OFFICE OF THE SPECIAL MASTERS
February 23, 1998

RICHARD JORDAN and JULIA JORDAN, Legal Representatives of the Estate of KARA JORDAN,	*	
Petitioners,	*	
vs.	*	No. 91-0113V
	*	PUBLISHED
SECRETARY OF THE DEPARTMENT OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Richard Gage, Cheyenne, WY, for petitioners.

Mark W. Rogers, Washington, DC, for respondent.

DECISION

On January 28, 1991, petitioners, on behalf of their daughter, Kara Jordan (hereinafter "Kara"), filed a petition for compensation under the National Childhood Vaccine Injury Act of 1986[(1)] (hereinafter the "Vaccine Act" or the "Act"). Petitioners alleged that DPT significantly aggravated Kara's preexisting, congenital encephalopathy, subsequently causing her death.

Petitioners have satisfied the prima facie criteria enunciated in 42 U.S.C. § 300aa-11(c) of the Act as they have not previously collected an award or settlement of a civil action for Kara's death, and Kara received her DPT vaccine in the United States.

A hearing was held in this case on August 5, 1997. Testifying for petitioners were Julia A. Jordan, Richard D. Jordan, and Dr. Marcel Kinsbourne. Testifying for respondent were Dr. M. Harold Fogelson and Dr. Leslie J. Raffel.

FACTS

Kara was born on December 16, 1985 at Reid Hospital with congenital abnormalities and seizures. Parents' Affidavit (unpaginated); Med. recs. at pp. 24-25. At birth, she was cyanotic, flaccid, generally lethargic, and had no cry. Med. recs. at pp. 17, 20, 24, & 35. She also had extended fetal tachycardia and hydrocephalus. Id. Her head was large, with a cephalohematoma. Med. recs. at p. 20. She was born with a thick meconium and was suctioned twice before being bagged with one hundred percent oxygen for one hour. Id. She had Apgars of 2 at one minute and 2 at five minutes. Id. She had a pneumothorax in the left pleural space. Med. recs. at p. 15. Shortly after birth, Kara experienced several episodes of eye deviation to the right which lasted for thirty-five to forty seconds. Med. recs. at pp. 21, 27 & 32. As a result, ten milligrams (mg.) of Phenobarbital was administered. Id. She was maintained on 4.4 mg. per kilogram of Phenobarbital which was later decreased to 4 mg. per kilogram every twelve hours. Med. recs. at p. 21.

Three to four hours after birth, Kara was transferred to Riley Hospital, where she remained on a ventilator for twenty-four hours. Id. The discharge summary from Riley notes that her tone was generally decreased. Med. recs. at p. 24. Her head circumference was 32 centimeters (cm). Id. She had poor gag and suck reflexes. Med. recs. at p. 26.

A report from Medical Genetics dated December 16, 1985 diagnosed Kara as having multiple congenital anomalies of unknown etiology. Med. recs. at p. 29. In its discussion section, the report notes that Kara had many features which, "when taken collectively, [are] seen in several of the chromosome deletion syndromes." Med. recs. at p. 30.

On December 23, 1985, Kara was transferred back to Reid Hospital. Med. recs. at p. 21. A history reflects meconium aspiration, respiratory distress with left pneumothorax, seizures, and dysmorphic features. Id. Her chest tube was removed on December 20, 1985. Id. She was on Ampicillin and Gentamycin for three days. Id. An EEG conducted on December 18, 1985 showed excessive amounts of diffuse Delta activity during sleep, with multifocal sharp transients. Med. recs. at pp. 21, 31. This was consistent with a diagnosis of mild to moderate encephalopathy. Id.

A report from the Genetics Department indicated several dysmorphic facial features, including telecanthus, low set ears, short nose, broad nasal ridge, micro-abnormal flexion of the palmar creases, and wide anterior fontanelle and sutures. Med. recs. at p. 21. She was described as having a high pitched cry[(2)] and she had questionable feeding problems with slow weight gain. Med. recs. at pp. 21-22. Her birth weight was 5 pounds, 7 ounces; however, seven days later, she weighed 5 pounds, 6 ounces. Med. recs. at pp. 17, 21. On discharge from Reid Hospital, she had gained only two ounces. Med. recs. at p. 23.

At three weeks of age, Kara saw Dr. E.D. Plasterer, a pediatrician. He noted that she had slow weight gain and was sleeping a lot, possibly due to Phenobarbital. Med. recs. at p. 86. On January 15, 1986, she again visited Dr. Plasterer. Id. A medical record from this visit notes that she was gaining weight adequately. Id. On January 21, 1986, she returned to his office with a cold. Id.

Kara received her first DPT on March 14, 1986, when she was three months of age. Med. recs. at p. 84. She saw Dr. Plasterer on March 20, 1986. Med. recs. at p. 87. A record from this visit reflects that Kara smiled and held her head up. Id.

Kara returned to Dr. Plasterer on April 24, 1986 with an upper respiratory infection. Med. recs. at p. 85. She was admitted to Reid Memorial Hospital on the same date with a complaint of nasal congestion,

cough, and poor feeding for several days. Med. recs. at p. 94. Her head circumference was 38 cm. Id. The impression was pneumonia, dysmorphic features, and very poor weight gain. Med. recs. at p. 95.

On April 29, 1986, Mrs. Jordan phoned Dr. Plasterer to report that Kara experienced increased spitting with formula. Med. recs. at p. 85. This occurred twice on both April 28 & 29, 1986. Id. She was on Amoxicillin. Id. Subsequently, a May 2, 1986 check-up revealed that her bronchitis was resolving. Id.

Kara received her second DPT on May 9, 1986, when she was five months of age. Med. recs. at p. 84. She saw Dr. Plasterer on May 16, 1986 and he noted that she had good weight gain (11 ½ ounces in fifteen days). Med. recs. at p. 88.

On May 27, 1986, Kara went to the Riley Hospital newborn follow-up clinical. Med. recs. at p. 97. She smiled occasionally and was now sucking her thumb and putting her hands together. Id. The Jordans were concerned about her overall developmental progress and poor weight gain. Id. Her weight, length, and head circumference were less than the 5th percentile. Id. Her physical examination was remarkable for open posterior and anterior fontanelles with palpable space between her sutures. Med. recs. at p. 97. She appeared to fix and follow although she had decreased tone and strength. Id. She was able to turn her head when prone and had contact and traction grasps. Id.

She returned to her pediatrician on June 12, 1986 with a stuffy nose. Med. recs. at p. 88. On July 8, 1986, Kara saw Dr. Joyce Fisher, a pediatrician at the Riley Hospital newborn follow-up program. Med. recs. at p. 101. She was due for her third DPT. Id. However, the family discussed omitting the pertussis component of the vaccine due to her seizure disorder and the doctor agreed that this would be a good idea. Id. Developmentally, Kara kicked her legs, smiled, cooed, and was beginning to hold a rattle. Id. The Jordans told Dr. Fischer that Kara had an exaggerated startle response, where she would throw her arms out and become stiff while her eyes blinked. Med. recs. at p. 101. It was unclear to Dr. Fischer if these were seizures or an exaggerated startle response. Id. Her weight, height, and head circumference were still below the 5th percentile. Id. At this time, her head circumference was 40.2 cm. Id.

A July 10, 1986 medical record from Riley Hospital notes that Kara was seizure-free until she received her second DPT.[(3)] Med. recs. at p. 99. After receiving her second DPT, her seizures resumed, increasing in frequency and duration over the past month. Id. An EEG performed the previous week revealed hypsarrhythmia and she was put on ACTH and Phenobarbital. Id. Her head circumference was 40.5 cm. Id. She did not fix or follow and her head control was poor. Med. recs. at p. 99. She seemed lethargic. Id. Developmentally, she was at the newborn level. Id.

Kara returned to Dr. Fischer on July 29, 1986. Med. recs. at p. 102. Her head control had improved and her eyes were beginning to fix better. Id. She may have had a cold. Id. Her head circumference was 40.7 cm. Id. Kara returned to her doctor on August 1, 1986 with an upper respiratory infection. Med. recs. at p. 88. At this time, she had not had seizures for two weeks. Id.

A note dated September 13, 1986 stated that Kara was not to receive any more pertussis vaccine. Med. recs. at p. 103. On September 30, 1986, Kara went back to the newborn follow-up program at Riley Hospital. Med. recs. at p. 104. At this time, Kara focused on faces more frequently and held her head up better. Id. Her head circumference was 41.2 cm. Id. She was obese, fussy, microcephalic, with dusky skin color and an erythematous spotty rash on both cheeks. Id. She had markedly decreased tone throughout with increased deep tendon reflexes. Med. recs. at p. 104. Developmentally, she was at the two-month level. Id. Her parents were instructed to taper the ACTH. Id. An EEG conducted that day showed moderately diffuse slow waves, which indicate a moderately diffuse encephalopathy. Id. There were no focal or epileptiform abnormalities. Med. recs. at p. 104. The rash was expected to disappear as

the ACTH was decreased. Id.

On the night of October 6, 1986, Kara fed poorly. Med. recs. at p. 106. Mrs. Jordan noted that Kara had breathing difficulty, which rapidly progressed to respiratory arrest. Med. recs. at pp. 106, 108. Kara died on October 6, 1986 of cardiorespiratory arrest. Med. recs. at p. 89.

A Reid Memorial Hospital record states that there was never an actual choking episode on the night of Kara's death. Med. recs. at p. 108. When the medics arrived, they noted complete cardiorespiratory arrest. Id. On October 8, 1986, Dr. Richard F. Garnet, Jr., reported to Reid Hospital that Kara had horse shoe kidneys, no uterus, and streaks for ovaries. Id. Dr. Garnet's preliminary autopsy report dated October 7, 1986 stated that Kara had multiple congenital anomalies, including horse shoe kidneys, dilatation of renal pelvis bilaterally, no uterus with small ovaries, facial abnormalities including low set posteriorly rotated ears, primary telecanthus, broad nasal bridge and short nose, borderline micrognathia, bilateral adrenal hyperplasia, and tracheobronchial tree containing thin hemorrhagic fluid. Med. recs. at p. 109.

On microscopic examination, a section of the right lung showed bronchopneumonia and interstitial pneumonitis. Med. recs. at p. 110. Dr. Garnet's final anatomic diagnosis included bronchopneumonia. Med. recs. at p. 111. Kara's death certificate states the cause of death as respiratory arrest due to or as a consequence of either central nervous system anomalies or multiple congenital anomalies. P. Ex. 3.

Dr. Paul Rider, who was the attending physician at Kara's autopsy, submitted an affidavit dated May 9, 1994.[(4)] P. Exs. 2 & 3. In this affidavit, he stated that Kara did not suffer any acute insult which could account for her death. P. Ex. 2, p. 1. While her bronchopneumonia does not fully explain her death, her neurological condition was a significant factor in her death. P. Ex. 2, p. 1, 2.

In a letter dated June 16, 1994, Dr. Rider stated that aspiration was what most likely happened to Kara. R. Ex. B (unpaginated). Furthermore, he felt that her significant congenital neurologic abnormalities contributed to her death. Id.

A cranial autopsy was performed on August 8, 1996 by Dr. Bernardino Ghetti and Dr. Biagio Azzarelli. P. Ex. 4. Kara's brain weighed 615 grams, showed some general foreshortening, and was ball-shaped. P. Ex. 4, p. 1. The Sylvian fissure was somewhat abbreviated and verticalized. Id. Normally, it would reach two-thirds of the total circumference; however, Kara's did not reach more than one-third. Id. The optic nerves were much too small, being no greater than 1 ¼ to 1 ½ mm. in thickness. Id.

Kara's cerebral surface was quite irregular with "small little gyri alternating in bizarre fashion." P. Ex. 4, p. 1. While both frontal lobes were severely involved, the fronto-parietal cortex was less involved. Id. The majority of the frontal lobe width was 5 mm.; however, it was 3mm. at some points. Id. The temporal lobe width ranged from 10 to 12 mm. Id. The pattern was anomalous. Id.

On microscopic section, Dr. Ghetti found developmental abnormalities of neuronal migration, which were particularly evident at the cerebellum. P. Ex. 4, p. 2. He and his colleague found islands of neurons in the cerebellar white matter. Id. An overall reduction of Purkinje cells[(5)] was clearly apparent. Id. The neocortex showed diffuse hypoxic changes. Id. The findings were consistent with an abnormality of neuronal migration which had occurred during Kara's embryonic development. P. Ex. 4, p. 2.

On microscopic examination, Dr. Azzarelli found similarly. The cerebral cortex showed extensive vacuolization[(6)] and few eosinophilic[(7)] neurons, which is suggestive of anoxic-ischemic[(8)] changes. P. Ex. 4, p. 3.

TESTIMONY

Mrs. Julia A. Jordan, Kara's mother, testified first for petitioners. She stated that Kara was very upset immediately following her two DPT vaccinations. Tr. at 8. She cried after each vaccination but Mrs. Jordan did not remember how long it took to calm her. Id.

On the evening of her second DPT, Kara did not scream or cry, but she did have a low grade fever. Tr. at 9. She was lying on her mother when Mrs. Jordan felt Kara experience a little startle or jump.[(9)] Id. This movement occurred only once that evening. Tr. at 10. It did not seem important to Mrs. Jordan at the time. Id. Kara did not stop breathing or cry in pain. Tr. at 10-11.

Kara continued to have these startle reactions "every now and then."[(10)] Tr. at 11. Gradually, these reactions became longer and more intense, exhibiting themselves in a series that could last up to one or two minutes. Id. Mrs. Jordan did not go to the doctor or tell him about these episodes. Tr. at 12. By August 11, 1986, Kara had been seizure-free for two weeks.[(11)] Tr. at 16-17.

On October 6, 1986, Mrs. Jordan, her mother, and her sister were with Kara. Tr. at 17-18. She tried to feed Kara at 6:00 or 6:30 p.m., but Kara refused to eat. Tr. at 18. Kara's breathing became labored. Tr. at 19. Mrs. Jordan called the doctor, but could not reach him. Id. Kara stopped breathing before the emergency medical technicians (EMTs) arrived. Id. Mrs. Jordan did not see Kara have a seizure or choke on anything. Id. Mr. Jordan was not home at the time. Tr. at 20. He arrived as the EMTs were trying to resuscitate Kara. Id.

On cross-examination, Mrs. Jordan admitted that she did not mention Kara's startling episodes when she visited the doctor on May 27, 1986. Tr. at 21. She does not recall how far apart the startles were or if they occurred more frequently by July 10, 1986.[(12)] Id.

Mrs. Jordan testified that Kara's overall development was poor. Tr. at 23. She never sat without support or crawled. Id. She never had symmetric movements of her hands and legs. Id. She could smile, but lost the ability to do so after her second DPT. Tr. at 23. Although Mrs. Jordan did not know the exact date when Kara lost this ability, the loss was permanent.[(13)] Tr. at 23-24. Mrs. Jordan did not recall any startles occurring after August 1986. Tr. at 24. She also did not remember her daughter having better head control or being able to fix her eyes after the second DPT.[(14)] Id.

Richard Jordan testified next for petitioners. During the time of Kara's second DPT and the months immediately following, he was working as an associate insurance agent. Tr. at 33. He kept long hours, with evening appointments two to three nights a week. Id. He disliked Kara's pediatrician, Dr. Plasterer, because he felt that the doctor did not properly record what Kara was experiencing. Tr. at 35.

Mr. Jordan first noticed Kara have startle reactions the weekend after her second DPT. Tr. at 36. He noticed a twitch. Id. As time went on, the twitch became a series. Id. This change occurred over less than one week. Id. He believed that Kara would hold her breath during these episodes. Id. Shortly after July 4, 1986, she had an EEG. Tr. at 37-38. Mr. Jordan was trained to administer ACTH, which controlled Kara's spasms. Tr. at 39. After July 1986, Mr. Jordan never witnessed Kara have another spasm. Tr. at 40.

As Kara's health stabilized throughout the summer, she tried to follow voices or footsteps. Tr. at 41. Although Mr. Jordan thought that Kara had lost all vision by the end of September, he believed that she could follow sound or vibration. Id. Mr. Jordan does not remember Kara sucking her thumb or putting

her hands together.[(15)] Tr. at 43. He also does not recall her kicking her leg, smiling, cooing, or holding a rattle.[(16)] Tr. at 44.

Dr. Marcel Kinsbourne, a pediatric neurologist, testified next for petitioners. He last treated children seventeen years ago. Tr. at 89. His opinion is that Kara had on-Table seizures. Tr. at 52-53. He bases this opinion on Mrs. Jordan's testimony and the Riley Hospital record which notes that Kara was seizure-free until receiving her second DPT. Id.

He diagnosed her exaggerated startles as infantile spasms, testifying that Kara had been on Phenobarbital for six months prior to her second DPT because she had ischemic, anoxic encephalopathy at birth with subtle seizures. Tr. at 53-54. Her multiple anomalies have affected her nervous system since birth. Tr. at 54. He further noted that her second EEG showed hypsarrhythmia. Tr. at 56.

Dr. Kinsbourne testified that infantile spasms are the most serious seizure disorder. Tr. at 57. The condition is associated with a falling off in mental development which leaves the individual mentally retarded. Id. The causal mechanism behind these seizures is not properly understood. Id. Even if the seizures are controlled, the child is retarded. Tr. at 57-58. In Kara's case, her infantile spasms remained untreated for more than two months until she received ACTH. Tr. at 58.

According to Dr. Kinsbourne, Kara's infantile spasms were due to a combination of her congenital problems and her DPT; however, DPT was the "trigger." Tr. at 71. Infantile spasms occur in the brain stem, basal ganglia, and the cortex. Tr. at 68. The interaction of her preexisting brain problem and the DPT caused the onset of her infantile spasms. Tr. at 73. It would be speculative to say that either her congenital abnormalities or her DPT was the sole cause of her death.[(17)]

Tr. at 87.

Dr. Kinsbourne considers Kara's breathing difficulties to be a factor in her death. Tr. at 58. She stopped breathing because her brain stem (medulla) stopped functioning; however, he was unsure why Kara's brain stem ceased functioning. Tr. at 58-59. There was nothing in Kara's history which would signal respiratory problems. Tr. at 59. However, on autopsy, serious neuromigrational problems were revealed, which included problems with the lower cranial nerve. Tr. at 59-60. This nerve is related to breathing. Id. Dr. Kinsbourne noted that Kara was at risk for respiratory distress due to the location of her congenital abnormalities; yet, these abnormalities do not fully explain her death. Tr. at 60. Kara's brother Matthew has the same malformations as Kara and he has not died. Tr. at 63. Thus, the syndrome with which Kara was born does not inevitably result in death. Tr. at 88.

Dr. Kinsbourne further stated that Kara could have died without infantile spasms, but he does not know how. Tr. at 65. The presence or absence of seizure does not correlate with her mental development. Tr. at 65-66. Relying on the medical records alone, Kara seemed to be getting better prior to her death. Tr. at 67.

Dr. Kinsbourne testified that there was more than one negative influence on Kara's brain, making her more vulnerable to respiratory distress. Tr. at 76. He noted that this other influence could have been occurring in Kara even though she did not have seizures and gained modest milestones. Tr. at 76-77. A week prior to Kara's death, her ACTH was tapered and this could have led to her collapse.[(18)] Tr. at 79-80. Her weight loss could also be attributed to the tapering of her ACTH.[(19)] Tr. at 82-83. Dr. Kinsbourne stated that Kara's death was not the result of an acute process, but a chronic one.[(20)] Tr. at 87.

Dr. Kinsbourne admitted on cross-examination that Kara could have died at ten months of age without having had infantile spasms. Tr. at 92. He opined that infantile spasms took a toll on Kara's brain simply because that is the effect that they have on the brain. Id. Although there is no literature to support this thesis since it is still an area of controversy, Dr. Kinsbourne believes that the brain continues to suffer regardless of whether the spasms are controlled. Tr. at 93. While a small percentage of individuals with infantile spasms develops normally, the majority slows down and loses ground, becoming severely retarded. Id.

With the exception of the Jordans' testimony, Dr. Kinsbourne admitted that there is no other evidence to support the conclusion that Kara's infantile spasms damaged her brain. Tr. at 94. However, infantile spasms are strongly associated with neuronal migrational disorders. Tr. at 98. Dr. Kinsbourne further admitted that neither a majority of the medical community nor the literature supports a finding that DPT causes infantile spasms. Id. Dr. Kinsbourne testified that his opinion would be strengthened if Kara's seizures had not resolved and her skills had not improved. Tr. at 131. He further stated that there is no evidence in the record as to the cause of Kara's death. Tr. at 132.

Dr. Kinsbourne admitted that there is no evidence that Kara's respiratory problems worsened after her DPT. Tr. at 114. She had acute lack of oxygen during her delivery and her first EEG showed ischemic, hypoxic encephalopathy due to anoxia. Tr. at 122-23. In contrast, her brother, Matthew, did not suffer from birth asphyxia. Tr. at 123. Although Kara's birth asphyxia resolved itself quickly, Dr. Kinsbourne cannot exclude it as a cause for what happened to her later in life. Tr. at 124-26.

When asked about Kara's head circumference, Dr. Kinsbourne noted that at ten-and-one half months, it was very much below the 5th percentile whereas her prior head circumference measurements were "modestly" below the 5th percentile. Tr. at 133. This change evidences that Kara was getting worse. Tr. at 137.

Dr. M. Harold Fogelson, a pediatric neurologist, testified first for respondent. He sees seizure patients on a daily basis and he has published articles on brain malformations. Tr. at 146-47. He performs three or four EEGs per patient per year and reads approximately 2,500 EEGs annually. Tr. at 147.

Dr. Fogelson testified as to Kara's pre-DPT condition by noting that her birth was complicated with multiple, late decelerations and variable decelerations. Tr. at 148. She had pulmonary problems from meconium. Id. She had no cry, and was cyanotic, with pneumothorax. Id. She had significant acidosis from low blood gases. Id. She also had seizures and apnea as well as many respiratory problems. Tr. at 148. Her EEG reflected multifocal sharp transients, which are indicative of seizures. Id. As a result, she was put on Phenobarbital. Id.

Dr. Fogelson further noted that Kara had unusual defects and developed slowly. Tr. at 149. She had frequent pulmonary infections. Id. After her first DPT, nothing adverse happened. Id. However, she experienced startles after her second DPT. Id. There was no marked deviation in her development and her head circumference did not fall off. Tr. at 149. At four months of age, her head circumference was 38 cm. while at five months of age it was 39.1 cm. Tr. at 150. Her head circumference increased to 40.2 cm. at six months and it was 40.7 cm. at seven months. Id. At nine and one-half months, it was 41.2 cm. Id. This constitutes a fairly smooth curve without marked deviations. Id.

On July 3, 1986, Kara had a routine EEG which showed hypsarrhythmia. Tr. at 151. She was given twenty units of ACTH daily and her startles stopped. Id. She did not lose or change her abilities. Id. A September 30, 1986 EEG did not show focal or epileptiform discharges although they had been seen at birth. Id.

Kara had cardiopulmonary arrest on October 6, 1986. Tr. at 151. On autopsy, her trachea had lost mucosa. Id. Her right lung showed bronchopneumonia and pneumonitis. Id. Kara's neuronal defects explain both her seizures and her multifocal EEG. Tr. at 152.

Dr. Fogelson opined that Kara's death was due to a combination of her brain malformations (neuronal migrational), hypoxic, ischemic encephalopathy, pulmonary problems and bronchopneumonia. Tr. at 153. He did not believe that DPT caused or triggered Kara's infantile spasms, testifying that her migrational brain defect was the more likely cause. Tr. at 154. Kara's infantile spasms were related to her cortical, medulla, and brain stem abnormalities.(21) Tr. at 153, 157-58. Dr. Fogelson further noted that the onset of her infantile spasms was coincidental to the DPT, with infantile spasms playing no role in her death. Tr. at 154-56. He did not know how neuronal migrational disorder and hypoxic, ischemic encephalopathy cause infantile spasms. Tr. at 180. However, one study reflects that 95 percent of patients with infantile spasms have neuromigrational defects (R. Ex. J).(22) Tr. at 181.

Dr. Fogelson stated that infantile spasms are neither associated with a degenerative condition nor cause damage themselves. Tr. at 156, 158. Rather, they manifest an underlying disorder, such as neuronal migrational disorder and hypoxic, ischemic encephalopathy as seen in this case. Tr. at 156-57. They are an overt manifestation of an EEG abnormality. Tr. at 169-70. Kara's EEG from September 30, 1986 did not show infantile spasms or multifocal, epileptiform discharges.(23) Tr. at 170.

On cross-examination, Dr. Fogelson admitted that Kara was neither on an apnea monitor upon release from her birth hospital nor did she have apnea anytime thereafter. Tr. at 160-61. Although the most significant, identifiable factor which accounted for Kara's death was her neurological condition, Dr. Fogelson stated that she would not have died at the time she did if she did not also have bronchopneumonia and interstitial pneumonitis. Tr. at 164-65. There was no autopsy evidence of aspiration; however, aspiration does not always display itself on autopsy. Tr. at 167.

With respect to Kara's head circumference, Dr. Fogelson stated that even if she had lived to eighteen months, it would have been below the 5th percentile. Tr. at 171-72. There was not a precipitous decrease in the velocity of her head circumference. Tr. at 173-74.

Dr. Leslie J. Raffel, a geneticist, testified next for respondent. She is board-certified in pediatrics and clinical genetics. Tr. at 191. She has seen over 1,000 patients. Tr. at 192. Dr. Raffel testified that the cause of Kara's underlying abnormality was an alteration in a single gene. Id. She had an autosomal recessive genetic disorder which caused multiple, congenital abnormalities.(24) Id.

Dr. Raffel stated that it is likely that underlying brain abnormalities made Kara a candidate for perinatal asphyxia. Id. Dr. Raffel does not believe that Kara's disorder was idiopathic. Id. She did not know the gene which caused Kara's disorder; however, there are hundreds of genetic disorders whose causative genes are unknown. Id. There are more than 50,000 genes, and 5,000 genetic disorders. Tr. at 194. Doctors know the causative genes in only one-quarter of these disorders. Id.

Dr. Raffel testified that Kara's neuromigrational disorder as well as her underlying brain abnormalities caused her infantile spasms. Tr. at 195. Hypoxia may have also played a role. Tr. at 196. Thus, Kara's death was due to a combination of her underlying genetic abnormality, a predilection for respiratory difficulties, and acute pneumonia. Id. Infantile spasms neither altered her neurological functioning nor contributed to her death. Tr. at 196-97. She continued to achieve some developmental milestones after the spasms began. Tr. at 197. The spasms responded very rapidly to ACTH and Kara did not exhibit clinical signs of infantile spasms at the time of her death. Id.

Dr. Raffel noted that Kara's head circumference was getting worse over time, but there was not a sharp drop-off.[(25)] Tr. at 197-98. A low-grade fever could have lowered Kara's seizure threshold after her second DPT, thus, causing her seizure. Tr. at 201-02. However, Kara's autopsy shows neuromigrational disorder, which is linked to infantile spasms.[(26)] Tr. at 207. While Dr. Raffel testified that the onset of her infantile spasms was most likely coincidental to her vaccination, DPT could have been an influence. Tr. at 210. Yet, Dr. Raffel also testified that it was characteristic for infantile spasms to occur at the time when Kara experienced them. Id.

DISCUSSION

Significant Aggravation

To prevail with a claim of significant aggravation under the principles enunciated by the Federal Circuit in Whitecotton v. Secretary, HHS, 81 F.3d 1099 (Fed. Cir. 1996), petitioners must show that: (1) the vaccinee's current condition is worse than her pre-vaccination condition and, (2) the onset of that significant worsening occurred within Table time. Petitioners' burden, however, is slightly more expansive than the above framework as they must also show that the vaccine injury led to the current condition. Song v. Secretary, HHS, 31 Fed. Cl. 61 (Fed. Cl.), aff'd, 41 F.3d 1520 (Fed. Cir. 1994).

Accepting that Kara's seizure disorder resumed within Table time of her second DPT, it would seem that petitioners have made a prima facie case for significant aggravation of her preexisting encephalopathy. However, the court is unable to reach this conclusion because Kara's death seems totally unrelated to her resumed seizure disorder. Since the vaccine injury appears unrelated to the child's ultimate condition, petitioners have failed to make out a prima facie case of significant aggravation.

The record clearly evidences that Kara experienced seizures at birth. When given the anti-convulsant, Phenobarbital, she was seizure-free until her second DPT when her seizures resumed. She was given ACTH and remained seizure-free until her death. Although her head circumference was below the 5th percentile, she continued to gain motor skills. Her head circumference increased; however, she remained microcephalic.

The medical records further support that Kara experienced numerous episodes of respiratory distress after her birth asphyxia. On January 21, 1986, Kara had her first cold. Med. recs. at p. 86. She had an upper respiratory infection on April 24, 1986 with nasal congestion, cough, and poor feeding. Med. recs. at p. 94. As a result, she was hospitalized and diagnosed as having pneumonia. Id. A May 2, 1986 medical record notes that her bronchitis was resolving. Med. recs. at p. 85. On June 12, 1986, she had a stuffy nose for two days, and may have had a cold on July 29, 1986. Med. recs. at p. 88, 102. Finally, she had an upper respiratory infection on August 1, 1986. Med. recs. at p. 88. Thus, the medical records reflect that Kara had a total of six colds or upper respiratory infections over an eight-month period.

What the medical records do not substantiate, however, is the Jordans' testimony that Kara's post-DPT state was a consistent decline. Well-established case law holds that information in contemporary medical records is more believable than that produced years later at trial. United States v. United States Gypsum Co., 333 U.S. 364, 396 (1948); Burns v. Secretary, HHS, 3 F.3d 415 (Fed. Cir. 1993); Ware v. Secretary, HHS, 28 Fed. Cl. 716, 719 (1993); Estate of Arrowood v. Secretary, HHS, 28 Fed. Cl. 453 (1993); Murphy v. Secretary, 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); Montgomery Coca-Cola Bottling Co. v. United States, 615 F.2d 1318, 1328 (1980). Contemporaneous medical records are considered trustworthy because they contain information necessary to make diagnoses and determine appropriate treatment. Cucuras v. Secretary, HHS, 993 F.2d 1525, 1528 (Fed. Cir. 1993):

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.

Id.

While Mr. and Mrs. Jordan depict a relentless decline in Kara's abilities and development after the second DPT, the medical records belie this description, reflecting that Kara was improving after her vaccination. She could smile, hold a rattle, and hold up her head. Med. recs. at pp. 99, 101. Developmentally, she went from a neonate level in July 1986 to that of a two-month old in September 1986. Med. recs. at pp. 99, 104.

Besides failing to prove a continual decline in development until her death, petitioners fail to prove that Kara seized at the time of her death. Mrs. Jordan was present when Kara died, noticing only that her daughter had fed poorly and had difficulty breathing. Kara had a history of these symptoms. Surely, if Kara had seized at death, Mrs. Jordan would have seen it. On autopsy, Kara was diagnosed as having bronchopneumonia. Her death certificate states the cause of death as respiratory arrest due to her central nervous system anomalies and her multiple congenital anomalies. There is no relationship between the initial aggravation of her seizures and her death.

Finally, the court accepts Dr. Fogelson's theory that Kara's death was caused by a combination of her preexisting genetic encephalopathy, birth asphyxia, and bronchopneumonia.(27) His testimony was buttressed by Dr. Raffel. Drs. Fogelson and Raffel are also more convincing than Dr. Kinsbourne due to their current practices. While Dr. Kinsbourne last treated children seventeen years ago, Drs. Fogelson and Raffel have active practices in pediatric neurology and genetics, respectively.

Petitioners' expert, Dr. Kinsbourne, could not offer any credible explanation of the cause of Kara's death. Particularly damaging to petitioners' case was the fact that Dr. Kinsbourne essentially admitted the weakness of his opinion on causation by stating that his opinion would be stronger if Kara had continued to seize. Kara did not seize. Rather, her heart stopped. Finally, while Dr. Kinsbourne stated that he did not know why Kara's brain stem stopped functioning, he admitted that Kara had neuromigrational disorder and birth asphyxia. The court had the definite impression that Dr. Kinsbourne was searching for some explanation for Kara's death that would satisfy petitioners' burden without quite believing it himself.

Dr. Rider, who treated Kara (and still treats Matthew) and was at the hospital when she died, believed the cause of Kara's death to be chronic conditions with which she was born.

It is indeed tragic to have two children born with a genetic disorder which leads to either mental retardation or death. However, the court cannot find DPT to be the cause of such tragic consequences in the light of the more convincing evidence set forth by respondent. Upon review of the record in its entirety, the court finds that Kara's death was caused by a combination of her neuromigrational disorder, birth asphyxia, and bronchopneumonia. Accordingly, petitioners have failed to satisfy their burden of proving significant aggravation.

CONCLUSION

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

IT IS SO ORDERED.

DATED: _____ _____

Laura D. Millman

Special Master

1. The statutory provisions governing the Vaccine Act are found in 42 U.S.C.A. § 300aa-10 et seq. (West 1994). The National Vaccine Injury Compensation Program comprises Part 2 of the Vaccine Act. For convenience, further reference will be to the relevant subsection of 42 U.S.C. § 300aa.

2. A Riley Hospital record dated January 16, 1986 described her as having a "cat cry" when being extubated. Med. recs. at p. 35.

3. During this seizure-free period, she was on anti-convulsants.

4. Dr. Rider is a colleague of Dr. Plasterer and the treating pediatrician for Kara's brother, Matthew Jordan. R. Ex. B. On behalf of Matthew, petitioners similarly filed a petition for compensation under the Act; however, this claim was dismissed by the court. See Jordan v. Secretary, HHS, No. 91-1344V, 1992 WL 300901 (Fed. Cl. Spec. Mstr. Oct. 2, 1992), aff'd, 1993 WL 849554 (Fed. Cl. 1993), appeal dismissed (unpub. Fed. Cir. 1994).

5. Purkinje's cells are large branching neurons in the middle layer of the cortex cerebelli. Dorland's Illustrated Medical Dictionary, 297 (27th ed. 1988).

6. A vacuole is a small space or cavity formed in the cell protoplasm. Id. at 1801.

7. Eosin is a plasma stain. Id. at 563.

8. Anoxia is a total lack of oxygen. Id. at 94. Ischemia is deficiency of blood in a part due to constriction or obstruction. Id. at 857.

9. Mrs. Jordan admitted that she did not actually see the movement. Tr. at 9.

10. Since Mrs. Jordan was working at a construction company during this time, Kara was cared for by a babysitter during the day. Tr. at 8, 11. Although Mrs. Jordan testified to seeing these startle reactions "every now and then," she had no personal knowledge whether they occurred while she was at work. Tr. at 11.

11. Mrs. Jordan testified that Kara had been on Phenobarbital since she was a newborn because a nurse believed that the child had seized immediately after birth. Tr. at 13.

12. According to the Riley Hospital records from July 10, 1986, Kara's seizures had increased in frequency over the prior month. Tr. at 22.

13. This testimony conflicts with notations in the medical records for May 27, 1986 and July 8, 1986.

Med. recs. at pp. 97, 101.

14. The medical records conflict with this testimony. A medical record dated May 27, 1986 notes that Kara appears to fix and follow. Med. recs. at p. 97. A July 29, 1986 record states that Kara was "holding her head up better and her eyes are beginning to fix better." Med. recs. at p. 102. Finally, a medical record for September 30, 1986 notes that she focuses on faces more frequently and has better head control. Med. recs. at p. 104.

15. This statement conflicts with the medical record dated May 27, 1986. Med. recs. at p. 97.

16. This testimony conflicts with the medical record for July 8, 1986. Med. recs. at p. 101.

17. Dr. Kinsbourne further testified that Kara's congenital abnormalities were massively involved in her death. Tr. at 109. However, neither the congenital abnormalities nor the infantile spasms alone can fully explain her death. Tr. at 111-12.

18. ACTH stimulates the adrenal cortex to produce increased cortical steroids. Tr. at 80. ACTH must be tapered gradually so it does not cause damage to its recipient. Tr. at 80-81. Because it is unclear how gradually Kara was tapered off ACTH, Dr. Kinsbourne thought it reasonable to assume that such may have caused her collapse. Tr. at 81.

19. Increased appetite and weight gain are associated with an increase in cortical steroids. Tr. at 83. Thus, when ACTH is tapered, the cortical steroids decrease and weight loss can occur. Id.

20. With respect to this point, Dr. Kinsbourne noted that nothing occurred at the moment of Kara's death which essentially caused her death. Tr. at 87. Rather, the events which resulted in her death occurred much earlier in her life. Id.

21. Dr. Fogelson testified that Kara's infantile spasms were caused by abnormalities in her cellular migration. Tr. at 153. This was evidenced by her facial abnormalities. Tr. at 154. Literature supports the theory that cortical migrational abnormalities are responsible for infantile spasms. Id.

22. Chugani, Harry T., & Conti, Jon R., "Etiologic Classification of Infantile Spasms in 140 Cases: Role of Positron Emission Tomography," J. Child Neurology (1):44-48 (Jan. 1996).

23. The results of this EEG were better than that of her first EEG which was conducted when she was a newborn. Tr. at 170.

24. While Matthew Jordan has the same disease as his sister, the children's parents are normal. Tr. at 192. Although the Jordan children have a pattern of identical, striking abnormalities, there is no literature on their disorder. Tr. at 193, 206-07.

25. This course was similar to that of her brother. Tr. at 198.

26. In addition, Kara's neuromigrational defect played a major role in her respiratory distress. Tr. at 205.

27. Respondent does not have the burden of proving the cause of Kara's death. That burden remains with petitioners, i.e., that the Table injury caused in fact the death. Hodges v. Secretary, HHS, 9 F.3d 958 (Fed. Cir. 1993); Hellebrand v. Secretary, HHS, 999 F.2d 1565 (Fed. Cir. 1993).