OFFICE OF SPECIAL MASTERS

No. 99-341V

(Filed: April 27, 2000)

ROBERT TERSEN and SHARON TERSEN, as parents and next friends of GREGORY TERSEN, a minor,	*	
Petitioners,	*	**TO BE PUBLISHED**
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Don Russo, Miami, FL, for petitioners.
R. Lynne Harris, Washington, DC, for respondent.

DECISION

MILLMAN, Special Master

On May 26, 1999, petitioners filed a petition on behalf of their son, Gregory Tersen (hereinafter, "Gregory"), 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

[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

a prima facie case pursuant to 42 U.S.C. § 300aa-11(c) by showing that: (1) they have not previously collected an award or settlement of a civil action for damages arising from the vaccine injury; and (2) DPT vaccine was administered to Gregory in the United States.

Petitioners allege that Gregory had an on-Table anaphylaxis and encephalopathy after receiving his first DPT vaccination on March 1, 1997. Respondent admits that Gregory had a vaccine reaction, but denies that it caused any permanent damage and ascribes Gregory's condition to his extreme prematurity, including periventricular leukomalacia (PVL). Because Gregory was born with extreme prematurity, the undersigned also considers whether or not petitioners have made a case for significant aggravation of Gregory's pre-existing encephalopathy. The medical records also depict Gregory as having a hypotensive-hyporesponsive shock collapse (HHE) and anaphylaxis post-vaccination.

The court held a hearing in this case on February 18, 2000. Testifying for petitioners was Dr. Michael Duchowny. Testifying for respondent was Dr. Mhairi G. MacDonald.

FACTS

Gregory was born on December 19, 1996, weighing 725 grams or one pound 14 ounces. Med. recs. at Ex. 3, p. 6, Ex. 8, p. 4. He was born at 26 weeks and was preterm. His mother had previously had three first trimester losses, one term delivery at 36 weeks gestation who died from pulmonary hypoplasia, and a prior pregnancy affected with Down's syndrome. Med. recs. at Ex. 4, pp. 8, 25.

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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.

Gregory was hospitalized at Lee Memorial Hospital from December 19, 1996 to March 14, 1997. Mrs. Tersen had bleeding for three weeks prior to delivery. Her membranes were ruptured for about 20 days prior to delivery. Some fetal distress was marked by decelerations. Med. recs. at Ex. 3, p. 7.

At birth, Gregory had respiratory distress, bronchopulmonary dysplasia (BPD), apnea of prematurity, patent ductus arteriosus, and nosocomial sepsis. Med. recs. at Ex. 3, p. 5. He was on ventilatory support with surfactant administered. He was able to be extubated by the fourth day of life. He was on room air by the fifth and sixth days of life. But he had a recurrent need for positive pressure support thereafter. He was back on ventilatory support at one month of age. He was extubated again to room air by 52 days of age, and placed on a nasal cannula until discharged from the hospital. Med. recs. at Ex. 3, p. 7.

Gregory had a head circumference at birth of 22 cm. At discharge, it was 31 cm, which is less than the 10th percentile. At the age of 64 days, he had myoclonic jerks.

On February 21, 1997, Dr. Margie A. Morales, a pediatric neurologist, examined Gregory. At 6:30 p.m., while he was sleeping, he had brief rhythmic extensor movements of both lower extremities which could not be stopped when he was held. Other than a high arch palate, he had no other dysmorphic features. Gregory was alert, awake, and active. He had mild increased tone with the lower extremities being greater than the upper extremities. Dr. Morales concluded that Gregory most likely had myoclonus of sleep, but one needed to exclude myoclonic seizures. Med. recs. at Ex. 3, pp. 18-19.

On February 22, 1997, Gregory underwent an EEG, which was normal, as Dr. Morales interpreted it. Med. recs. at Ex. 3, p. 39. An EEG done on February 24, 1997 was normal. He was

felt to have benign myoclonus as these episodes appeared to occur while he was sleeping. Med. recs. at Ex. 3, p. 9.

Early in the morning of March 1, 1997, at the age of 71 days, he received his first DPT vaccination and appeared to have a shock collapse type of reaction. He became quite pale and hypotonic, and he clinically desaturated. A spinal tap showed that his protein rose to 135. He developed more marked myoclonic jerks (neonatal seizures). Med. recs. at Ex. 3, p. 9.

On March 1, 1997, Dr. William F. Liu wrote that Gregory received the DPT and appeared to have an acute reaction immediately after injection. He became extremely pale and was noted to have poor respiratory effort requiring blow-by oxygen. He had clinical as well as monitored (pulse oximeter) desaturations. He had several repetitive episodes of desaturation. Clinically, Gregory appeared fairly well at the time of examination, although the nurses noted frequent myoclonic jerks. Dr. Liu saw myoclonic jerks of Gregory's right arm and possibly left arm lasting less than one minute which did not abate with restraint. Med. recs. at Ex. 3, p. 128.

On March 2, 1997, Dr. Liu wrote that Gregory's desaturations might reflect some persistent evidence of chronic lung disease which the additional problems of an immunization reaction of shock collapse might have exacerbated. Med. recs. at Ex. 3, p. 130. He also wrote that an immunization reaction of shock collapse is noted to occur in about 1 out of every 1,750 dosages of DPT. Med. recs. at Ex. 3, p. 131.

An EEG done on March 3, 1997 was abnormal, demonstrating rhythmic sharp wave activity associated with myoclonic jerks, seen originating primarily in the right frontal cerebral regions. Gregory was diagnosed with seizures. Med. recs. at Ex. 3, pp. 9, 40.

Dr. Morales saw Gregory on March 3, 1997, at 5:00 p.m. Med. recs. at Ex. 3, p. 134. He was alert, awake, and in no acute distress. His anterior fontanelle was soft and not bulging. He had

positive gag and suck reflex. He had elevated tone most prominent in the lower extremities. His deep tendon reflexes were brisk. He had positive grasp. His routine blood work was normal. She prescribed Phenobarbital. Id.

On March 4, 1997, Gregory was doing better. He had no episode associated with seizure activity, but two episodes of desaturation associated with feedings occurring spontaneously. Med. recs. at Ex. 3, p. 135.

Dr. D.L. Caangay noted on March 4, 1997 that in between seizures, Gregory was otherwise doing very well and gaining weight. His physical examination was normal. Dr. Caangay stated, "I cannot find any neurological deficits. The baby is alert and active." Med. recs. at Ex. 3, p. 136.

On March 5, 1997, Dr. Liu wrote his impression: presumptive neonatal seizures associated with DPT immunization versus an underlying central nervous system (CNS) process that may have been exacerbated by the vaccination. Med. recs. at Ex. 3, p. 138.

A third EEG done on March 6, 1997 continued to show epileptiform activity originating in the frontal regions, but was improved from his March 3, 1997 EEG. Med. recs. at Ex. 3, pp. 9, 41. The doctors could not rule out an underlying predisposition to seizures marked by what appeared to be benign myoclonus prior to his immunization and overt seizure activity after it. He had an abnormal brain stem audio evoked response screen. Med. recs. at Ex. 3, p. 5.

On March 9, 1997, Dr. Liu wrote that Gregory had chronic lung disease and was on Aldactone and Diuril, but off Theophylline. It was possible some additional desaturations were associated with breakthrough seizures. The seizure process was presumably secondary to DPT immunization. Dr. Liu could not rule out an underlying process since Gregory was already having myoclonic jerks prior to the immunization which the immunization significantly exacerbated. Med. recs. at Ex. 3, p. 145.

Dr. Mohamed M. Faisal noted on March 10, 1997 that Gregory's clinical examination was neurologically normal. His CNS remained unchanged. Gregory's pupils were equal and reactive to light. He was alert, and had good suck, swallow, and normal tone. Dr. Faisal did not see any hypertonicity. Med. recs. at Ex. 3, p. 146.

On March 12, 1997, Gregory was given IPV (polio). Dr. Liu noted that Gregory had received HiB and Hepatitis B vaccinations on the previous day. Med. recs. at Ex. 3, p. 151. Dr. Liu stated on March 13, 1997 that Gregory had not had any episodes of myoclonus since March 8, 1997. Med. recs. at Ex. 3, p. 152.

On April 1, 1997, Dr. Morales at the Nemours Children's Clinic recorded that Gregory's apnea alarms occurred predominantly when he was in deep sleep. He had brief, daily rhythmic clonic activity involving the upper or lower extremities. Gregory was on Phenobarbital and oxygen. On physical examination, he was alert, awake, focused, and beginning to track. He had good suck and gag reflex. He had minimal head lag. He had abnormalities in tone and brisk reflexes. Med. recs. at Ex. 11, p. 5.

Also on April 1, 1997, Gregory saw Dr. Eduardo J. Riff, a pediatric pulmonologist, who diagnosed mild BPD and apnea of prematurity. Med. recs. at Ex. 11, p. 19.

On April 23, 1997, Dr. Riff noted that since the prior clinic visit, Gregory's mother had been giving Gregory his oxygen intermittently. Dr. Riff recorded, "I have cautioned the mother about discontinuing the supplemental oxygen. Awake his oxygen saturation is marginal and asleep it is clearly low. I have instructed the mother that Gregory needs to be on supplemental oxygen continuously." Med. recs. at Ex. 11, p. 21.

On July 21, 1997, Dr. Morales noted that Gregory had been doing well. He had been off an apnea monitor for one and one-half months. He had been off oxygen for two weeks. Gregory did

not have any seizures and was tolerating Phenobarbital. Developmentally, he smiled, cooed, and laughed, but did not squeal. He brought his hands to midline and to his mouth. He attempted to reach for objects. His head circumference was close to the 2nd percentile. His weight was in the 10-15th percentile. He had convergent strabismus, the right more than the left. He was showing motor delays, abnormalities in tone, and hyperreflexia. He remained without seizures. Med. recs. at Ex. 11, p. 8.

On July 29, 1997, Gregory had a CT scan of his brain which showed probable agenesis of the corpus callosum. Med. recs. at Ex. 9, p. 1. Also on July 29, 1997, he had an EEG, which Dr. John Osterman interpreted as normal. Med. recs. at Ex. 9, p. 2.

On September 15, 1997, Dr. Riff noted that supplemental oxygen had been discontinued and Gregory had been doing very well. Med. recs. at Ex. 11, p. 23.

On January 19, 1998, Dr. Morales noted that Gregory was alert and awake. He established eye contact and smiled. He had good head control, but was unable to sit independently. He had increased tone and hyperreflexia throughout. His corpus callosum appeared thin, but present. He had mild enlargement of the subarachnoid spaces with prominent sylvan fissures. He did not have seizures. He was off Phenobarbital. He had delays in all areas of development, most prominently in his motor and expressive language skills. He had spastic cerebral palsy (CP) with persistence of developmental reflexes, spastic diparesis, and hyperreflexia. Med. recs. at Ex. 11, p. 12.

On February 2, 1998, Gregory had an MRI which was abnormal. He had white matter with decreased volume suggestive of possible PVL. Med. recs. at Ex. 11, p. 53. On March 30, 1998, Gregory saw Dr. J. Javier Muniz-Quinones, a pediatric gastroenterologist, for feeding refusal. Med. recs. at Ex. 11, p. 58.

On September 15, 1998, Dr. Morales recorded that Gregory had remained seizure-free. He had abnormal hearing screens and was having episodes of vomiting with poor weight gain. She started him on Propulsid, and the vomiting and weight loss stopped. Gregory did not sit independently. He had occupational therapy twice a week, physical therapy three times a week, and speech therapy once a week. He did not show developmental regression.

He did not have loss of consciousness, eye blinking, eye fluttering, or tonic or clonic activity. He did have chin quivering. His head circumference was less than the 2nd percentile. On February 2, 1998, an MRI was performed which was abnormal, showing decreased volume of white matter suggestive of PVL. His corpus callosum was intact but attenuated. Dr. Morales' impression was that Gregory had developmental delay secondary to spastic CP. He was a premature infant of 26 weeks gestation. He had visual impairment secondary to retinopathy, and prominent strabismus. He had an abnormal hearing screen, and language delay most likely secondary to cognitive deficits. Gregory had poor weight gain and gastroesophageal reflux, and was currently on Propulsid. He had a history of neonatal seizures secondary to a reaction to DPT, but remained without further seizures, and was not on anticonvulsants. His most recent EEG was normal. Med. recs. at Ex. 11, p. 15.

On October 29, 1998, Gregory saw Dr. Joseph J. Dallessio at Lee Memorial Hospital. Gregory had apnea and CP. The history given was that he was premature at one pound 14 ounces. He was delivered secondary to an incompetent cervix. He had one DPT vaccination and developed seizures with that, and has been developmentally delayed as well. He did not have any current seizure disorder and was not on any anticonvulsants. Gregory was a 22-month-old child functioning at a 3-4 month level. Med. recs. at Ex. 8, p. 4.

Written Submissions

Respondent filed Exs. C-F, consisting of literature pertaining to this case:

Ex. C: "Neonatology: Pathophysiology and Management of the Newborn," by Forrest C. Bennett, chap. 59 of Developmental Outcome, 5th ed., edited by Gordon B. Avery, Mary Ann Fletcher, and Mhairi G. MacDonald (1999);

Ex. D: "Outcomes of Extremely Low Birth Weight Infants," by M. Hack, H. Friedman, and A. Fanaroff, *Pediatrics* 98:931-37 (1996);

Ex. E: "Neurologic Disorders in the Newborn, Brain Injury in the Premature Infant," by Joseph J. Volpe, Clinics in Perinatology 24:567-87 (1997);

Ex. F: "Neonatal Neurology, Consensus and Controversy in the Clinical Management of Neonatal Seizures," by Eli M. Mizrahi, Clinics in Perinatology 16:485-500 (1989).

In Ex. C, the author describes very low-birth weight (VLBW) infants as at risk for, among other things, extensive cystic PVL and prolonged seizures or other abnormal neurologic behavior. Ex. C, p. 1482. "The major neurosensory impairments associated with prematurity are cerebral palsy, particularly of the spastic diplegia type; mental retardation...; sensorineural hearing loss; and visual impairment." Ex. C, pp. 1483-84. The author states that major developmental disabilities may occur with a chronic seizure disorder and are usually clinically apparent by 2 years of age. Ex. C, p. 1484. The disability rate in boys exceeds that in girls, and in LBW infants, it is two to five times more common than in full-birth weight infants. Id. The percentage of LBW infants who survive with one or more major impairments and weigh less than 1,000 grams is 25%. Id. In those weighing less than 800 grams at birth, the prevalence of major impairments in survivors is 22%. Id.

The most common disability in VLBW infants is CP, which occurs in 6-10%. Two-thirds of children born with spastic diplegia are born before 37 weeks of gestation. Id. Spastic diplegia is a clinical manifestation of PVL, which is caused mainly by hypoxic-ischemic injury to the periventricular white matter. Ex. C, p. 1485. Mental retardation occurs in 4-5% of VLBW infants.

Id. They also have increased risk for sensorineural and conductive hearing loss at a rate of 2-3% or even 5-9%. Id.

Developmental disabilities for LBW infants include cognitive delays, speech and language disorders, persistent neuromotor abnormalities, and perceptual problems. Ex. C, p. 1487. However, "individual developmental outcome remains very difficult to predict prospectively with accuracy in the NICU, and infants with apparently similar neonatal courses may develop entirely differently." Ex. C, p. 1488.

Most LBW infants have an IQ within the average range. Id. The largest deficit in full-scale IQ occurred among children weighing less than 1,500 grams at birth. Ex. C, p. 1489. Low- birth weight and gestational age were negatively correlated with language development as well. Id. The neuromotor development of LBW, premature infants for their first two years is more delayed and worrisome than for full-term, healthy infants. Ex. C, pp. 1489-90.

Exhibit D mentions that infants of 600 to 750 gram birth weight have a 35% rate of mild to severe neurodevelopmental impairment. Ex. D, pp. 935-36. Exhibit E states that 5-15% of infants born weighing less than 1,500 grams have major spastic motor deficits (CP) with intellectual deficits commonly accompanying them, and 25-50% have less prominent developmental disabilities involving motility, cognition, and behavior. Ex. E, p. 567.

The major neurologic manifestation of brain injury in the premature infant is spastic motor deficit, with the lower extremities more affected than the upper extremities, and commonly accompanied by intellectual deficits. Id.

"The major neuropathology for the spastic motor deficits, with or without accompanying intellectual deficits, are periventricular leukomalacia and periventricular hemorrhagic infarction." Ex. E, pp. 567-68.

"Periventricular leukomalacia, in its overt form, refers to necrosis of white matter ... observed particularly often in ... the premature infant...." Ex. E, p. 574. "The pathologic features of periventricular leukomalacia are distinctive and consist primarily of both focal periventricular necrosis and more diffuse cerebral white matter injury...." Id.

"The major long-term sequela of periventricular leukomalacia is spastic diplegia, the major motor deficit subsequently observed in premature infants." Ex. E, p. 576. "Patients with spastic diplegia with significant involvement of upper extremities exhibit other manifestations of severer cerebral disturbance, including prominent intellectual deficits." Id.

"Neonatal seizures indicate the presence of central nervous system (CNS) dysfunction and may contribute to additional brain injury." Ex. F, p. 485. "The seizures that occur with a close association to EEG seizure activity are generated by an epileptic mechanism." Ex. F, p. 487. "Neonatal seizures are thought to be the most distinctive and frequent clinical sign of CNS dysfunction to the newborn." Ex. F, p. 490. "Repeated epileptic seizures may be deleterious to the developing brain." Ex. F, p. 491.

TESTIMONY

Dr. Michael Duchowny testified first for petitioners. Tr. at 4. He is the director of the neuroscience program at Miami Children's Hospital and was a Fellow in Neurology at Harvard Medical School. Tr. at 5-6. He received his medical degree from Albert Einstein and interned at the University of Chicago. Tr. at 5. He is a clinical professor of neurology in pediatric neurology. Tr. at 6. In 1991, the Florida Department of Health and Rehabilitation Services requested that he represent them in evaluating birth-related injuries. *Id.* He is board-certified in pediatrics and neurology, and is on many editorial boards. Tr. at 7.

Dr. Duchowny first saw Gregory as his patient on February 23, 1999. *Id.* Gregory was non-fluent, but quite attentive. Tr. at 8. All his limbs were spastic and his reflexes were brisk. *Id.* He was two years old and could not sit without support, which is a six-month milestone. *Id.* He had very simple awareness. *Id.* For instance, he knew his body parts. Tr. at 8-9. One eye had laziness of movement. Tr. at 9. He had mouth and tongue delay. *Id.* He also had marked speech delay and motor delay. *Id.* He had severe deficits. *Id.* Dr. Duchowny separates all these deficits from sequelae of prematurity because he considers Gregory's condition to reflect much more widespread impairment. *Id.* He is more globally damaged in motor and language areas than prematurity would result in. *Id.* Gregory has had surgery for contractures and has incredibly severe motor damage. *Id.*

Dr. Duchowny testified that before Gregory received his DPT vaccination, Dr. Duchowny would have been able to determine if he had neurological impairment because he examines two-month-old children. Tr. at 10. On February 21, 1997, Dr. Morales, whom he regards as a very competent pediatric neurologist, wrote that Gregory was doing fairly well on the whole although he had quivering of his chin when crying and tremoring which she felt to be normal. Tr. at 10-11. His EEG on February 22, 1997 was normal and he was felt to have benign myoclonic movements of sleep. Tr. at 11. Dr. Morales did not feel that Gregory needed neurologic follow-up at this point. Tr. at 12.

In the early morning of March 1, 1997, Gregory received DPT and the records are clear that he had an acute change. *Id.* He decompensated and went into a shock-like state. Tr. at 13. He had pallor, loss of motor tone, and desaturation (he was low on oxygen). *Id.* His myoclonic activity became more prominent. *Id.* There was no infectious process. *Id.* His second EEG on March 3, 1997 showed epileptiform activity and new onset of seizures. *Id.* Gregory was started on

Phenobarbital. *Id.* He had acute deterioration and a central nervous system reaction to the pertussis component of the DPT. *Id.* His shock was due to DPT. Tr. at 13-14. His general condition after vaccination was pertussis-related. Tr. at 14. The records after vaccination show a change in Gregory's overall condition: autonomic instability (heart rate drop), periodic respirations, and seizure-like phenomena. *Id.* It was a severe medical event. *Id.*

Dr. Duchowny stated that premature infants are at higher risk for DPT reactions, although prematurity is not a contraindication to administering DPT. Tr. at 15. Gregory's neurological disabilities are likely to be permanent. Tr. at 16. Dr. Duchowny found it difficult to answer what would have been Gregory's sequelae of prematurity if he had not received DPT. Tr. at 16-17. Gregory weighed 725 grams at birth. Tr. at 16. He was in the high-risk category. Tr. at 17. His MRI showed abnormalities which are seen in prematurity but he could have done well nevertheless. *Id.* Without the DPT, Gregory would have done a lot better; he would have had mild disability. *Id.*

Gregory's DPT sequelae were motor, language, and cognitive disabilities, and seizures. Tr. at 17-18. To explain the motor disability connection with DPT, Dr. Duchowny testified that the mechanism of DPT encephalopathy involves the white matter of the deep areas of the brain, typically affecting the motor tracts. Tr. at 19. It is autoimmune and perivascular (surrounding the blood vessels), causing compromise. *Id.* To explain the language disability, Dr. Duchowny testified that the tracts for visual, motor, and spatial abilities go through the white matter. *Id.*

Dr. Duchowny testified that Gregory's periventricular leukomalacia is related to his prematurity. Tr. at 20.

Dr. Duchowny linked Gregory's cognitive deficit to the DPT. Tr. at 21. Reactions to DPT vary from catastrophic to mild deterioration. *Id.* Gregory was at risk of epilepsy even though his seizures stopped. Tr. at 21-22. Gregory is in the moderate category. Tr. at 22.

Dr. Duchowny stated that seizures are related to an autoimmune reaction. Tr. at 23. Gregory had an acute DPT encephalopathy and now has a static encephalopathy. *Id.* Gregory may have also had anaphylaxis. Tr. at 24.

Gregory received the DPT at two months of age. Tr. at 25. His brain had matured past the point of vulnerability for periventricular lesions. *Id.* It is certainly true that very low-birth weight infants with severe postnatal courses have the types of deficits that Gregory has. Tr. at 27. But had he not received DPT, Gregory would not have had this course. *Id.* Gregory did extremely well before DPT. *Id.* Now he has much more sequelae than he would have had. Tr. at 27-28. The long term outlook for very low-birth weight infants is variable. Tr. at 29. One out of four or five very low-birth weight infants is impaired. Tr. at 30. Periventricular lesions relate to spasticity. Tr. at 31.

Gregory's retinopathy of prematurity is due to his prematurity, not to DPT. Tr. at 34. His hearing loss may be due to prematurity. Tr. at 37. Dr. Duchowny needs more information to determine that. *Id.*

On February 21, 1997, which was Gregory's two-month examination before he received DPT, he could have had pathological (abnormal) reflexes, deep tendon reflex abnormalities (too high or too low), and abnormal muscle tone (resting state of contraction of the muscles), which are all observable at two months, but Gregory did not have them. Tr. at 35-36.

Dr. Duchowny testified that he consults on neonates all the time. Tr. at 39-40. When he saw Gregory, Gregory was two years old. Tr. at 40. It is difficult to test a two-year-old but one can do

it. *Id.* He sees a large number of children that age. *Id.* Based on their behavior and acquisition of normal milestones, he can evaluate two-year-olds . *Id.*

Gregory was born at 26 weeks. Tr. at 41. He had problems supporting respiration because of his immature lungs. *Id.* He had a stable course and required minimal respiratory support, only a nasal cannula. *Id.* He was intubated from his date of birth until February 21, 1997. Tr. at 42. He was placed on less intrusive constant positive air pressure or C-PAP on February 22, 1997. *Id.* His desaturation episodes increased and he deteriorated and was put back on C-PAP. *Id.* He was reintubated and placed on a ventilator. Tr. at 43. He was taken off the ventilator but they could not wean him from C-PAP. *Id.* He was back on a nasal cannula with 100% oxygen. Tr. at 43. He remained on room air for two days, and had frequent desaturation spells. *Id.* Gregory was always on Theophylline to prevent apnea, and the doctors added Aldactone and Diurel. Tr. at 43-44. On February 24th, he was on room air and relatively stable with a few desaturations. Tr. at 44. On February 27th, the doctors withdrew Aldactone, Diurel, and Theophylline. *Id.*

On February 20, 1997, Dr. Morales noted increased tone in Gregory's lower extremities greater than in his upper, which disappeared after a while. Tr. at 45. Gregory's CT scan on February 22nd showed periventricular leukomalacia. Tr. at 46. The white matter was more hypodense, showing early PVL. *Id.* Dr. Morales prescribed a neurodevelopmental follow-up, but at Lee Memorial Hospital, Dr. Morales said there was not to be a follow-up. *Id.*

Dr. Duchowny testified that he based his opinion that Gregory had a DPT reaction on the description in the medical records and his neurological outcome. Tr. at 47. Gregory went into shock, and had an acute change. Tr. at 47-48. Dr. Duchowny knows what Gregory looked like at the age of two years. Tr. at 48. On March 1, 1997 after midnight, Gregory did not have immediate

neurological symptoms. Tr. at 48-49. Dr. Lui indicated that Gregory had a CNS reaction to DPT. Tr. at 49-50.

On March 3, 1997, two days after the DPT, Dr. Morales noted that Gregory was alert, awake, had a positive gag and a positive suck. Tr. at 52. Dr. Duchowny testified that some of Gregory's spasticity in his lower extremities was due to his PVL, but the spasticity in his upper extremities would not be due to his PVL. Tr. at 55. PVL can involve the upper motor tracts, but it generally does not. Tr. at 56.

Dr. Duchowny testified that Dr. Morales did a quick examination, which is why she did not find Gregory to have severe neurologic deficits. Tr. at 57-58. Any abnormal neurological findings were subtle. Tr. at 58. She did not see gross neurological disease. *Id.*

Dr. Mhairi G. MacDonald testified for respondent. Tr. at 61. She is a pediatrician and neonatologist who is director of neonatology at Loudon Hospital Center. Tr. at 61-62. She trained at Johns Hopkins. Tr. at 63. She was director and chairman at Children's Hospital for 20 years. *Id.* In 1976, she finished a neonatology fellowship. Tr. at 64.

Gregory was born weighing one pound nine ounces or 730 grams. *Id.* The average full term baby weighs 7 ½ pounds. *Id.* He was at high risk of not surviving. Tr. at 65. He had a 25% likelihood of having severe anomalies. Tr. at 67. His discharge diagnosis included respiratory distress syndrome due to immaturity of his lungs. Tr. at 68. He had little detergent or surfactant in his lungs, a common condition of prematurity. *Id.* His bronchopulmonary dysplasia was due to mechanical airways secondary to intubation and received oxygen (an iatrogenic condition). *Id.*

Gregory had apnea of prematurity due to immature areas of the brain that control breathing. Tr. at 69-70. His treatment consisted of Theophylline and C-PAP. Tr. at 70. He had patent ductus arteriosus (flooding the lungs with blood) for which he took Indomethacin. Tr. at 71. Dr.

MacDonald has treated several hundred or 1,000 premature babies of 725 grams weight. Tr. at 72. She sees long-term problems. *Id.*

Gregory would have been followed because of the risk of cerebral palsy. *Id.* The degree of cerebral palsy depends on the extent of the periventricular leukomalacia. Tr. at 72-73. Spastic diplegia does not affect only the lower extremities. Tr. at 73. It affects all the limbs. *Id.* The majority of these babies have spastic quadriplegia, often with the legs worse than the arms. *Id.* Very few are normal. Tr. at 75. Their major impairments are spastic quadriplegia, severe mental retardation (below 80 IQ), partial blindness, and an inability to feed themselves. Tr. at 77. Gregory had a fairly rocky course as a neonate. *Id.*

In explaining what a neurologist would find on examination, Dr. MacDonald stated that a neurological examination is not possible and would not tell you anything. Tr. at 88. In a premature baby, one can tell at 12 months of age for the first time that the baby has motor abnormalities and CNS problems. Tr. at 89-90. If it were a full-term baby, one could discern motor abnormalities and CNS problems at 9 months for the first time. *Id.* At 4 months of age, one can note things and notify the mother that they may persist. Tr. at 90. At 2 months of age, an asymmetric tonic neck reflex is normal. *Id.* The worst child will be obtunded which can show up earlier than 4 months. Tr. at 90-91.

Dr. MacDonald stated that Gregory's respiratory difficulty set him up to have a response to anything and for a deterioration. Tr. at 106. His episodes of desaturation were not unusual. *Id.* The doctors could not wean him from oxygen. Tr. at 107. He had severe bronchopulmonary dysplasia. *Id.* His jerking started in January 1997. *Id.* On February 20, 1997, Dr. Morales noted increased tone in all his extremities, the lower more than the upper. *Id.* Dr. MacDonald would worry about that, but could not predict severe damage. Tr. at 108. He was diagnosed with benign myoclonus. *Id.*

Gregory's first EEG which was normal does not mean his brain was normal because seizures are deep in the brain and do not manifest on the surface. Tr. at 109. In his two abnormal subsequent EEGs, he had clinical jerking with EEG correlations and vice versa. *Id.* One does not expect every baby to have myoclonic jerks. *Id.* This alerts the doctor. *Id.* There is a statistical likelihood of having long-term serious neurological sequelae. Tr. at 110.

On the February 22, 1997 CT scan, Gregory's white matter was more hypotensive. *Id.* This is classic as diffuse periventricular leukomalacia. Tr. at 110-11. It does not usually show up on ultrasounds and is more likely in white males who weigh less than 800 grams and have early episodes of hypotension and poor birth course. Tr. at 111. The long tracts which come down from the surface of the brain are particularly vulnerable to widespread damage. Tr. at 112. The legs will always be affected. Tr. at 113. The whole body is affected. *Id.*

Gregory had diffuse loss of white matter density, which means that more than his legs would be involved. *Id.* Periventricular leukomalacia can cause long-term morbidity. Tr. at 114. It means bad things. *Id.* On March 2, 1997, the neonatologist noted that Gregory's DPT reaction exacerbated his chronic lung disease. Tr. at 114-15. The doctors should not have given Gregory DPT, Dr. MacDonald testified, because he needed more oxygen after it and regressed. Tr. at 115. Even at one year of age, when his Achilles tendon was released, he became apneic. Tr. at 115-16. Doctors should not give DPT to children who weigh less than 2000 grams, and Gregory received it. Tr. at 116. There is no evidence he went into shock, but no one took his blood pressure. *Id.*

Gregory did after DPT what he was doing before but more of it. *Id.* His myoclonic jerks were exacerbated, but this was a transient response. *Id.* His mental status did not change. Tr. at 117. The episode was not at all significant for his long-term prognosis. *Id.* All his respiratory drugs had been removed for 24 hours before he received DPT. *Id.*

After DPT, Gregory ate without difficulty. *Id.* On March 3, 1997, Dr. Morales placed Gregory on Phenobarbital which was a dicey response because it could have made his seizures worse because they were myoclonic, but it helped Gregory. Tr. at 117-18. His second and third EEGs noted changes, but the July EEG was normal. Tr. at 118. An EEG has a 50% chance of not picking up what one is looking for. *Id.* The amount of artifact makes an EEG difficult to interpret. Tr. at 118-19.

Dr. MacDonald testified that Gregory's DPT brought out the electrical activity in his brain so the EEG could pick it up, but it did not cause permanent brain damage. Tr. at 119. On April 1, 1997, Dr. Morales saw Gregory. *Id.* His parents reported brief rhythmic activity in the morning. *Id.* By July 21, 1997, Dr. Morales noted that Gregory was not having any more seizures. *Id.* On July 29, 1997, his EEG was normal. *Id.* He was developmentally normal for his adjusted age on April 1, 1997. Tr. at 120. He had close monitoring for cerebral palsy and mental retardation. Tr. at 121. On July 21, 1997, he was noted to have motor delays, abnormal tone, and hyperreflexia. *Id.* On January 18, 1998, he was diagnosed with spastic cerebral palsy. Tr. at 122.

Dr. MacDonald testified that DPT did not cause Gregory's current condition. Tr. at 123. His current condition is all secondary to his extreme immaturity and his periventricular leukomalacia. *Id.* His eye findings are more typical of his size. Tr. at 124. Dr. MacDonald has treated about 10 cases of DPT encephalopathy over 30 years. Tr. at 124-25. We do not know if Gregory has cognitive deficits. Tr. at 133. Gregory was high risk because he would have required follow-up. *Id.* He was having an apneic spell after his DPT because he was cyanotic and dusky. Tr. at 137.

DISCUSSION

This is not an easy case to decide. Petitioners have a superb pediatric neurologist, Dr. Duchowny, who is accomplished in his practice, his teaching, and his service for the State of Florida evaluating neurologic deficits in children. He testified that DPT caused Gregory to be far more damaged than he otherwise would have been. But Gregory has a condition, PVL, which Dr. Duchowny attributed to his prematurity and the sequelae of PVL are exactly what Gregory has. The literature respondent submitted and the testimony of respondent's expert, Dr. MacDonald, a neonatalogist who has vast experience dealing with neonates in critical care, described the expected sequelae of PVL: spastic quadriplegia, motor deficits, hearing defects, retinopathy, and most probably cognitive defects.

Dr. Duchowny ascribed Gregory's retinopathy to his prematurity but was unsure of the cause of his hearing difficulties. He demurred from ascribing Gregory's spastic quadriplegia to his prematurity because he said PVL is frequently associated with spastic diplegia, not quadriplegia. However, when PVL is severe enough, it can affect the upper extremities as well.

What makes this case so difficult is that Gregory did indeed have a DPT reaction. He went into shock, and he started seizing. He might even have been seizing before vaccination, according to Dr. Morales, whom Dr. Duchowny knows and highly respects. The issue is whether DPT significantly aggravated Gregory's preexisting encephalopathy and vulnerability to seizing, or Gregory's post-vaccination course tracked what would be expected in an infant born at 26 weeks who weighed less than 800 grams and has PVL, together with prevaccination hypertonicity, severe apnea, and myoclonic movements.

Dr. Duchowny testified that Gregory's post-vaccinal seizures caused encephalopathy and worsened his condition. However, four doctors, Drs. Liu, Morales, Caangay, and Faisal, saw him within days of the vaccination and none diagnosed any encephalopathic symptoms. Gregory's

second EEG reflected epileptiform activity and his third EEG, although better, was abnormal. But his subsequent EEGs were normal after he was placed on Phenobarbital. As Dr. Morales noted, by July 21, 1997, Gregory was off Phenobarbital and not having any seizures.

The Vaccine Act requires that the sequelae of a vaccine injury persist for longer than six months. 42 U.S.C. § 300aa-11(c)(1)(D)(I). March 1st through July 21st is less than five months. Without Gregory's having had acute encephalopathy or a seizure disorder that continued beyond six months, the undersigned cannot find any basis for Dr. Duchowny's testimony that Gregory's DPT reaction resulted in permanent sequelae or that the Vaccine Act permits compensation here.

When Dr. Duchowny described how a DPT reaction affects the white matter of the brain and the motor tracts, causing motor deficits, he ignored the role of Gregory's PVL. If Dr. Duchowny had testified that DPT, not prematurity, caused Gregory's PVL, then petitioners would have prevailed in this case. But he did not.[2] He testified that prematurity caused Gregory's PVL. The court holds that all of Gregory's current condition is due to his prematurity which led, as a primary complication, to his PVL, which caused his CP, retinopathy, hearing defects, and probable cognitive deficits.

The undersigned recognizes that Gregory has a static encephalopathy post-vaccination as he did pre-vaccination, but he did not have an acute encephalopathy. When Drs. Liu and Morales examined him post-vaccination, he seemed awake and alert. His anterior fontanelle was soft and flat.

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[2] There is some medical literature discussing the role of endotoxin in the production of telencephalic leukoencephalopathy, representing perhaps an early form of PVL, but there is no testimony in the instant case that Gregory had telencephalic leukoencephalopathy. See F.H. Gilles, et al., "Neonatal endotoxin encephalopathy," *Ann Neurol* 2:49 (1977), and F.H. Gilles, et al., "Endotoxin leucoencephalopathy in the telencephalon of the newborn kitten," *J Neurol Sci* 27:183 (1976). Moreover, Dr. Duchowny testified that Gregory's brain had matured past the point of vulnerability for periventricular lesions by the time he received DPT.

He was in no acute distress. He did not have any change in affect. Drs. Caangay and Faisal confirmed no neurologic deficits.

Dr. Duchowny testified that he can tell in two-month-old infants whether they are neurologically compromised (although Dr. MacDonald questioned whether neurologic deficits would appear before the age of four months). When asked to explain why Dr. Morales did not find Gregory to be neurologically challenged after his DPT reaction, Dr. Duchowny surmised that she had examined him quickly and the changes were subtle. That is inconsistent with his earlier testimony that Gregory experienced a severe medical event. As for Dr. Liu, Dr. Duchowny's explanation for his failure to find neurologic harm was that Dr. Liu is not a neurologist. These are not satisfactory explanations nor do they deal with Dr. Caangay's and Dr. Faisal's failures to find neurologic deficits. It is inconceivable that all four doctors were in a rush and unable to detect subtle neurologic damage after a supposedly severe medical event.

If the undersigned is to believe Dr. MacDonald, i.e., that neurologic deficits do not appear until the age of four months, then Dr. Duchowny's testimony that he can detect neuropathy in a two-month-old makes no sense. On the other hand, if the undersigned is to believe Dr. Duchowny, i.e., that one can detect neuropathy in a two-month-old, then Dr. Morales should have found Gregory to be significantly worse neurologically post-vaccination (especially since Dr. Duchowny stated that she is highly competent). And she did not, as her three colleagues did not.

The court finds it ironic that Dr. Duchowny testified that, although significantly premature, Gregory was appropriately vaccinated at the age of two months, whereas Dr. MacDonald stated that the doctors should never have vaccinated him because he was at high risk for complications since he weighed under 2,000 grams. But, Dr. MacDonald continued, although Gregory had an apneic episode, he did not have permanent sequelae from his vaccine reaction.

The undersigned agrees with Dr. Duchowny that one cannot tell just from prematurity whether a child will be abnormal, have CP or spastic diplegia plus other deficits. But here, we have a child with PVL whose known sequelae match Gregory's deficits completely. "The major long-term sequela of periventricular leukomalacia is spastic diplegia, the major motor deficit subsequently observed in premature infants." R. Ex. E, p. 576. "Patients with spastic diplegia with significant involvement of upper extremities exhibit other manifestations of severer cerebral disturbance, including prominent intellectual deficits." Id. See also R. Ex. C (discussing the array of sequelae of prematurity, including CP, motor deficits, hearing and vision problems, and seizures, all of which Gregory has or had).

The undersigned does not see the basis for Dr. Duchowny's testimony that without DPT, Gregory's deficits would have been milder. Gregory declined neurologically over time and not suddenly except for the frank neonatal seizures he experienced in conjunction with his vaccination. His seizures were brought under control with Phenobarbital and there is no credible evidence that they did lasting harm to his brain. Moreover, he became seizure-free and went off anti-convulsants within five months. Dr. Duchowny stated it is difficult at Gregory's current age to determine what cognitive defects he will have over time, but he is severely handicapped. It is unclear to the undersigned, considering Gregory's prematurity and extensive PVL, how his deficits could have been milder absent DPT.

Petitioners allege in their petition that DPT caused Gregory on-Table anaphylaxis and encephalopathy. Clearly, the medical records state he had anaphylaxis. But the effects of his anaphylaxis did not persist for more than six months. Therefore, petitioners have not made a prima facie case of on-Table anaphylaxis.

As for encephalopathy, Gregory was born with a static encephalopathy manifested by an abnormal brain due to his prematurity. He had the structural abnormalities of a thin corpus callosum and PVL. He manifested myoclonus and mild hypertonicity before vaccination. Petitioners can prevail in the area of encephalopathy only if DPT significantly aggravated Gregory's preexisting encephalopathy. Whitecotton v. Secretary, HHS, 81 F.3d 1099 (Fed. Cir. 1996). They have satisfied one of the requirements that the Federal Circuit set down, i.e., Gregory's current status is significantly worse than his pre-vaccination status, at least in terms of the clinical manifestations of his prematurity.

The statute defines significant aggravation as "any change for the worse in a preexisting condition which results in markedly greater disability, pain, or illness accompanied by substantial deterioration of health." 42 U.S.C. § 300aa-33(4).

Gregory saw four doctors within days of his DPT reaction, and none recorded any acute encephalopathic symptoms: Dr. Liu (March 2nd), Dr. Morales (March 3rd), Dr. Caangay (March 4th), and Dr. Faisal (March 10th). Gregory did not have acute encephalopathy after DPT. Gregory's sudden change for the worse after his DPT vaccination was transient, and did not result in "markedly greater disability, pain, or illness accompanied by substantial deterioration of health." Id. He recovered from his apneic episode (or anaphylaxis) and his seizures were treated and disappeared.

The undersigned holds that there is no causal nexus between his anaphylaxis and neonatal seizures at the time of vaccination and his current condition, and thus petitioners have not made a prima facie case of on-Table significant aggravation of his preexisting encephalopathy. See Hossack v. Secretary, HHS, 32 Fed. Cl. 769 (1995); Song v. Secretary, HHS, 31 Fed. Cl. 61 (1994), aff'd without opinion, 41 F.3d 1520 (Fed. Cir. 1994); contra Gruber v. Secretary, HHS, No. 95-34V,

1998 WL 928423 (Fed. Cl. Spec. Mstr. Dec. 22, 1998) (causal nexus not requisite for significant aggravation).

CONCLUSION

This case is dismissed with prejudice for failure to prove a prima facie case. 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.

The parties can expedite the entry of judgment by jointly filing a notice not to seek review. After judgment is entered, petitioner may expedite payment by filing immediately an election to accept judgment.

IT IS SO ORDERED.

DATE_____ _____

Laura D. Millman
Special Master