II. ULTIMATE FINDINGS OF FACT

Both parties' experts were personally and professionally credible; that premise is beyond a cavil of doubt in the Court's mind. Having heard both experts on numerous occasions over the preceding years, the Court was again impressed by the knowledge of each, and of their command over the subject matter addressed. They both comported themselves as professionals of class and academic distinction. However, the way to address whether an expert witness has proffered a credible, reliable theory, that logically conforms to the specific facts of the case, is to assess the theories of causation and the support for the experts' testimony in medical records and/or literature, not to mask personal preference with expert witness credibility determinations. *Andreu v. Sec'y of HHS*, 569 F. 3d 1367 (Fed. Cir. 2009) ("A special master [cannot] cloak the application of an erroneous legal standard in the guise of a credibility determination, and thereby shield it from appellate review. A trial court makes a credibility determination in order to assess the candor of a fact witness, not to evaluate whether an expert witness' medical theory is supported by the weight of epidemiological evidence."); *see also Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579 (1993) (An expert witness' theory is scientifically valid when it supports the conclusion that "it purports to show."); *Garcia v. Sec'y of HHS*, No. 05-0720V, 2008 WL 5068934 (Fed. Cl. Spec. Mstr. Nov. 12, 2008) ("the question of whether an expert's theory possesses scientific bona fides goes to the persuasiveness of the evidence on the question of aetiology and causation"). "Weighing the persuasiveness of particular evidence often requires a finder of fact to assess the reliability of testimony, including expert testimony, and we have made clear that the special masters have that responsibility in Vaccine Act cases.... [To say that] the special master may not 'cloak the application of an erroneous legal standard in the guise of a credibility determination, and thereby shield it from appellate review' ... is not to say, however, that a special master, as the finder of fact in a Vaccine Act case, is prohibited from making credibility determinations regarding expert testimony." *Moberly v. Sec'y of HHS*, 592 F.3d 1315 (Fed. Cir. 2010) .

Therefore, the Court's task now is to analyze the differences between the opinions offered to determine whether Petitioner has established a logical sequence of cause and effect, having occurred in a medically appropriate time frame, which is biologically plausible to tie together the factual sequence and explain Petitioner's injury. *See Althen v. Sec'y of HHS*, 418 F. 3d 1274, 1278 (Fed. Cir. 2005); *Pafford v. Sec'y of HHS*, 451 F. 3d 1352, 1355 (Fed. Cir. 2006), *rehearing and rehearing en banc denied*, (Oct. 24, 2006), *cert. den.*, 168 L. Ed. 2d 242, 75 U.S.L.W. 3644 (2007); *Walther v. Sec'y of HHS*, 485 F. 3d 1146 (Fed. Cir. 2007); *de Bazan v. Sec'y of HHS*, 539 F. 3d 1347, 1352 (Fed. Cir. 2008).

Based on his reliance on the medical records, the opinion of the treating neurologist, and the medical literature filed, the Court found Dr. Kinsbourne's testimony very helpful, and was persuaded that Brooke suffered a fever as a result of her MMR vaccination that caused her to seize. The absence of symptoms associated with stroke persuaded the Court that onset of the stroke followed her prolonged seizure on 18 May 2005. This finding is consistent with all of the records filed in this case. Even the MRI from 20 May 2005 can be read as consistent with this finding, as Dr. Raymond himself stipulated. Tr. at 99-100. All told, Petitioners presented a *prima facie* case that the MMR vaccine caused the seizures, and eventually *status epilepticus*, suffered by Brooke.

Respondent did not offer sufficient evidence to undermine or overcome Petitioners' proof of the seizure's primacy. Dr. Raymond believed the stroke preceded the seizure, based primarily, if not solely, on the 20 May 2005 MRI, which, he later conceded, could be consistent with a stroke on 18 May. The Court found to be very dubious Dr. Raymond's construction of maternal reports of clumsiness as neurological injury, and was not persuaded thereby. The only patent evidence of stroke arose in the record after the event of the seizure.

A slightly different issue is whether the seizure caused the stroke. If any moderate exertion, even bearing down in passing a stool, could cause an embolism to break free into the bloodstream, certainly the physical strain of a seizure could do the same. However, there is no way to know that absolutely for certain. It is noteworthy that there was no evidence of the stroke before the seizure, but such evidence accumulated shortly thereafter, which at least links the stroke temporally to the seizure. Also, there was no other event mentioned in the medical records or affidavits that would serve as an alternative precipitating event for the stroke. In sum, the Court is persuaded to attribute the onset of the stroke to the stress of the seizure.

Though Dr. Raymond raised the potential influence of the PFO and the (heterozygous) genetic mutation as alternative causes of the stroke, neither rises to overcome the MMR vaccination as a substantial factor. First, the role of either as a risk factor was still somewhat speculative, although the PFO seemed to have more adherents among the medical community, as between the two. But, secondly, even if the PFO created a place for an embolism to form, the vaccine-fever-related seizure most likely served as the precipitating event of the stroke, and only the most deterministic genetic mutations operate completely independently as legal superseding factors. In short, neither of these explanations serves to supplant the MMR vaccine as a substantial factor in causing the fever that triggered the seizure that precipitated the stroke.

In short, the Court finds that Brooke's injury would not have occurred but for vaccination-induced fever that immediately preceded her injuries of initial *status epilepticus* and stroke.

### III. CONCLUSIONS OF LAW

As aforementioned, the Court is authorized to award compensation for claims where the medical records or medical opinion have demonstrated by preponderant evidence that either a cognizable Table Injury occurred within the prescribed period or that an injury was actually caused by the vaccination in question. § 13(a)(1). If Petitioners had claimed that Brooke had suffered a "Table" injury, to them would §13(a)(1)(A) have assigned the burden of proving such by a preponderance of the evidence. In this case, however, Petitioners do not claim a presumption of causation afforded by the Vaccine Injury Table, and thus the Petition may prevail only if it can be demonstrated to a preponderant standard of evidence that the vaccination in question, more likely than not, actually caused the injury alleged. *See* § 11(c)(1)(C)(ii)(I) & (II); *Grant v. Sec'y of HHS*, 956 F. 2d 1144 (Fed. Cir. 1992); *Strother v. Sec'y of HHS*, 21 Cl. Ct. 365, 369-70 (1990), *aff'd*, 950 F. 2d 731 (Fed. Cir. 1991). The Federal Circuit has indicated that, to prevail, every petitioner must:

show a medical theory causally connecting the vaccination and the injury. Causation in fact requires proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury. A reputable medical or scientific explanation must support this logical sequence of cause and effect.

*Grant*, 956 F. 2d at 1148 (citations omitted); *see also Strother*, 21 Cl. Ct. at 370.

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

[Petitioner's] burden is to show by preponderant evidence that the vaccination brought about [the] injury by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury.

*Althen v. Sec'y of HHS*, 418 F. 3d 1274, 1278 (Fed. Cir. 2005).

As part of that analysis, the Federal Circuit recently explained:

[T]he proximate temporal relationship prong requires preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's aetiology, it is medically acceptable to infer causation-in-fact.

*de Bazan v. Sec'y of HHS*, 539 F. 3d 1347, 1352 (Fed. Cir. 2008).

Under this analysis, while a petitioner is not required to propose or prove definitively that a specific biological mechanism can and did cause the injury, he must still proffer a plausible medical theory that causally connects the vaccine with the injury alleged. *See Knudsen v. Sec'y of HHS*, 35 F. 3d 543, 549 (1994).

As a matter of elucidation, the Undersigned takes note of the following two-part test, which has been vindicated and viewed with approval by the Federal Circuit,<sup>9</sup> and which guides the Court's practical approach to analyzing the *Althen* elements:

The Undersigned has often bifurcated the issue of actual causation into the "can it" prong and the "did it" prong: (1) whether there is a scientifically plausible theory which explains that such injury could follow directly from vaccination; and (2) whether that theory's process was at work in the instant case, based on the factual evidentiary record extant.

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<sup>9</sup> *See Pafford v. Sec'y of HHS*, No. 01-0165V, 2004 WL 1717359, 2004 U.S. Claims LEXIS 179, \*16, slip op. at 7 (Fed. Cl. Spec. Mstr. Jul. 16, 2004), *aff'd*, 64 Fed. Cl. 19 (2005), *aff'd* 451 F. 3d 1352, 1356 (2006) ("this court perceives no significant difference between the Special Master's test and that established by this court in *Althen* and *Shyface*"), *rehearing and rehearing en banc denied*, (Oct. 24, 2006), *cert. den.*, 168 L. Ed. 2d 242, 75 U.S.L.W. 3644 (2007).

*Weeks v. Sec’y of HHS*, No. 05-0295V, 2007 WL 1263957, 2007 U.S. Claims LEXIS 127, slip op. at 25, n. 15 (Fed. Cl. Spec. Mstr. Apr. 13, 2007).

Of importance in this case, it is part of Petitioners’ burden in proving actual causation to “prove by preponderant evidence both that [the] vaccinations were a substantial factor in causing the illness, disability, injury or condition and that the harm would not have occurred in the absence of the vaccination.” *Pafford v. Sec’y of HHS*, 451 F. 3d 1352, 1355 (Fed. Cir. 2006), *rehearing and rehearing en banc denied*, (Oct. 24, 2006), *cert. den.*, 168 L. Ed. 2d 242, 75 U.S.L.W. 3644 (2007), citing *Shyface v. Sec’y of HHS*, 165 F. 3d 1344, 1352 (Fed. Cir.1999). This threshold is the litmus test of the cause-in-fact (a.k.a. but-for causation) rule: that petitioner would not have sustained the damages complained of, *but for* the effect of the vaccine. *See generally Shyface, supra*. “[T]he relevant inquiry ...[is]... ‘has the petitioner proven ... that her injury was in fact caused by the ... vaccine, rather than by some other *superseding*[,] *intervening* cause?’ ...[The petitioner need not] rule out every possible explanation ...[but]... must simply show ... that her injury was caused by a vaccine.” *Johnson v. Sec’y of HHS*, 33 Fed. Cl. 712, 721 (1995), *aff’d* 99 F. 3d 1160 (Fed. Cir. 1996) (emphasis added).

“To prove causation, a petitioner in a Vaccine Act case must show that the vaccine was ‘not only a but-for cause of the injury but also a substantial factor in bringing about the injury.’” *Moberly v. Sec’y of HHS*, \_\_ F.3d \_\_, 2010 WL 118661 (Fed. Cir. 2010) quoting *Shyface v. Sec’y of HHS*, 165 F.3d 1344, 1352-53 (Fed. Cir. 1999); *see also Id.* citing *Walther v. Sec’y of HHS*, 485 F.3d 1146, 1151 (Fed. Cir. 2007) (for causation analysis in off-Table cases, the Restatement (Second) of Torts applies and ‘the petitioner is treated as the equivalent of the tort plaintiff’). In the watershed case of *Shyface v. Sec’y of HHS*, 165 F. 3d at 1352, the Federal Circuit “adopt[ed] the Restatement [(2d) of Torts] rule for purposes of determining vaccine injury, that an action is the ‘legal cause’ of harm if that action is a ‘substantial factor’ in bringing about the harm, and that the harm would not have occurred but for the action,” and that rule continues to guide the Court today in the instant matter.<sup>10</sup> *Cf. Hargrove v. Sec’y of HHS*, No. 05-0694V, 2009 WL 1220986 \* 39-40 (Fed. Cl. Spec. Mstr. Apr. 14, 2009).

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<sup>10</sup> The mandate of the Federal Circuit in *Shyface* to follow the RESTATEMENT (2D) OF TORTS on the application of actual causation did not indicate how this Court should approach the tectonic shift of the common law into the later Restatement(s). The short answer to this question is that the Federal Circuit incorporated the RESTATEMENT (2D) OF TORTS, and until the Circuit does otherwise to change that gloss, that is the mandatory precedent binding on this Court. By way of more detailed analysis, given the Circuit’s reasoning in *Shyface* for incorporating the Restatement, *i.e.* that Congress contemplated the common law (in its then contemporaneous understanding) within the Vaccine Act draftsmanship, thus presuming the common law as a background legislative intent, it would appear that only the Second Restatement is binding on this Court in matters touching on actual causation, because that is the version in use at the time of the Act’s drafting and passage. Likewise, when the Federal Circuit decided *Shyface* in 1999, the RESTATEMENT (3D) OF TORTS: PRODUCTS LIABILITY had already become available in published form, and yet the Circuit did not choose to incorporate or even reference that Restatement’s provisions at all, notwithstanding the potential corollary to the Program’s focus on causation in the absence of a fault element. Had it done so, a contrary argument could have been made that the Circuit’s reading of congressional intent was a progressing correspondence to whatever Restatement provisions were most current. However, this would seem to correspond to the more dubious “statutory purpose” canon of interpretation. The Court’s reading of *Shyface* leads to a result that the Third Restatement should be viewed at most as persuasive, but not mandatory authority, and is not to be followed where it conflicts with the Second Restatement.

Here, *Shyface*'s incorporation of the concept of causation in tort law is essential to resolution of this matter. In this case, the central, essential facts are almost all undisputed. What is necessary is to induce what result is called for under the law when viewing these facts in relief, against the immarcescible backdrop of the common law.<sup>11</sup>

Regarding the epistemological quandary raised *supra*, the Court notes the position taken by the common law, one that contemplates the radical alternatives of human choice and unpredictable events. If it is deterministic at all, it is in an absolute sense, wherein the causal factors determining eventual outcomes are overwhelming complex, so as to overwhelm, elude and confound human knowledge. "In a philosophical sense, the consequences of an act go forward to eternity, and the causes of an event go back to the dawn of human events, and beyond."<sup>12</sup> Keeton at 264. This is the basis for what is termed "cause in fact," which is based upon a "but for" formulation. Keeton at 265 ("[T]he classic test for determining cause in fact directs the factfinder to compare what did occur with what would have occurred if hypothetical, contrary-to-fact conditions had existed ... the term "cause in fact" embraces all things which have so far contributed to the result that without them it would not have occurred.") (some internal marks omitted). To rephrase slightly for this context the concise statement of the rule, a vaccine "is a cause of the [injury] if the [injury] would not have occurred but for that [vaccine]; conversely, the [vaccine] is not a cause of the [injury] if the [injury] would have occurred without it." Keeton at 266. Of interest in this case, "The conception of causation in fact extends not only to positive acts and active physical forces, but also to pre-existing passive conditions which have played a material part in bringing about the event." Keeton at 265.

This open-ended universe of infinite chains of cause and effect must be truncated for human comprehension in order to attach legal responsibility, however. Finitude demands an outer limit, a line to be drawn to divide what is a cause attaching liability—a legally proximate cause—from that which averts such responsibility. "As a practical matter, legal responsibility must be limited to those causes which are so closely connected with the result and of such significance that the law is justified in imposing liability." Keeton at 264. This is due to the understanding that, "The event without millions of causes is simply inconceivable; and the mere fact of causation, as distinguished from the nature and degree of the causal connection, can provide no clue of any kind to singling out those which are to be held legally responsible." Keeton at 266. Based on this conceptual understanding, therefore, it is certainly legal error to require the thing, act, or process at issue (here the vaccine) to be identified as *the* "sole cause," *the* "dominant cause," or *the* "proximate cause" of the harm

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<sup>11</sup> For the sake of brevity, rather than discuss myriad common law case opinions, the Court will reference W. Page Keeton, PROSSER & KEETON ON TORTS (5th ed.1984) (hereinafter "Keeton at \_\_\_") and leave the reader to pursue further inquiries to the cases cited therein. Mr. Prosser was a strong and respected force in the crafting of the Second Restatement, and served as Reporter thereto for some time.

<sup>12</sup> Outside of the law, this precept is referred to as "sensitive dependence on initial conditions" or, more colloquially, "the butterfly effect." It was identified by Henri Poincaré in the nineteenth century and elucidated by Edward Lorenz, a pioneer of chaos theory mathematics. However, it is grounded in Newtonian mathematics within the concept of the "evolution rule of the dynamical system" ( $\Phi^t$ ).

complained of.<sup>13</sup> Keeton at 266. The correct formulation for determining proximate cause, then, is to ask if something is *a* substantial factor.<sup>14</sup> Of central importance in this case, if something is *a* substantial factor in causing the injury alleged, causation and liability are not avoided “merely because other causes have contributed to the result, since such causes, innumerable, are always present.” Keeton at 268.

For the Court here to award compensation and hold that the vaccine “actually caused” Brooke’s injury, “it is only necessary that it be *a* legal cause of the harm. It is not necessary that it be *the* cause, using the word ‘the’ as meaning the sole and even the predominant cause.” RESTATEMENT (2D) OF TORTS § 430, Comment d (emphases added); *cf.* RESTATEMENT (3D) OF TORTS: LIABILITY FOR PHYSICAL AND EMOTIONAL HARM § 26, comment c (“tortious conduct need only be *a* factual cause of the other’s harm.”) (emphasis in original). Furthermore, if the sequelae of the vaccination at issue did “actively and continuously operate to bring about” Brooke’s injury, vaccine causation is not precluded even where “the active and substantially simultaneous operation of the effects [of her genetic susceptibility] is also a substantial factor in bringing about the harm.” *Id.* at § 439. The modern trend in common law cases, embedded in the rationale of the Restatement, is to ask why legal cause should be avoided where the complained-of cause coexists alongside other causes. Keeton at 301.

As explained *supra* at the Court’s findings of fact, Brooke’s injuries—seizures, stroke, and neurologic sequelae, which began following an unbroken chain of events, beginning with the MMR vaccination—would not have occurred *but for* her vaccination.

The Restatement indicates that, if the administration of the vaccine(s) to Brooke “creates or increases the foreseeable risk of harm” that preexisted and coexisted in a PFO or a genetic predisposition (as “a force of nature”), and the vaccine is found to be a substantial factor in causing her injury, then neither the PFO nor the genetic predisposition can constitute “a superseding cause.” RESTATEMENT (2D) OF TORTS § 442A and Comment a thereunto. Likewise, “Where the [vaccine] creates or increases the risk of a particular harm and is a substantial factor in causing that harm, the fact that the harm is brought about through the intervention of another force does not relieve

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<sup>13</sup> Prosser and Keeton go on to describe a circumstance where the but-for causation test fails but where liability is imposed even still, based upon the “substantial factor” test:

If two causes concur to bring about an event, and either one of them, operating alone, would have been sufficient to cause the identical result, some other test is needed.... [E.g.,] The defendant sets a fire, which merges with a fire from some other source; the combined fires burn the plaintiff’s property, but either one would have done it alone. In such cases, it is quite clear that each cause has in fact played so important a part in producing the result that responsibility should be imposed upon it...

Keeton at 266-67.

<sup>14</sup> “The ‘substantial factor’ formulation is one concerning legal significance rather than factual quantum.” Keeton at 267. *See also* RESTATEMENT (2D) OF TORTS § 430, Comment a (“The word “substantial” is used to denote the fact that the defendant’s conduct has such an effect in producing the harm as to lead reasonable men to regard it as a cause, using that word in the popular sense, in which there always lurks the idea of responsibility, rather than in the so-called “philosophic sense,” which includes every one of the great number of events without which any happening would not have occurred.”).

[Respondent] of liability, except where the harm is ... not within the scope of the risk created by the [vaccine].” RESTATEMENT (2D) OF TORTS § 442B.<sup>15</sup> Stated in the contrapositive, a causative factor unrelated to the vaccine may only be accounted as superseding (*i.e.*, negating the vaccine’s causative impact) where its operation is “extraordinary” *and* where the resulting harm therefrom is qualitatively distinct from the risk posed by the vaccine. RESTATEMENT (2D) OF TORTS § 451.

Applying the general rule from the common law of torts, compensation is appropriate even when the vaccine “operates upon a concealed physical condition, such as ... a latent disease, or susceptibility to disease, to produce consequences” incapable of reasonable anticipation. Keeton at 291. Additionally, where the vaccine combines with a preexisting condition (such as genetic predisposition), the extent of the ultimate sequela need not have been foreseeable. Keeton at 292. As every aspiring attorney learns, “a defendant takes a plaintiff as he finds him,” a rule most familiarly illustrated in the “eggshell skull” case of *Dulieu v. White & Sons*, 2 K.B. 669, 679 (1901). RESTATEMENT (2D) OF TORTS § 461 (“The negligent actor is subject to liability for harm to another although a physical condition of the other which is neither known nor should be known to the actor makes the injury greater than that which the actor as a reasonable man should have foreseen as a probable result of his conduct.”). *Cf.* RESTATEMENT (3D) OF TORTS: LIABILITY FOR PHYSICAL AND EMOTIONAL HARM § 31 (“When an actor’s tortious conduct causes harm to a person that, because of a preexisting physical or mental condition or other characteristics of the person, is of a greater magnitude or different type than might reasonably be expected, the actor is nevertheless subject to liability for all such harm to the person.”).

On this same general theme, the concept of foreseeability may also enter a discussion of legal cause. If fever or a seizure are possible risks a specially-vulnerable vaccinee might sustain, the common law incorporated into the Restatement would militate for a finding of legal causation. *See* Restatement at § 442B. Additionally, “Foreseeable intervening forces are within the scope of the” risk created by the vaccine, and therefore also of Respondent’s liability; “intervening causes which fall fairly in this category will not supersede” the substantiality of the vaccine as a cause. Keeton at 303-304; *see also Id.* at note 18.

Applying the Federal Circuit’s decision in *Shyface* and, by incorporation, the common law as summated in the Restatement, it seems altogether clear, that an abnormally persistent PFO or a genetic mutation, exerting an independent influence while acting in conjunction with a vaccine

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<sup>15</sup> *See also* Comment b to that section:

If the [vaccine] has created or increased the risk that a particular harm to the [petitioner] will occur, and has been a substantial factor in causing that harm, it is immaterial to the [Respondent’s] liability that the harm is brought about in a manner which no one ... could possibly have been expected to foresee or anticipate. This is true not only where the result is produced by the direct operation of the [vaccine] upon conditions or circumstances existing at the time [*e.g.*, genetic predisposition], but also where it is brought about through the intervention of other forces which the actor could not have expected, whether they be forces of nature, or the actions of animals... This is to say that any harm which is in itself foreseeable, as to which the [vaccine] has created or increased the recognizable risk, is always “proximate,” no matter how it is brought about...

reaction, cannot be accounted a superseding cause overwhelming the substantiality of the vaccine as a cause.

In sum, the Court concludes as a matter of law that, weighing Brooke's MMR vaccination as a causal factor, it was a substantial factor in bringing about her injury, and was not superseded by other preexisting factors, which may or may not have also been substantial factors. Hence, the Court **RULES** that Petitioners are entitled to compensation, to be determined by further proceedings.

#### **IV. CONCLUSION**

Therefore, in light of the foregoing, the Court **RULES** in favor of entitlement in this matter. The parties are instructed to contact the Court for further proceedings, regarding the issue of damages. This matter will be transferred *posthaste*, and the parties are **ordered** to contact the Court to schedule a status conference with the Chambers to which this matter will be reassigned.

**IT IS SO ORDERED.**

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**Richard B. Abell**  
Special Master