OFFICE OF SPECIAL MASTERS

June 28, 2002

BART and MARIE SNEAD, as Parents, Guardians and Next Friends of SARAH ANN SNEAD, a minor,	*	
Petitioner,	*	**PUBLISHED**
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Stephanie J. Hartley, Jacksonville, FL, for petitioners.
Melonie J. McCall, Washington, DC, for respondent.

DECISION

MILLMAN, Special Master

On June 5, 2001, petitioners filed a petition on behalf of their daughter, Sarah Ann Snead (hereinafter, "Sarah"), for compensation under the National Childhood Vaccine Injury Act of 1986[1] (hereinafter the "Vaccine Act" or the "Act"). Petitioners have satisfied the requirements for a prima facie case pursuant to 42 U.S.C. § 300aa-11(c) by showing that: (1) they have not previously

[1] The National Vaccine Injury Compensation Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, 42 U.S.C.A. §300aa-1 et seq. (West 1991), as amended by Title II of the Health Information, Health Promotion, and Vaccine Injury Compensation Amendments of November 26, 1991 (105 Stat. 1102). For convenience, further references will be to the relevant subsection of 42 U.S.C.A. § 300aa.

collected an award or settlement of a civil action for damages arising from the alleged vaccine injury; and (2) DPT vaccine was administered to Sarah in the United States.

Petitioners alleged that Sarah experienced an on-Table residual seizure disorder (RSD) after her first DPT vaccination on June 9, 1998 when she was two months old. RSD is no longer a Table injury.[2] On November 8, 2001, petitioners filed an amended petition, stating that Sarah's first DPT caused in fact her seizure disorder.

The undersigned did not hold a hearing in this case. Section 300aa-12(d)(3)(B)(v) states that a special master "may conduct such hearings as may be reasonable and necessary."[3]

FACTS

Sarah was born on April 6, 1998. Med. recs. at Ex. 1. On April 20, 1998, her pediatrician, Dr. Carl Shealy, noted that she had a very mild club foot. Med. recs. at Ex. 4, p. 1.

On June 6, 1998, at her two-month checkup, Dr. Shealy noted that Sarah's feet were straightening out. She had nasal congestion. He gave her DPT, HiB, OPV, and Hepatitis B vaccinations. Id.

Two months afterward, on August 17, 1998, at Sarah's four-month checkup, Mrs. Snead told Dr. Shealy that she was still very concerned about Sarah's ankles and Sarah had great difficulty wearing her AFOs (ankle-foot orthosis). Dr. Albert T. Gilpin, an orthopedist, thought Sarah might be weak in terms of dorsiflexion and recommended that a neurologist check Sarah. Mrs. and Mrs.

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[2] Effective March 10, 1995, the Vaccine Injury Table includes only anaphylaxis and encephalopathy as Table injuries after DPT. 42 C.F.R. 100.3(a). 60 Fed. Reg. 7694 (Feb. 8, 1995).

[3] Rule 8(d) of the Vaccine Rules of the United States Court of Federal Claims, Appendix B, states: "The special master may decide a case on the basis of written filings without an evidentiary hearing."

Snead were also concerned about Sarah's vision. At first, Sarah seemed to track toys well. On physical examination, Sarah was a heavy child who was interactive and seemed to track well. Her feet were smaller. She was a little waddly in terms of holding up her head. Mr. and Mrs. Snead were concerned about the frenulum (narrow ridge) of her tongue. The frenulum was slightly short, but Dr. Shealy did not believe clipping was necessary. Sarah's feet stretched to 90 degrees easily, but tended to flop with inversion. He noted she had poor tone. Dr. Shealy administered Sarah's second DPT, HiB, Hepatitis B, and polio vaccines. Med. recs. at Ex. 4, pp. 1-2; Exhibit 19, p. 316.

On August 21, 1998, Sarah saw Dr. Lawrence B. Mauldin, a pediatric neurologist. He noted she was born with club feet and had slowness of development, head lag, excessive protrusion of her tongue, a failure to fix well visually even though her eye movements were conjugate, overall hypotonia, particularly of the axial musculature, foot drop (her feet were pushed down and turned in and did not dorsiflex), absent ankle jerks, and possible thinning of the musculature distally. Med. recs. at Ex. 6, p. 1.

On September 17, 1998, Sarah saw an ophthalmologist, Dr. Wilson G. McWilliams who opined in a letter to Dr. Shealy, dated October 2, 1998, that Sarah's lack of visual interest could be secondary to overall developmental delay. Med. recs. at Ex. 15.

Sarah saw a clinical geneticist, Dr. Kate B. Clarkson, on October 7, 1998. Dr. Clarkson noted that she had positional foot deformities, generalized hypotonia, and delayed visual maturation. Her delay in gross motor skills could very well have been the result of hypotonia. Med. recs. at Ex. 12.

On October 14, 1998, for Sarah's six-month checkup, Dr. Shealy diagnosed a myotonic dystrophy or genetic disorder, poor tone with head lag, and right ectropion[4] of the eye, foot drop. He administered Sarah's third DPT, HiB, Hepatitis B, and polio vaccines. Med. recs. at Ex. 4, pp. 3, 6.

On November 12, 1998, Sarah saw Dr. Shealy. She was seven months old. He noted she had episodes most days after feeding where she would stare as if she wanted to vomit, her head and arms would flex forward, and she would crunch her belly forward. She cried for about five minutes especially afterward and usually went to sleep. Five minutes after she cried, she would be alert and sometimes playful. She had a lazy right eye. There was a small nodule on her right mid-thigh, probably where she got her injection. Dr. Shealy diagnosed possible seizure, developmental delay, possible genetic defect syndrome. Med. recs. at Ex. 4, p. 3.

On November 16, 1998, Dr. Mauldin saw Sarah again and noted improvement in muscle tone but poor visual function. He suspected she was cortically blind. Mrs. Snead gave a history that, over the past week or two, Sarah had started having infantile spasm seizures. The family had a videotape that they showed to Dr. Maudlin who noted that it showed typical flexospasms. The family said these began after her last DPT vaccination. Sarah's grandmother noted that Sarah had fever a couple of days later and then a few spasms which went away until the prior week or two, after which, she began having cluster spasms once or twice a day. Med. recs. at Ex. 6, p. 4.

On November 30, 1998, Sarah had an EEG which was abnormal and showed multifocal spike activity. Med. recs. at Ex. 6, p. 5.

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[4] "Ectropion" is rolling outward of the margin, here of the eye. Stedman's Medical Dictionary, 27th ed. (2000), at 566.

On January 11, 1999, Mrs. Snead complained to Dr. Shealy that Sarah had reflux, burping, and belching. She was concerned this might contribute to Sarah's seizure problem. Dr. Mauldin told her that Sarah should not receive any more pertussis vaccinations but he did not feel the vaccine caused her seizures. Med. recs. at Ex. 4, p. 4.

On February 5, 1999, Sarah had an MRI which showed some periventricular leukomalacia[5] which, as Dr. Mauldin wrote in a progress note to Dr, Shealy, would help account for her slow development, seizures, and cortical visual loss. The myelin pattern appeared mildly delayed for her age. There was also a cystic mass in the conus at the lower end of her spinal cord, which probably accounted for her arthrogryposis (persistent contraction or flexure) of the feet. Med. recs. at Ex. 6, p. 9; Ex. 13.

Dr. Mauldin wrote another progress report to Dr. Shealy on March 9, 1999, stating that he went over the fact with the Snead family that the MRI scan of the brain showed periventricular leukomalacia which is a form of damage to the white matter associated with slow development. He thought that a syrinx (tubular cavity) in the lower portion of her spinal cord (T11-12) was a developmental abnormality, and was related to her arthrogryposis in terms of causing damage to her lower motor neurons. Med. recs. at Ex. 6, p. 15.

Sarah saw Dr. Shealy on July 6, 1999. He wrote that she has congenital birth defects and sees various doctors as well as takes speech therapy. Med. recs. at Ex. 4, p. 5.

Sarah had a repeat MRI on October 20, 1999, and, on the same date, Dr. Mauldin wrote a progress note to Dr. Shealy, stating that the MRI showed thinning of the white matter with

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[5] Periventricular leukomalacia is cerebral infarctions symmetrically in the white matter adjacent to the lateral ventricles. Clinical Pediatric Neurology. A Signs and Symptoms Approach, 3d ed., by G.M. Fenichel (1997), at 103.

periventricular leukomalacia and some prominence of the cerebellar folia, indicating cerebellar atrophy. This would account for her poor balance and head control. The Sneads told Dr. Mauldin that they felt strongly that DPT caused Sarah's problems. Med. recs. at Ex. 6, p. 18; Ex. 14.

On November 30, 2000, Dr. Shealy notes that Sarah saw Dr. Richard A. Saunders, an ophthalmologist in Charleston, who mentioned to Mrs. Snead that Sarah might have a progressive genetic illness called Batten disease or a similar neurodegenerative disease. Med. recs. at Ex. 4, p. 9; Exhibit 20..

Mrs. Snead filled out an undated VAERS (Vaccine Adverse Event Reporting System) form for Sarah, omitting the date of onset or description of symptoms, but listing in an attachment a description of symptoms: decline in vision after her first DPT in June 1998; decline in muscle tone after her second DPT in August 1998; and within 36 hours (but the "36" was crossed out and the number "4" written instead) of the DPT in October 1998, she began to have sporadic spasms. Med. recs. at Ex. 7, pp. 1, 2.

Other Submissions

Petitioner provided the affidavit of her pediatrician, Dr. Carl Shealy, dated May 11, 2001. He states that Sarah's second DPT on October 14, 1998[6] was followed within hour by seizures and

[6] This was actually her third DPT.

that the DPT caused her seizures. He does not give a basis for his opinion. Med. recs. at Ex. 5, p. 1.

Petitioner filed an amended affidavit from Dr. Shealy, dated October 17, 2001, stating that DPT caused Sarah's seizure disorder and "that any congenital or genetic disorders she may have had were worsened by the DPT vaccines. This is due to the fact that her neurologic condition and onset of seizures significantly and dramatically changed after the October 14, 1998 set of shots, which included the DPT." P. Ex. 21, p. 1.

Petitioner provided the affidavit of her pediatric neurologist, Dr. Lawrence B. Mauldin, dated May 17, 2001, that Sarah's 2d DPT on October 14, 1998[7] was followed within hours by seizures. He does not state that DPT caused her seizures, merely that her seizures fall within the Vaccine Injury Table definition of RSD. Med. recs. at Ex. 9, p. 1.

Mrs. Snead submitted an affidavit, dated October 1, 2001, stating that, at around two and one-half months of age, she noticed that Sarah's tracking was slower than before and one eye would lag. Sarah's vision over time worsened. Other people would comment on Sarah's soft tone, but Mrs. Snead did not want to think about that. She felt Sarah was no longer reaching and her head was lagging due to her poor vision. She hoped everything would work out. When Sarah reached three months, Sarah would not look at her toys and her interest in things declined. Everyone around Mrs. Snead told her Sarah's muscles were too soft. Sarah's head control got worse. It seemed Sarah started to regress at two and one-half to three months.

Sarah's regression continued through her fourth month. The doctors did not diagnose anything specific. Within hours of her immunizations, she began to crunch and cry in pain a few times

[7] Again, this was her third DPT.

a day. Mrs. Snead thought she had a stomach ache. After thirteen months of age, Sarah's seizures went away. P. Ex. 11.

Petitioner submitted a supplemental affidavit from Mrs. Maria Snead, dated November 20, 2001. After Sarah's third DPT vaccination on October 14, 1998, Mrs. Snead left Sarah with her mother-in-law, Yvonne Snead. She left town the next day (October 15th) for three days and Yvonne Snead took care of Sarah during this period. Maria Snead wrote that Sarah's onset of seizures occurred within 36 hours of her third DPT on her VAERS form because she was not sure when the seizures began. Yvonne Snead told her Sarah had "done something different" the afternoon of the vaccination and she changed the 36 to 4 hours. In the beginning, they did not know that Sarah was having seizures. They were very slight and were just a verbal sound as if you squeezed her. Then they progressed to a jerk and she would cry a little. If you did not watch her, you would miss them. They were sporadic in the beginning and did not occur every day. Usually after eating, she would act as if she were throwing up. Her head would come forward and her arms would flex forward and she would grab her stomach, crunch forward and cry. P. Ex. 24.

Petitioners submitted the affidavit of Sarah's grandmother, Yvonne D. Snead, dated September 30, 2001. She saw Sarah in July 1998 after her first series of vaccination and went on vacation with her and the family. She noticed that Sarah's eyes had changed: she was moving them around more but not fixing on anything. She did not seem to pay much attention to her surroundings. Her head bobbed more than before. Her son, Bart Snead, said that he did not think that Sarah could see. Sarah's muscle tone was mushy. She was not strong.

She saw Sarah again at the end of July 1998, and she and her daughter Brenda expressed their concern about her. Every time they saw her during the summer, Sarah seemed to respond less and

less. She told Maria Snead in early August that Sarah acted as if she had had a stroke. In August 1998, Sarah could not control her head well and did not have much strength in her arms. Her eyes continued to move more than usual. She was unresponsive.

She was with Sarah when she had her third DPT and other vaccinations, leaving two nodules on her legs. Sarah was warm and fretful. Mrs. Snead did not take her temperature but she felt a little warm. She would cry and crunch as if in pain. Mrs. Snead would pick her up and hold her and Sarah would be all right. This occurred about four hours after the vaccinations. The episodes continued through the next three days. She and her son and daughter-in-law thought it was just fretfulness from the vaccinations. P. Ex. 11.

Petitioners filed the affidavit of Sarah's paternal aunt, Brenda A. Bridges, dated October 3, 2001. She saw Sarah at the age of six weeks and she seemed normal. She saw Sarah in July 1998 and she was very limp and not trying to hold herself in any way. She was dead weight and would turn her head, but not move her arms and legs at all or make eye contact. P. Ex. 11.

Mrs. Snead submitted her baby book (Exhibit 17). On page 262, she lists "Caring for Baby" describing dates of and treatments for illness:

4-7-98	mild case club feet	casts on both feet to the knee
8-21-98	Dr. Mauldin - feet - nerves	casts off 5/14/98
9-17-98	vision problems	
10-7-98	genetic doctor - run some tests - all normal	
11-19-98	"crunches"	possible seizures
11-30-98	Dr, Mauldin - myoclonic seizures - on Klonopin	
4-2-99	ear infection	Amoxicillin
8-26-99	bronchitis	Cefzil/Robitussin
1-21-00	sinusitis/conjunctivitis	E-mycin/Amoxicillin
3-6-00	bronchitis	Rocephin, Cefzil
2-2-01	car accident	none

According to her notes on page 264, at the age of six months, on October 14, 1998, Sarah weighted 21 pounds and seven ounces. But on March 11, 1999, at the age of 11 months, she weighed 20 pounds, and she continued to weigh 20 pounds when she was 12 months old (4/29/99) until she was 18 months old (10/8/99), while her height stayed at 32 inches, and her head circumference stayed at 17 ½ inches. She did not weigh 21 pounds until she was two years old (4/6/00). She had gained one-half inch in height and in head circumference by then. On page 270, Mrs. Snead wrote that Sarah first held a bottle on August 1999 (when she would have been 16 months old).

Respondent submitted two reports from Dr. Yuval Shafrir, a pediatric neurologist, dated March 30, 2002 and April 3, 2002, as well as 10 articles to which Dr. Shafrir refers in his reports. R. Exhibits A and D. Sarah was born with bilateral foot deformity. By June 25, 1998, her pediatric orthopedic surgeon did not see a great deal of motor function in either foot. By August 17, 1998, she was noted to have poor tone and questionable visual tracking, and Dr. Shealy referred her to Dr. Mauldin, a pediatric neurologist. Dr. Mauldin saw Sarah on August 21, 1998 and he found several abnormalities: poor visual fixation, hypotonia, foot drop, and thinning of the musculature. Her MRI at ten months showed a delayed myelination pattern, and posterior parietal and occipital periventricular leukomalacia. These are typically signs of genetic, prenatal, and early neonatal encephalopathies.

Dr. Shafrir states that many neurological diseases with onset in infancy will occur after an immunization. Sarah's neurological decline, particularly her visual problems, occurred after her first immunization. It is not rare to have no reason for an infant's severe global developmental delay. In about half of patients with severe global developmental delay studied at a noted hospital, Montreal

Children's Hospital at McGill University, no etiology of the developmental delay was found. Sarah had a clear abnormality, a white matter disorder, with an etiology in the immediate perinatal, or prenatal, periods, typically of hypoxic-ischemic or metabolic-genetic origin. The finding of arthrogryposis is highly suggestive of an early prenatal insult, which may explain the periventricular leukomalacia.

There was no acute encephalopathy in this case. Neither Dr. Shealy nor Dr. Mauldin assert in their affidavits that Sarah's initial neurological deterioration with loss of visual alertness was causally related to her DPT vaccination on June 9, 1998. As for infantile spasms, they are an age specific process of the immature brain as a result of a diverse spectrum of insults. The seizures appear in the vast majority of patients during the first year of life, mostly in the first six months. Therefore, the seizures appear shortly after one of the three DPT vaccinations which are given at ages two, four, and six months. Infantile spasms have never been reported as a manifestation of acute encephalopathy. They are always the result of chronic dysfunction of the central nervous system (CNS), related to genetic, metabolic, and structural-developmental brain lesions, or residual lesions after an acute CNS insult such as hypoxia, infection, or hypoglycemia. Therefore, the appearance of infantile spasms after an insult such as a vaccination does not prove, and actually refutes, any causal relationship. Even when an acute brain insult is the cause of infantile spasms, the spasms do not appear immediately but after a delayed period of several weeks to months. Dr. Shafrir cites his own clinical experience as well as medical literature in support of his opinion.

He also states that the National Childhood Encephalopathy Study (NCES), an extensive study in Britain that examined the relationship between DPT and acute neurologic injury, did not show any causal relationship between DPT and infantile spasms. Other studies reported in the medical literature

failed similarly to find any relationship. The Institute of Medicine (IOM) reported no causal relationship between DPT and infantile spasms.

Dr. Shafrir concludes his first letter by stating that Sarah had a known severe neurological impairment before she began having infantile spasms. She had arthrogryposis, periventricular leukomalacia, and cortical blindness. Periventricular leukomalacia is a well-documented cause of infantile spasms. Arthrogryposis is frequently associated with neuronal migration disorders which also are frequent causes of infantile spasms.

In his second letter, Dr. Shafrir comments on the progressive nature of Sarah's neurological abnormalities in the first year of her life.

Respondent's Exhibit D are the articles and literature to which Dr. Shafrir referred in his first letter. Exhibit 3 is "Etiologic determination of childhood developmental delay," by M.I. Shevell, et al., 23 Brain & Development 228-35 (2001). Out of 80 children with global developmental delay,[8] the authors determined the etiology in 44 cases.[9] Id. at 231.

Exhibit 4 is "West Syndrome: etiological and prognostic aspects," by K. Watanabe, 20 *Brain & Development* 1-8 (1998). The author describes periventricular leukomalacia as a cause of West syndrome, one of whose signs is infantile spasms. Id. at 1.

Exhibit 5 is "Infantile Spasms" by M. Wong, et al., 24 *Ped. Neur.* 89-98 (2001). The authors describe the peak onset of infantile spasms as between four and six months of age. Id. at 90. In 70

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[8] "Global developmental delay was defined as a significant delay in two or more developmental domains (gross/fine motor, cognition, speech/language, personal/social, or activities of daily living)." 23 *Brain & Development* at 235.

[9] These causes were: cerebral dysgenesis (10 cases), hypoxic-ischemic encephalopathy (9 cases), toxin exposure (9 cases), chromosomal abnormalities (6 cases), psychosocial neglect (3), neuromuscular disorder (2), genetic syndromes (2), and other (3). Id. at 231.

to 80 percent of cases, the etiology may be found. Half the cases in which etiology is determined have prenatal causes. In cases in which there is no known cause, infantile spasms "are usually presumed to have an age-related multifactorial genetic predisposition." Id. at 93. The prognosis of infantile spasms is poor. Id. at 94.

Exhibit 6 is "West Syndrome Following Deep Hypothermic Infant Cardiac Surgery," by A.J. du Plessis, et al., 11 *Ped. Neur.* 246-51 (1994). The authors report four infants who developed infantile spasms after cardiac surgery. The onset of the spasms was months after the surgeries. The authors state, "Cerebral dysgeneses, particularly neuronal migration defects, form the neuropathologic substrate for infantile spasms in approximately 60% of patients [citing studies]." Id. at 250.

Exhibit 7 is "The Timing of Brain Insults in Preterm Infants Who Later Developed West Syndrome," by A. Okumura, et al., 32 *Neuroped.* 245-49 (2001). Their study included 11 infants with periventricular leukomalacia who developed West syndrome. Id. at 245.

Exhibit 8 is "Infantile Spasms and Pertussis Immunisation," by M.H. Bellman, et al., *The Lancet* 1031-33 (1983). In reviewing the results of the NCES, the authors state that the onset of infantile spasms occurs around the same time as immunizations, which they term a temporal coincidence. Id. at 1033. They refer to studies in Denmark and Japan which showed the incidence of onset of infantile spasms remaining unchanged when the vaccination schedule changed, suggesting a causal link was unlikely. Id. The same number of children who had onset of infantile spasms after DT as after DPT in the NCES indicates a "non-specific response." Id. They conclude that DPT does not directly cause infantile spasms in children with structurally normal brains but may precipitate spasms in those children who have tuberous sclerosis or Down's syndrome or other illnesses in which spasms are known to occur. Id.

Exhibit 9 is "Infantile spasms and early immunization against whooping cough. Danish survey from 1970 to 1975," by J.C. Melchior, 52 *Arch. Disease in Childhood* 134-37 (1977). Melchior compared the onset of infantile spasms from 1957 to 1967 when DPT immunization was administered at 5, 6, and 15 months of age with the onset of infantile spasms from 1970 to 1975 when pertussis vaccine was given at 5 and 9 weeks of age and at 10 months of age, and found no change in the age of onset. He concluded that any immunization followed by onset of infantile spasms was probably a coincidence. Id. at 136.

Exhibit 10 is missing.

Exhibit 11 is "Periventricular Leukomalacia and West Syndrome," by A. Okumura, et al., 38 *Develop. Med. & Child Neur.* 13-18 (1996). They state that periventricular leukomalacia (PVL) "is a hypoxic ischaemic lesion." Id. at 13. It is not uncommon for PVL infants to develop West syndrome. Id. They suggest that PVL could be a leading cause for West syndrome. Id. at 17.

Exhibit 12 is "Arthrogryposis Multiplex Congenita: Spectrum of Pathologic Changes," by B.Q. Banker, 17 *Hum. Pathol.* 656-72 (1986). Banks states that the overwhelming number of 96 infants and children she studied who had arthrogryposis had a neurogenic form which was usually associated with other congenital abnormalities. Id. at 656. She mentions that myelination of the cerebral white matter is delayed. Id. at 661.

Exhibit 13 is "Epileptic seizures, arthrogryposis, and migrational brain disorders: a syndrome?" by E. Brodtkorb, et al., 90 *Acta Neurol. Scand.* 232-40 (1994). The authors studied arthrogryposis multiplex congenita (AMC) in adults and concluded that AMC, epileptic seizures, and brain migrational disorders may form a previously-undescribed syndrome. Id. at 232. Bilateral clubfeet are included in the category of AMC. Id. Four of the six patients studied had migrational

anomalies in their brains. Id. at 236. They conclude, "Maldevelopments of the brain and the spinal cord may be associated, since the neurons in each of these parts of the CNS go through a similar process of proliferation, migration, and differentiation." Id. at 239.

DISCUSSION

The Vaccine Act affords petitioners two theories of recovery, thereby allowing them to prove causation by showing that either: (1) a Table-injury occurred or (2) the vaccine was the cause-in-fact of the injury. The former theory is governed by Section 14(a) of the Act which contains a Vaccine Injury Table. If the injuries described in this Table occur within the statutorily defined time period, petitioners have proven the existence of a "Table-injury," creating a rebuttable presumption of causation. To rebut this presumption, respondent must provide affirmative evidence demonstrating that a known factor unrelated was the cause-in-fact of the vaccinee's condition. 42 U.S.C. § 300aa-13(a)(1)(B).

Although petitioners initially claimed the injury of RSD as a Table injury, that is no longer on the Table. Their one expert medical report from their pediatric neurologist, Dr. Mauldin, stated that Sarah had RSD. as defined by the Vaccine Table. Since the only Table injuries remaining for DPT are anaphylaxis and encephalopathy, Dr. Mauldin's expert opinion that Sarah had the Table injury of RSD is irrelevant. At numerous telephonic status conferences, the undersigned asked petitioners' counsel to go back to Dr. Mauldin and get a report that would causally relate Sarah's DPT vaccination(s) to her neurologic condition. Counsel reported back to the undersigned that Dr. Mauldin would not give her this report. She did not obtain it from any other neurologist. The only report asserting causation in fact comes from the family practice physician, Dr. Shealy.

Causation in Fact

To satisfy their burden of proving causation in fact, petitioners must offer "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 v. Secretary, HHS, 956 F.2d 1144, 1148 (Fed. Cir. 1992). Agarwsal v. Secretary, HHS, 33 Fed. Cl. 482, 487 (1995); see also Knudsen v. Secretary, HHS, 35 F.3d 543, 548 (Fed. Cir. 1994); Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579 (1993).

Petitioners must not only show that but for the vaccine, the vaccinee would not have had the injury, but also that the vaccine was a substantial factor in bringing about her injury. Shyface v. Secretary, HHS, 165 F.3d 1344 (Fed. Cir. 1999).

Without more, "evidence showing an absence of other causes does not meet petitioners' affirmative duty to show actual or legal causation." Grant, supra, 956 F.2d at 1149. Mere temporal association is not sufficient to prove causation in fact. Hasler v. US, 718 F.2d 202, 205 (6th Cir. 1983), cert. denied, 469 U.S. 817 (1984).

In Dr. Shealy's first affidavit, he states that DPT caused Sarah's RSD without giving any basis. This does not satisfy petitioners' burden to show a logical sequence of cause and effect based upon a reputable medical explanation.

In Dr. Shealy's second affidavit, he states that DPT vaccines significantly worsened Sarah's neuropathy because her seizures significantly and dramatically changed after her October 14, 1998 series of vaccinations. He bases his opinion upon his care and treatment of Sarah, and his experience and education. Dr. Shealy's experience is as a family practice physician. Dr. Shealy ignores Sarah's congenital difficulties, developmental delay, and steadily progressive symptomatology in his opinion.

He relies heavily on the temporal coincidence that, as Sarah's neurological illness progressed in symptomatology, she was also receiving vaccinations. Sarah never had an acute encephalopathy.

Even if Sarah experienced the onset of infantile spasms the afternoon of her third DPT, that does not mean that DPT is the cause of them. As respondent's Dr. Shafrir discussed in his thorough report (letter number 1), DPT does not cause infantile spasms. The medical literature to which he refers, especially the NCES, supports the conclusion that the IOM reached: DPT does not cause infantile spasms. The medical literature filed with Dr. Shafrir's reports discusses the frequency of association of periventricular leukomalacia and arthrogryposis, both of which Sarah has, with the onset of infantile spasms (particularly, West syndrome). Although respondent does not have the burden of proving what is the cause of Sarah's neurological illness, it seems far more likely that Sarah has a congenital problem which manifested itself in her brain abnormality (delayed myelination, periventricular leukomalacia), feet abnormality (clubfeet and arthrogryposis), and subsequent infantile spasms.

The undersigned in numerous opinions has held that DPT does not cause infantile spasms. See Barnes v. Secretary of HHS,1997 WL 620115 (Fed. Cl. Spec. Mstr. Sept. 15, 1997). This decision was affirmed on appeal under the names Hanlon 40 Fed. Cl. 625 (1998) and Plavin (40 Fed. Cl. 609 (1998)). The Federal Circuit affirmed the undersigned's decision as well in Hanlon v. Secretary of HHS, 191 F.3d 1344 (Fed. Cir. 1999), cert. denied, 120 S. Ct. 2212 (2000); Plavin v. Secretary of HHS, 184 F.3d (Fed. Cir. 1999), cert. denied, 120 S. Ct. 2212 (2000). See also, Turner v. Secretary of HHS; Flanagan v. Secretary of HHS, 268 F.3d 1334 (Fed. Cir. 2001) (consolidated appeal). These were all cases dealing with tuberous sclerosis in which the vaccinees did have the benefit of the statutory presumption of causation of seizures. Nevertheless, the undersigned found

after due consideration of all the evidence that their underlying neurological illness was the cause of their conditions.

Dr. Shealy ignores what Dr. Shafrir emphasizes, namely that Sarah had problems from birth which manifested themselves slowly and inexorably over time. The affidavits from her mother, grandmother, and aunt show that her head lag, hypotonia, and failure to make eye contact were progressive developments over time. Maria Snead expressed what every mother must feel: she did not want to think that her daughter was ill, so she pushed the symptoms out of her mind. By the time Sarah received her third DPT, she was already diagnosed as developmentally delayed and probably cortically blind.

Dr. Shafrir, being a pediatric neurologist, is more credible in his opinion than Dr. Shealy, whose experience is in family medicine. Dr. Shafrir opined that Sarah's third DPT had nothing to do with the onset of her infantile spasms, and that she has signs of congenital injury which lend support to the idea that her infantile spasms are part of a neuromigrational defect disorder. Sarah's global delay began before the onset of her infantile spasms and it continued after her spasms ended.

Dr. Mauldin appears to be a responsible and honest pediatric neurologist. Early on in the medical records, he expressed the opinion to Mrs. Snead that the vaccination had nothing to do with Sarah's neurologic condition. It is understandable that parents would want to blame something for their daughter's devastation rather than live with the idea that there is no known cause. But, unfortunately, for almost half of children with devastating neurologic disease, there is no known cause.

Petitioners have not prevailed on a theory of significant aggravation or causation in fact.

CONCLUSION

This case is dismissed with prejudice. In the absence of a motion for review filed pursuant to RCFC Appendix B, the clerk of the court is directed to enter judgment in accordance herewith.

IT IS SO ORDERED.

_____ _____

DATE Laura D. Millman
Special Master