In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 98-0438V

(Filed: March 26, 2001)

CARMEN RICE, mother and legal guardian to OWEN BURMAN, a minor,	*	
Petitioner,	*	**TO BE PUBLISHED**
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Mark Silverman, Birmingham, MI, for Petitioner.

R. Lynne Harris, United States Department of Justice, Washington, DC, for Respondent.

DECISION

French, Special Master.

This matter arises under 42 U.S.C. §300aa-2 *et seq.*, the National Childhood Vaccine Injury Compensation Act. [1] On May 12, 1998, Petitioner filed her claim in this court alleging that as a result of the Diphtheria-Pertussis-Tetanus (hereinafter "DPT") vaccinations administered on June 27 and August 3, 1995, her infant son, Owen Burman, sustained an encephalopathy.

Respondent defends by arguing that Petitioner has not provided sufficient evidence to prove actual causation nor has she supported an on-Table claim. Respondent argues

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[1] The National Vaccine Injury Compensation Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755 (codified as amended at 42 U.S.C.A. §§300aa-1 through -34 (West 1991 & Supp. 1998)). References shall be to the relevant subsection of 42 U.S.C.A. §300aa.

further that there is a preponderance of the evidence that Owen's post-vaccination medical problems were due to a factor unrelated to the vaccine. Respondent contends that Owen exhibited signs of neurological dysfunction prior to the aforementioned DPT vaccinations and his neurological deficits are the result of Periventricular Leukomalacia (hereinafter "PVL").

A hearing was held on March 15, 2000 in Washington, DC. Petitioner presented the testimony of Mrs. Carmen Rice, Owen's biological mother, the expert testimony of Dr. John Jacoby, pediatrician, and the expert testimony of Dr. William M. Hammesfahr, neurologist.[2] Respondent presented the expert testimony of Dr. John MacDonald, pediatric neurologist. Mrs. Rice's testimony was presented with candor and consistency. The court found her to be a highly credible witness.

FINDINGS OF FACT

The facts of this case are complicated and require the undersigned to include considerable detail. The following are the facts as supported by a preponderance of the evidence. Owen was born on April 29, 1995, the product of a 32-week preterm delivery. He weighed three pounds and seven ounces. His estimated date of delivery had been July 7, 1995. [3] Owen's Apgar scores, however, were encouraging, eight and nine at one and five minutes, respectively. Petitioner's Exhibit B, at p. 53. [4] After delivery, Owen required intubation and ventilation and was admitted to the Newborn/Neonatal Intensive Care Unit (hereinafter "NICU"). P. Ex. B, at p. 114. While hospitalized, he exhibited the usual complications of prematurity, but he fed well, and did extremely well for a premature infant. He was on mechanical ventilation for only three days with an additional seven days of supplemental oxygen and treatment with Survanta. P. Ex. B, at p. 63. Mrs. Rice testified that the doctors and nurses told her how extraordinarily he was doing for his prematurity. He was discharged much earlier than expected. His symptoms steadily resolved spontaneously. He remained in the NICU until May 30, 1995. Id. Owen was discharged home with an apnea monitor at Mrs. Rice's request, on his 31st day of life, May 30, 1995.[5]

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[2] On May 22, 2000, Respondent filed a post-hearing Response to the Oral Testimony of Dr. William M. Hammesfahr. The court permitted Respondent to address the testimony of Dr. Hammesfahr because the substance of his testimony was not disclosed until the date of hearing. The undersigned reviewed Respondent's Response and will address it later in the decision.

[3] At birth Owen was 30 to 31 weeks by gestational age and 32 weeks by newborn examination (including neuro-muscular maturity rating).

[4] Reference to Petitioner's filings will hereinafter be "P. Ex. __, at p. __."

[5] Mrs. Rice testified that Owen was not going to be discharged home with a monitor but it was her request that Owen go home on an apnea monitor. Transcript of March 15, 2000 hearing (hereinafter Tr.) at 24. In addition, Mrs. Rice testified that one indication of Owen's excellent course after birth was the fact that he was discharged home earlier than expected. "You know, they told me not to expect him to go home until his due date, which would have been July 7th, but he actually came home on May 31st, so that's an indication of how well he did." Tr. at 23.

There were no additional complications.

Mrs. Rice described Owen as a calm gentle infant, who cried only when he was hungry or required a diaper change. However, he had some feeding problems. During next five months of life, Owen saw a series of four different pediatricians for that condition. Owen's first pediatrician, Dr. George Blum, suggested an evaluation for the presence of gastroesophageal reflux. P. Ex. D, at pp. 12-13. He referred Owen to a pediatric cardiologist, Dr. Elliott Weinhouse, who found no evidence of cardiovascular or pulmonary abnormality. He recommended a barium esophageal swallow to rule out the possibility of reflux. Id. Mrs. Rice made four changes of formula, but the child continued to have gassiness at times. He appeared to be uncomfortable while eating. After yet another formula change, Owen received his first DPT on June 27, 1995. P. Ex. D, at p. 400. Mrs. Rice testified that beginning on the second day after the administration of the DPT, Owen acquired an altogether different problem.

According to Mrs. Rice, Owen "was doing a lot of crying" and "a lot of sleeping" on the day of the vaccination. Tr. at 30-31. She attributed Owen's sleepiness to the fact that she had premedicated him with Tylenol and continued to administer Tylenol after the vaccine. Approximately two days after the vaccine, in the early morning hours of June 30th, Owen experienced a dramatic episode of eye rolling and projectile vomiting. A description of the event follows:

> . . . I was feeding him and at one point he stopped sucking the bottle and his eyes was [sic] kind of rolled back in his head, and I thought that he fell asleep while he was eating. And so I said, "Owen, Owen," you know, 'cause his eyes were kind of fluttering, and his eyes had gone back and it scared me very much.
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> * * *
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> Then finally, you know, he like his eyes came back and he looked at me, and then I started feeding him again, and then he started that projectile throwing up.

Tr. at 31-32.

After this episode, Owen was put to bed. Upon awakening the next morning, at about 10:00 a.m., Owen experienced a second episode of projectile vomiting. Tr. at 33. Mrs. Rice telephoned Dr. Blum's office and was told to bring the child into the office. [6] The notes from this visit record the eye rolling and episodes of projectile vomiting, the events that brought Owen to the doctor's office. The Doctor also recorded Mrs. Rice's concerns -- "still fussy, gassy and not eating well." "turgor OK." P. Ex. D, at p. 400.

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[6] Mrs. Rice testified that she questioned the doctor's office about the relationship between Owen's condition and the vaccinations "They said 'no'. They said bring him in." Tr. at 33-34. She was told that Owen's condition was not caused by the vaccination. Tr. at 39.

The next record of Owen's medical conditions was recorded approximately one week later, on August 3, 1995, when he was brought again to see Dr. Blum. Mrs. Rice provided a description of what occurred during the time period between June 30 and August 3, 1995. She described further changes in Owen's eating habits after the vaccination, but more dramatically, Mrs. Rice described the onset of relentless screaming. "It was like blood-curdling screams." Tr. at 35-36. Dr. Blum's notes state that the child was "very fussy and crying" for 24 hours. P. Ex. D, at p. 401. Dr. Blum noted also that Owen could not smile spontaneously. According to Mrs. Rice's testimony, she was told "since he's not feeling well already, we might as well give him his next set of shots so you don't have to come back in a few weeks." The doctor administered a second set of vaccinations. Mrs. Rice testified that after this vaccination, "the screaming just got worse." Tr. at 40. Understandably, this was the last time that Owen received medical care from Dr. Blum. Id. Thereafter, Mrs. Rice would go from doctor to doctor seeking help.

From August 17, 1995 to October 3, 1995, Owen received medical care from Dr. Nahed Zakaria. At the first office visit, Dr. Zakaria noted that Mrs. Rice's chief complaint was that Owen "won't stop crying." P. Ex. E, at p. 418. Dr. Zakaria's impression at this visit was that Owen was a "normal preemie with colic/constipation." Id. One month later, on September 19, 1995, Dr. Zakaria noted that Owen was "not eating well, crying a lot, sounds congested when eating." P. Ex. E, at p. 419. Dr. Zakaria noted increased muscle tone which was more prominent in the upper extremities. Id. His impression: "Feeding intolerance/colic." Id. A trial of Lactofree formula was suggested. Two days later, on September 21, 1995, Owen again presented with complaints of, "Doesn't want to eat. Screams all the time." P. Ex. E, at p. 420. Dr. Zakaria ordered an upper gastrointestinal study which was performed on October 6, 1995.

On September 27, 1995, Owen was taken to see yet another pediatrician, his third. The medical records for this visit to Dr. Lalit Shah, noted that "his screaming had got worse," particularly while trying to eat. P. Ex. G, at p. 445. Her review of the upper GI study, revealed a normal anatomy with mild to moderate esophageal reflux. P. Ex. G, at p. 429. She prescribed "Reglan" which provided some temporary relief. On October 9, Dr. Shah noted that Owen was "doing great." (emphasis in original). P. Ex. G, at p. 437. That condition, apparently, was transitory. Dr. Gabora notes on the 23rd of October that the relief didn't last. By October 25, when Owen returned to Dr. Shah she noted the following problems: "Does not roll over, does not sit, does not focus." P. Ex. G, at p. 441. Inexplicably, in spite of all these concerns which should have alerted the doctor to a possible neurological condition, Owen was given his third set of immunizations at this visit. P. Ex. G, at p. 442. In Mrs. Rice's words, ". . . [T]hen he received his third DPT on the 25th and everything went haywire." Tr. at 59.

Dr. Shah referred Owen to a pediatric gastroenterologist, Dr. Souheil Gebara, for evaluation. Dr. Gebara reported his findings to Dr. Shah. In a letter dated October 26, 1995, Dr. Gebara noted that Owen was developmental delayed. Dr. Gebara recorded his impression that Owen's poor feeding may be diagnosed as possible odynophagia which could be secondary to esophagitis although, he might have an underlying neurologic

disease.[7] P. Ex. G, at p. 439. This was the first time that any doctor considered the possibility that Owen's condition had a neurological etiology. Dr. Gebara changed Owen's medications and referred him for a complete neurologic evaluation.

On October 27, 1995, just two days after his third set of immunizations, Owen was brought again to the attention of Dr. Gebara because of increased irritability. P. Ex. G, at p. 443. Mrs. Rice reported that Owen had been "screaming for many hours." Dr. Gebara's solution, seemed to help but only briefly. Id. A few days later, on November 6, 1995, Dr. Gebara again saw Owen with complaints of irritability; Mrs. Rice reported that Owen "has been crying for many hours every day since Saturday." P. Ex. G, at p. 446.

Owen was then brought to Dr. John Manica, a pediatric neurologist, who performed a neurologic evaluation on November 7, 1995. Dr. Manica, observed that Owen was very stiff with posturing of his extremities, "has upper motor neuron dysfunction, mild to moderate spastic tetraparesis[8] with posturing, brisk reflexes and upgoing toes. At times he has irritability." P. Ex. G, at pp. 448-49. Concluding, Dr. Manica stated that "Owen probably fits into the broad category of youngsters who may have sustained an undefinable injury during pregnancy, who have developmental immaturity, or who have undersophistication of brain at the ultrastructural level (microscopic cerebral dysgenesis)." Id. Dr. Manica ordered an MRI and a therapy program. Id. Two days later, on November 9, 1995, Owen was seen by Dr. Fidelina Baraceros, a pediatrician. It was noted that Owen does not reach or roll over, that Owen was growing but "not as expected," and his development was described as "slow." P. Ex. H, at p. 451. On November 13, Owen again started vomiting and screaming. He was hospitalized again. Owen returned to Dr. Manica's office on December 1, 1995 at which time Dr. Manica reviewed the MRI with the parents. It was discovered that Owen's MRI showed periventricular leukomalacia (hereinafter PVL). P. Ex. G, at p. 432. PVL will be discussed hereafter.

Owen was taken to Dr. Jane Perrin, a physiatrist, for an evaluation. Dr. Perrin's impression was "cerebral palsy, spastic quadriparesis ? Etiology.?" [sic] Dr. Perrin suggested that Owen's cerebral palsy "may well be related to the prematurity." She was uncertain whether a milestone had been lost versus early rolling from extensor thrust. [Extensor thrust is a movement associated with cerebral palsy,] but she did not see any extensor thrust on that day. Whether Owen lost the ability to roll over as opposed to a recognizable spastic movement, also called "spastic flip" will be addressed hereafter. P. Ex. H, at p. 450.

On December 18, 1995, Owen was examined by Dr. Alexa Canady, pediatric neurosurgeon. Dr. Canady described Owen as happy with a good social smile but with little motoric activity. P. Ex. H, at p. 466. Further, "He does not reach for objects. He did

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[7] Odynophagia is defined as pain on deglutition; deglutition is defined as the act of swallowing. Dorland's Illustrated Medical Dictionary, 440; 1168 (27th ed. 1988).

[8] Tetraparesis is defined as muscular weakness affecting all four extremities. Id. at 1702.

not roll over. He is unable to maintain a sitting position. He has increased tone in his upper extremities and to a lesser extent in his lower extremities. . . . His cranial nerve examination appears to be normal." Id. Dr. Canady agreed with Dr. Manica's impression that Owen's MRI revealed changes consistent with PVL. Id. Concluding, Dr. Canady remarked that Owen has cerebral palsy "most likely related to his prematurity." Id. Finally, "we do not expect this child to be entirely normal although we do expect him to make developmental gains." P. Ex. H, at p. 467.

Thereafter, on December 26, 1995, Owen was evaluated at the gastroenterology clinic at Children's Hospital of Michigan (hereinafter "CHOM"). Drs. Lin and Madani examined Owen and suggested changes in Owen's medication and formula and ordered a gastric scintiscan[9] and pH probe. P. Ex. J, at pp. 523-45. On February 13, 1996, Owen was seen for follow-up. Owens' test results revealed significant esophageal reflux and noted that Owen continues to experience irritability and arching with feedings. P. Ex. J, at p. 590. Drs. Lin and Madani recorded the following: "Impressions remain that Owen's gastroesophageal reflux and feeding difficulties are more likely rooted in neurodevelopmental issues associated with prematurity and prolonged NICU stay, rather than food allergies per se." P. Ex. J, at p. 591.

Dr. Michael Nigro, pediatric neurologist at CHOM, evaluated Owen on February 26, 1996. [10] After a thorough review of Owen's history, Dr. Nigro concluded that Owen "exhibits significant evidence of upper motor neuron impairment with spasticity, hyperreflexia and clasp-knife." P. Ex. J, at p. 572. Further, Dr. Nigro stated that, "[t]he period of regression sounds significant and raises the possibility of an acquired or late manifesting progressive neurological disorder. . . . The possibility of unrecognized delayed development without true regression is also considered although less likely in view of the negative cerebral ultrasounds as a neonatal, the lack of any history of significant acquired acute neurological involvement even as a newborn and what is reported as regression and the persisting hyperacoustic response." Id. Dr. Nigro ordered an EEG and an MRI in order to compare Owen's previous MRI of 1995. [11] On a subsequent visit to Dr. Nigro on October 8, 1996, after a review of Owen's neonatal ultrasound, he opined that Owen suffered from "chronic encephalopathy with spastic cerebral palsy". The etiology remained unclear. P. Ex. J, at pp. 568-69.

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[9] Scintiscan is defined as a two-dimensional representation of the gamma rays emitted by a radioisotope, revealing its varying concentration in a specific tissue of the body, such as the brain, kidney or thyroid gland. Dorland's Illustrated Medical Dictionary, 1494 (27th ed. 1988).

[10] In Dr. Nigro's lengthy history of Owen, he noted that, "At three months of age, he [Owen] was rolling over both ways, bringing his head towards the chest and seemed to have better head control." P. Ex. J, at p. 571. Dr. Nigro continued that Owen, "subsequently developed less interaction seemed 'weak and out of it'. He was not focusing well, seemed to be staring more." Id.

[11] The MRI ordered by Dr. Nigro was interpreted as "white matter loss with abnormal signal involving white matter of the cerebral hemispheres. Corpus callosum is myelinated but markedly thinned." The EEG was mildly abnormal. P. Ex. J, at pp. 621, 626.

ADDITIONAL FACTUAL ISSUE

At the hearing, a debate arose among the experts concerning Owen's ability and subsequent loss of the ability, to roll over. According to Mrs. Rice, Owen began to roll over on July 18, 1995. Tr. at 47. Dr. Blum's office notes for August 3, 1995 document that Owen could roll over both ways. The infant was three months old at the time. Petitioner argues that Owen lost the ability to roll over after the administration of the DPT vaccine. Loss of this developmental skill, Petitioner alleges, would suggest that a neurological injury had occurred, due to an adverse reaction to the DPT vaccine.

Respondent contends that Owen probably did not lose roll-over capability, because, more likely than not, the child was merely demonstrating a pathological reflex consistent with a well-known movement related to cerebral palsy. Respondent's expert argues that at the age of three months, it would be unlikely that an infant would have achieved the ability to roll over, and that what his mother observed was probably a neurological condition called "spastic flip", a pathological reflex, (asymmetric tonic neck reflex, or ATMR) that one might mistake for a voluntary rollover. The issue, though not dispositive, is relevant.

Dr. MacDonald, for Respondent, explains that there can be abnormal postures, especially a baby with cerebral palsy, where they don't really flip themselves over. They develop a fairly good rolling response but it's not spontaneous. It's part of their cerebral palsy. This, he observes, was described by the physiatrist, Dr. Perrin, as ATMR. They can arch and flip themselves over, and many parents, and some doctors, might interpret this as being a normal pattern when in fact it's not.

The possibility of pathological reflex (spastic flip) must be taken into consideration in this case. However, in deciding which position is more likely, the court would be guilty of speculating whether Owen lost rollover ability or whether the rollover was initiated by early unidentified cerebral palsy. Certain factors support each position, and both are possible explanations. Dr. MacDonald was not present to observe the baby; Dr. Blum saw him regularly. Dr. Blum, however, might have missed the possibility that the child's rollover resulted from spasticity rather than a bona fide developmental milestone. Moreover, it is not clear whether Dr. Blum saw Owen roll over or whether he simply recorded Mrs. Rice's opinion. The court notes that the physiatrist/therapist, Dr. Perrin, to whom Dr. MacDonald refers and who described this abnormal flip, was observing Owen long after the alleged rollovers. Mrs. Rice testified that when Owen came home from the hospital, and at the three-month visit when Dr. Blum documented the rollovers (both ways), Owen showed no signs of cerebral palsy, stiffness or spasticity. Mrs. Rice had experience with other babies and insists that he rolled over "like a regular baby rolls."

Upon questioning, Dr. MacDonald acknowledged that Owen did not exhibit spasticity when he came home from the hospital and that spasticity develops slowly over time. In fact, the occupational therapy records to which Dr. MacDonald refers, establish that it was almost two years after the DPT vaccination that the therapist recorded her notes and diagnosed spasticity of all four extremities. She stated in her records that she did not see

the spastic flip on the occasion when she saw him. At the present time, Owen turns over by using these abnormal reflexes because he has now developed spasticity of all four extremities. Tr. at p. 48, 239. The court declines to guess which position is correct. Inasmuch as other evidence of lost milestones exists, the court need not find one way or the other, nor rely on this issue, to determine this case.

EXPERT TESTIMONY

Dr. John MacDonald, M.D., for Respondent :

Dr. MacDonald has spent 23 years as a child neurologist covering the children's hospitals and other hospitals in Minneapolis, Minnesota where he is usually the consultant providing neurological service and working with the pediatricians and others. He is a graduate of the University of Michigan, spent two years in Norfolk doing general pediatrics for the U.S. Navy, and trained in pediatric neurology for three years at the University of Miami.

Dr. MacDonald is of the opinion that Owen's present condition is fully explained by two factors both of which are unrelated to the vaccinations. Those factors are, first, the undeniable presence of periventricular leukomalacia (PVL), a lesion in the brain, and second, the child's premature birth which he considers the likely cause of the PVL. Dr. MacDonald testified that although medical science has seen tremendous improvement in extending the survivability of premature infants, prematurity still involves risk to various organ symptoms, most notably, to the brain. The most demonstrable damage is the incidence of cerebral palsy. Tr. at 179. Prematurity can account for brain injury, he states, "and that's what we are talking about today." Tr. at 180. PVL does not cause prematurity, but is a frequent result of prematurity.

PVL is observed as an abnormal lesion deep within the developing brain, next to the ventricular system, in which the white matter or tissue of the brain (the leuko) becomes morbidly softened due to failure of blood flow, preventing the brain from completing its development in that area. Medical science has determined that this process takes place in the early third trimester of pregnancy, between about 26-32-34 weeks. Brain damage can occur at that time. This does not occur in full term infants. Tr. at 181-82. Moreover, the damage may not be detected until much later. In other words, PVL is the residue or result of developmental failure. The fact that PVL was not observed on the sonogram taken at birth does not rule out the possibility of its presence. Tr. at 187. Dr. MacDonald testified that in 70 percent of the time PVL will not be detected on the initial ultrasound or sonogram. As an example, he states, cerebral palsy is usually not diagnosed until much later. One would not necessarily see abnormalities in the nursery. Tr. at 192.

Dr. MacDonald agrees with Drs. Mannaka, Cannady, and Perron, three of Owen's doctors, who were of the opinion that Owen's problems were consistent with pre-maturity-related issues. Dr. MacDonald testified that it is not surprising that it was not until his first MRI scan that the PVL lesion was observed. He testified further that the treating doctors

diagnosed cerebral palsy, probably because it is the most expected evidence of PVL.

For these reasons, Dr. MacDonald argues, statistically, if one compares the relative risk of a vaccine-related encephalopathy and the relative risk of a PVL etiology of Owen's injuries, it would be far more likely that his injuries could be ascribed to PVL. According to Dr. MacDonald, four percent of all premature babies are going to be at risk for PVL, whereas the risk of damage caused by the vaccine is considered to be 1 out of 300,000. Dr. MacDonald admits that it is certainly possible for a child who is premature and who has PVL might also have an adverse reaction to a vaccine. In other words, when asked if that child could have another problem put on top of the PVL, Dr. MacDonald answered, "of course." "All I can say is it looks like PVL, it smells like PVL and it is PVL, and it's common." Tr. at 207. Dr. MacDonald would not diagnose Owen's injuries to be vaccine related without more dramatic evidence of encephalopathy. To summarize, Dr. MacDonald's opinion is based on the following:

> You have a lesion on scan that we know is related to prematurity. We have a baby that was born premature. We have cerebral palsy and . . . it's at least a four percent chance [at 32 weeks prematurity], which is enormous risk of developing CP. That seems to me a very clear cut case to make a diagnosis. Tr. at 196.

Dr. John Eric Jacoby, MD., M.P.H., for Petitioner:

Dr. John Jacoby is board certified by the National Board of Medical Examiners, and by the American Board of Pediatrics. He has served as Instructor in Pediatrics and Psychiatry at Harvard Medical School and serves at various hospitals, most predominantly at Mt. Sinai Hospital, in New York City. After reviewing the records of Owen Burman, Dr. Jacoby concluded that the child sustained an acquired encephalopathy caused directly by, and in reaction to, his DPT vaccination. It is his opinion, to a reasonable degree of medical certainty, that without the administration of the DPT, Owen would be a completely normal child. P. Ex. W, at p. 1280.

Dr. Jacoby believes the evidence favors strongly Petitioner's claim. First, Dr. Jacoby counters that prematurity is not, in and of itself, evidence of future problems. In this case, the child progressed rapidly to overcome the effects of prematurity. He received the appropriate, typical, and customary treatment for his level of prematurity. He received mechanical ventilation for two or three days only and his condition resolved appropriately. He demonstrated normal APGAR scores of 8 and 9, he was discharged earlier than expected, and he was felt to be quite healthy and normal at the time of discharge. An early ultrasound demonstrated a normal brain, the child gained appropriate weight, the symptoms of prematurity resolved and had no apparent impact on the child, and the child's clinical course was smooth both at birth and during his initial hospitalization. His feeding intolerance is a common occurrence and does not cause brain damage. Moreover, although his feeding problems did not abate, the excruciating screaming which seemed to exacerbate his early problems attributed to GER, did not develop until after his vaccination.

Dr. Jacoby argues further, that he progressed appropriately, achieved milestones, and no credible evidence of neurological deficits existed until after the administration of his first, second, and third vaccinations.

Dr. Jacoby explains further that the pertussis vaccine, particularly the specific type of vaccine administered in this case, was known to cause reactions in some individuals, and on some rare occasions, has been known to cause permanent brain damage as he believes it did in Owen's case. Tr. at 71. He notes that the DPT vaccine administered to Owen has now been replaced by DPaT, a safer vaccine. Tr. at 74-75. In his opinion, Owen should not have been given the second and third DPT shots inasmuch as he was apparently supersensitive to the first. Dr. Jacoby called attention to the fact that medical evidence exists demonstrating that permanent damage is more likely when repeated shots are given in cases where reactions are observed at the first administration. The individual is at even greater risk of a stronger reaction if given again. In other words, according to Dr. Jacoby, Owen was susceptible to an even worse reaction when he received his third DPT shot. That risk, he states, "has been well known in the literature for several years." Tr. at 70-72. Mrs. Rice confirmed at hearing that "when Owen received his third DPT . . . "everything went haywire." Tr. at 59. Owen had an extensive work-up and no alternative cause of his encephalopathy was found.

In response to Dr. MacDonald's testimony relating to the statistical risk of PVL, Dr. Jacoby argues that at a gestational age of 32 weeks, Owen's risk of damage would not be high, but low, "uncommon, less than one in 20" based on the article filed by Respondent, "PVL Risk Factors Revisited," which provides statistics on the incidence of PVL according to gestational age. Tr. at 77, 81. In other words, according to Petitioner's expert, "he doesn't fit the risks" of permanent PVL- related damage to the brain. Tr. at 82-83.

Dr. Jacoby believes that one cannot diagnose a PVL cause without relying on mere supposition. His theory of damage is supported by these factors: Evidence of a neurologically well child until after the vaccine was administered; and no credible evidence supports any other event or episode to account for his condition. On the other hand, he finds strong evidence of an injury, an encephalopathic event, within Table time of the vaccination. Tr. at 83-84.[12]

Dr. Jacoby argues that the doctors were concentrating on treatment of Owen's gastroesophageal reflux and missed the underlying problem. He dismisses the GER as irrelevant. He states as follows:

I think that all the doctors were looking for a cause and missing the cause of

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[12] Petitioner's raise the issue that extra axial fluid was observed in the MRI of the brain. Petitioner's expert argued that its presence would rule out a PVL etiology related to prematurity. The testimony presented on this issue was highly equivocal, and failed to convince the court that the presence of extra axial fluid had any significant effect one way or the other. Based on the lack of convincing testimony to the contrary, the court concludes that the presence of extra axial fluid had no significant impact in determining causation in this case. Tr. 86-92.

> the pertussis. I think why? Maybe they discounted the mother's story and were just looking for stuff that's popular. . . . Babies are always having a little bit of spitting up, and in Owen's case it just went too far . . . and the reflux is really a red herring, not an injury, not an illness. This is a brain case. This [reflux] is a mild thing. . . . If this baby would have grown up normal and not had the [vaccine] reactions, you wouldn't be here today and we would have said yes, Owen had a lot of colic when he was a baby. The feeding and reflux problems are separate problems. But there is no question that from some source, he became [also] a brain damaged baby. Tr. at 95-96 .[13]

Dr. Jacoby cites the following as evidence of the onset of an encephalopathic condition following his DPT shot: Eye rolling was a standard type of seizure episode and was temporally related to the shot. Tr. at 98. Seizures, although frequently benign, can also be a sign of brain dysfunction. Owen's subsequent clinical course is evidence that the seizures were not benign. Projectile vomiting is a sign of illness and goes with brain irritation. Projectile vomiting is different from normal spitting up and would be a cause for the doctor to be called. It is consistent with the pertussis reaction. It would not be caused by Gastroesophageal reflux but is a symptom suggestive of an acute neurological problem. Tr. at 100-01. The child lost milestones already achieved.

Dr. Jacoby offers the following additional evidence of encephalopathic signs observed after vaccination: Owen lost the ability to smile; he no longer rolled over; his eyes began to cross after vaccination; he developed increased muscle tone, more prominent in the upper extremities; he changed from a quiet happy baby to a child with continuous excessive screaming that lasted for days and weeks; and he had difficulties swallowing. Owen had an ophthalmological examination prior to the vaccination, and the examination proved to be normal. On a second examination, however, performed after the DPT vaccination, the ophthalmologist observed evidence of damage to the optical nerve which evolved into optic nerve atrophy, clear evidence that some adverse event in the interim had had a negative impact on the brain affecting the child's neurological condition. The optic nerve is part of the brain. Dr. Jacoby does not believe it was due to PVL but was due to an acquired source. Owen's case, he argues, is one of very severe brain damage for a child. In his opinion it would have been noted at birth were he to have had some preexisting condition before the shots. It could not have been missed . Tr. at 106-07.

Dr. Jacoby does not question the existence of PVL. His argument is that attribution of cause to the PVL is simply hypothetical and cannot be proved. On cross examination, Dr. Jacoby described one research finding that gives him additional reasons to believe that the pertussis should be considered the preferred attributing cause. Dr. Jacoby cites evidence that when kittens were given injections of endotoxins [endotoxin is the suspected antigen in the pertussis vaccine] the kittens developed periventricular leukomalacia. Based on this animal research, he considers PVL to be a "non-specific condition." Based on the medical literature, he is convinced that the pertussis is capable of injuring the white matter

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[13] Dr. Jacoby believes that Respondent's reliance on the existence of a spastic flip is simply speculation.

of the brain. Either factor, therefore, could have caused the injuries observed. He does not present this information as proof in this case, but argues that one cannot out of hand, prove that PVL was a primary cause of injury. Owen's injuries are significant, he continues, and two possible causes exist, but one, the PVL, is a theoretical, unprovable possibility only. The PVL was not observed until after the administration of the DPT, and the possibility exists that the pertussis component of the vaccine, he believes, could have caused the PVL. Tr. at 126.

A review of Dr. Kenneth Swaiman's learned treatise Pediatric Neurology, Principles and Practice 499 (2d ed. 1994) supports Dr. Jacoby's argument:

> "A role for endotoxin also has been suggested in the pathogenesis of perinatal telencephalic leukoencephalopathy . . . an early form of periventricular leukomalacia." Id.

Dr. William Hammesfahr, M.D., for Petitioner:

Dr. Hammesfahr is a board certified pediatric neurologist who practices in Clearwater, Florida. Dr. Hammesfahr is one of Owen's treating physicians and believes that Owen has severe and extensive brain injury secondary to his vaccination. Tr. at 138. This doctor presents a novel theory of the mechanism of injury, to wit, "vasospastic disease, --vascular disease." He explains that it gives the appearance of PVL, but it is not -- it is a separate entity but just happens to have the same appearance. He states that this is a very well recognized condition which is inflammation of the vascular system and is caused by the vaccine that causes injury to the nervous system or to the blood vessels. Tr. at 138-166.

Dr. Hammesfahr's theory is intriguing, and, according to his oral testimony, his theory is well respected within his medical community as a mechanism for causing injury by vaccine. Respondent was not informed that this doctor would be testifying in this case, and Respondent's counsel was unprepared to address his allegations. Respondent requested, and the court permitted, a filling of a post-hearing brief to address this unusual method of proceedings. Dr. Hammesfahr could not cite any published material supporting his theory with the exception of those he himself published. Under the circumstances, the court considers Dr. Hammesfahr's testimony to be of interest, but not yet adequately supported outside his own community. The court is of the opinion that it would be inappropriate to consider the Doctor's testimony in determining the outcome of this case. The court, therefore, has not considered the opinion evidence of this witness, or his conclusions, in its decision.

DISCUSSION

Both experts acknowledge that Owen Burman presently suffers from chronic encephalopathy. The majority of factual evidence in this case, however, and most of the

evidence presented in the medical records, relate not to the condition of his encephalopathy, but to the child's unusual feeding problems.[14] Both experts tend to agree that although Owen's feeding intolerance may have been more severe than most, his early (pre-vaccine GER) is a side issue, not a cause of his encephalopathy, and therefore is irrelevant to the outcome of this case. The court agrees. His esophagus was found to be normal, and with one notable exception, no expert related these GER issues to his encephalopathy. Evidence shows, however, that his early colic or intolerance became something altogether different after the DPT vaccination. He was worse and he had acquired additional symptoms.

The court, does not overlook the fact that Dr. Gebara, who treated Owen after the administration of the DPT, raised the possibility that his worsened condition may have had a neurological basis -- "Odynophagia" (Painful swallowing) may suggest evidence of neurological dysfunction. In the court's experience with other vaccine cases, expert testimony has indicated that swallowing problems may be a sign of encephalopathy. Dr. Gebara believed the child should be referred to a neurologist. The experts do not allege that GRE causes brain damage, but Dr. Gebara's referral suggests that it may possibly be a result of brain damage. The evidence is clear also that the child's problems worsened after the administration of the vaccine and significantly so after he was administered his third DPT shot.

Having dismissed GER as a possible cause, the court is persuaded also that Owen's prematurity, has not been established as a significant cause of Owen's encephalopathy. Prematurity in and of itself, in this case, does not account for all of the child's symptoms. Many, if not most premature infants survive without brain damage, and a preponderance of the evidence indicates that in spite of significant feeding intolerance, he continued to develop normally until after the vaccinations.

The court is persuaded, as well, that PVL has not been established as a predominant cause of Owen's neurological injury. Respondent has convinced the court that PVL may suggest a possible role in his neurological outcome, but one cannot discern what proportion of the child's neurological abnormalities should be attributed to PVL inasmuch as evidence of an adverse reaction to the DPT is evident as well. PVL as a primary cause has simply not been proved. Dr. MacDonald admits that much of his opinion is based on the statistical probabilities. Reliance on Statistical probabilities has been held inadequate evidence of causation. *See*, Knudsen v. Secretary of HHS**,** 35 F.3d 543 (Fed. Cir. 1994). The evidence supports only the possibility that Owen Burman's encephalopathy could have been the expected progression of PVL, and that theory is speculative. Under these circumstances, Respondent's evidence is inadequate.

In short, the court finds petitioner's theory of causation more persuasive and better supported. The presence of encephalopathic signs, confirmed by a neurologist (Dr. Nigro) as "regression" and "loss of milestones" within the requisite time frame, is stronger evidence of a Table case. Tr. at 44-45, 236-37.

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[14] The court will use the term GER, throughout the remainder of this decision although other terms have been used also during these proceedings.

The court is persuaded that a preponderance of evidence supports a finding that Owen lost milestones already achieved; [15] he no longer would smile; lost head control, lost the ability to sit up propped on the sofa and would fall forward; he stopped sitting upright in his exersaucer, stopped moving his legs up and down or spinning himself using his feet and stopped pushing his legs in the exersaucer; he became less interactive, was not focusing well, was staring more, he developed increased muscle tone, more prominent in the upper extremities; his head became floppy; Mrs. Rice complained to the doctor that something had happened that changed the way he held his arms,--"something was different." Dr. Shaw observed this condition and for that reason referred Owen to a neurologist. Additionally, Owen's pre-vaccination opthalmological exam was normal; the second such exam, however, performed after vaccination, revealed retinal nerve atrophy and he was classified as visually impaired; Tr. at 50; after his third shot, his eyes began to cross; The optical nerve changes strongly suggests an acquired injury to the central nervous system as opposed to prematurity or pre-natal dysfunction.

Finally, Dr. Nigro, Owen's treating pediatric neurologist, observed that the child had a notable falling off of head circumference, from the 75th percentile before vaccination, decreasing to the 2nd percentile after vaccination constituting evidence of brain damage that occurred at some time after his shots. Moreover, his head circumference demonstrated a falling off after each of the DPT shots. And after the third shot, "it just dropped." In other words, the timing of decreasing circumference (indicating failure of brain growth) coincided exactly following the shots, from normal to "falling off the chart." Dr. Nigro insists this factor alone is evidence of the occurrence of an acute brain injury. Dr. Nigro informed Mrs. Rice that there was a regression. Genetic and metabolic concerns were ruled out. Tr. at 44-45. Dr. Nigro confirmed that he had incurred some type of a brain injury at that time. Id. at 46. These factors convince the court that Owen sustained a vaccine-related injury to the brain and was a significant contributor to his present neurological deficits.

ALTERNATIVE THEORY OF CAUSATION

In the event that a reviewing judge should disagree with the court's analysis in this case, the court finds that Petitioners may prevail on a second basis as well. The court acknowledges that it is altogether possible that a combination of both PVL and the Pertussis component of the DPT vaccine may have concurrently contributed to the child's unfortunate outcome. In the case of Shyface vs. Secretary of HHS, 165 F.3d 1344 (Fed. Cir. 1999), the Court of Appeals for the Federal Circuit held that a petitioner does not have to establish that the vaccine was the sole cause of injury, nor even the predominant cause. In Shyface, the experts for both parties agreed that two factors were present, an E. Coli infection, and a DPT vaccination, each of which could have caused the injury (in that case, death). Neither doctor was willing to apportion blame between the two, and neither doctor could establish which was more likely. They acknowledged that both had a likely role in the infant's death. The Court of Appeals held that Petitioner had to prove only that the vaccine was a "substantial factor" in the injury and that "but for" the vaccine, the child may

[15] See the earlier discussion of the rollover controversy in the section on factual findings.

not have sustained the level of injury that followed the DPT.

It is agreed in DPT cases that there are no footprints to identify a vaccine-related injury to the brain. Even autopsy cannot identify etiology or prove a vaccine link to the injury. For the most part, in the case of encephalopathy, one must rely on the clinical course. The court is convinced that sufficient evidence exists in this case that both alleged causes could have been contributors but no evidence is available to measure the levels of their contributions. For these reasons, the court is of the opinion that Petitioner in this case may prevail under the Shyface guidelines as well.

Although the following may be considered overly redundant, the court feels constrained to repeat the reasons for its decision favoring Petitioner's claim. The court is convinced that Owen sustained an encephalopathic event that, to this day, has never resolved. The court is convinced that his neurological deficits are causally related to that event at least in significant part. The first manifestation of that event was the onset of seizures that occurred within the 72 hour time frame followed by other signs and symptoms. Respondent has failed to establish, by a preponderance of evidence, that the injury was due to PVL, although it may have been a factor. According to Dr. Kenneth Swaiman's Textbook on Pediatric Neurology, Principles and Practice, (2d ed. 1994), PVL can be mild or severe depending on the size of the lesion which ranges from small areas to multi cystic involvement. No evidence was provided as to the extensiveness of Owen's PVL. The court, therefore, cannot hypothesize to the relative severity of his expected condition. Also, Dr. Swaiman writes that a role for endotoxin has been suggested in PVL's pathogenesis as Dr. Jacoby testified. Id. at 499. Ample evidence implicates the vaccine as set forth in the court's discussion, whereas PVL has been proved to be only a possible cause. The statistical probability that PVL is far more common than a vaccine injury, is irrelevant. Case law holds one may not rely on statistical probability to defeat a claim and the evidence here supports the onset of an acute encephalopathic event. Knudsen, 35 F.3d at 550.

CONCLUSION

Petitioners are entitled to compensation in an amount to be determined. The parties are directed to enter into discussions for the purpose of determining the amount of funds reasonably necessary for Owen's future care and rehabilitation.

IT IS SO ORDERED.

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E. LaVon French
Special Master