OFFICE OF SPECIAL MASTERS

No. 97-518V

(Filed: January 19, 2000)

JOHN HENRY HERKERT, by HANS J. HERKERT, Father and Next Friend,	*	
Petitioner,	*	**TO BE PUBLISHED**
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Daniel P. Donnelly, Garrison, NY, for petitioner.
Claudia B. Gangi, Washington, DC, for respondent.

DECISION

MILLMAN, Special Master

On July 31, 1997, petitioner filed a petition on behalf of his son, John Henry Herkert (hereinafter, "John Henry"), for compensation under the National Childhood Vaccine Injury Act of 1986[1] (hereinafter the "Vaccine Act" or the "Act"). Petitioner has satisfied the requirements

[1] The National Vaccine Injury Compensation Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, 42 U.S.C.A. §300aa-1 et seq. (West 1991), as amended by Title II of the Health Information, Health Promotion, and Vaccine Injury Compensation

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

Petitioners allege that DPaT was a substantial factor in John Henry's contraction of acute transverse myelitis (TM) by immunomodulation, causing the cytomegalovirus (CMV) from which he had been recovering to become newly virulent. Respondent concedes that John Henry had TM but states that CMV was the sole cause of it.

The court held a hearing in this case on September 24, 1999. Testifying for petitioner were Dr. Kevin C. Geraghty and Dr. Ronald Gabriel. Testifying for respondent was Dr. Gerald V. Raymond.

FACTS

John Henry was born on February 6, 1993. Med. recs. at Ex. 1, p. 1. He received his first DPT vaccination on April 8, 1993 at the age of two months. Med. recs. at Ex. 3, p. 1. He received his second DPT vaccination on June 3, 1993 at the age of four months. Med. recs. at Ex. 3, p. 2. Mrs. Herkert called the doctor's office and the nurse noted that John Henry was crying the same day as the vaccination and was difficult to console. The nurse told her to give him Tylenol and to call in one hour. Mrs. Herkert later reported everything was all right. Med. recs. at Ex. 3, p. 10.

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.

John Henry received his third DPT vaccination on August 7, 1993 at the age of six months. Med. recs. at Ex. 3, p. 2. He received his fourth DPT (but actually his first acellular DPT) on August 4, 1994 at 7:00 p.m. at the age of 18 months. Med. recs. at Ex. 3, pp. 3, 7.

During John Henry's examination August 4, 1994, Dr. Peter Gergely put John Henry on his knee to check his back and spine for any deformity. During the entire examination, John Henry was crying and fighting. Then, suddenly, he became very quiet for approximately three to five seconds. In addition, he went completely limp for about three to five seconds while lying over the doctor's knee with his back flexed and his head down. When John Henry's mother asked if he were all right, the doctor lifted him up. John Henry responded immediately by crying and fighting again. John Henry then walked by himself to his mother and climbed onto her lap. He appeared completely alert and back to his normal self. Med. recs. at Ex. 3, p. 16.

John Henry returned to Dr. Gergely the next day, August 5, 1994, at 9:30 a.m., with his father who stated that John Henry had been very listless since the prior evening and thrown up once. John Henry was afebrile and had one intense crying episode before the onset of his listlessness. He was cold and clammy with pooling of blood in his hands. His deep tendon reflexes were depressed. Dr. Gergely thought he had encephalitis. Med. recs. at Ex. 3, p. 15.

John Henry was taken to the Julia L. Butterfield Memorial Hospital Emergency Room on August 5, 1994. The history given was that he had been lethargic since 8:00 a.m. that day. His diaper was wet that morning. A rash was noted on his palms and hands and John Henry had a five-second episode of limpness. At 11:30 a.m., he was limp, lethargic, cried weakly, and had poor muscle tone. The Emergency Room put warming blankets around him, and elevated his legs. His temperature was dropping. Toxicology results were negative. Med. recs. at Ex. 6, p. 1.

The history given was that shortly after his vaccinations the prior day with acellular DPT and OPV, he became lethargic and refused to walk. Med. recs. at Ex. 6, p. 4. He awakened that day limp, lethargic, and barely responsive. Id. He had a rash on his hands and feet, no reflexes, no response to pain, and was barely responsive at all. Id.

John Henry became hypothermic, with his temperature dropping to 94.5 degrees. His blood sugar rose to 260, and subsequently dropped to 128. Dr. Gergely opined John Henry had either encephalitis or intracranial hemorrhage. Med. recs. at Ex. 6, p. 6.

A spinal tap showed that John Henry had a protein of 127 (normal is from 12 to 60) on August 5, 1994. Med. recs. at Ex. 6, p. 13. John Henry was transferred to Columbia Presbyterian Medical Center on August 5, 1994, where he remained until August 22, 1994 when he was transferred to Blythedale Children's Hospital. Med. recs. at Ex. 7, p. 1. The diagnosis was TM, quadriplegia, paralysis of the chest wall muscles, pneumonia, and respiratory distress. Med. recs. at Ex. 7, pp. 1-2.

John Henry's tests for Epstein Barr virus and varicella IgG were negative. Med. recs. at Ex. 8, p. 3. About one week prior to admission, John Henry had had an upper respiratory infection. Med. recs. at Ex. 8, p. 9. On August 5, 1994, at Columbia Presbyterian Hospital, John Henry was pale, ill-appearing, lethargic, and minimally responsive to pain. Med. recs. at Ex. 7, p. 1. When he had been at his doctor's office, he was shocky with a blood pressure of 54 over 20, a pulse of 110, and a temperature of 98.2 degrees. He was barely responsive, photophobic, areflexic, unresponsive to pain, and became hypothermic to 94 degrees but improved. His CSF protein was 127. Med. recs. at Ex. 7, p. 3. His diagnosis was myelitis of uncertain etiology. The doctor thought that immunizations and/or mechanical insult to the cord may have triggered an

inflammation which was now clearly severely diffuse in the spinal cord. The doctor could not exclude the role of infection but, given the lack of symptoms even 36 hours previously, infection seemed less likely the cause. Med. recs. at Ex. 7, p. 4.

A consulting dermatologist on August 6, 1994 noted having personally seen a case of TM after DPT and OPV, but did not recall any cutaneous involvement. Med. recs. at Ex. 7, p. 12. A pediatric intensive care unit admission note stated John Henry's primary medical doctor had found him shocky. Med. recs. at Ex. 7, p. 6.

About two weeks prior to admission, John Henry and his parents experienced a flu-like illness with upper respiratory symptoms. These symptoms resolved over a week's time although John Henry was still slightly hoarse on August 4, 1994. Med. recs. at Ex. 7, p. 29.

On August 24, 1994, John Henry had congestion in his lungs. He had a fever of 100 degrees the day before. By August 25, 1994, he had increased respiratory distress. The doctor queried whether he had TM from the DPT vaccination on August 4, 1994. Med. recs. at Ex. 9, p. 1. On August 26, 1993, Dr. Howard A. Zucker noted presumed aspiration pneumonitis. John Henry had hyperreflexia in his lower extremities and hyporeflexia in his upper extremities. Med. recs. at Ex. 9, p. 29. On August 29, 1994, CMV was isolated in John's urine. Med. recs. at Ex. 9, p. 235. On September 2, 1994, he had a tracheostomy. Med. recs. at Ex. 9, p. 257. John was and is a quadriplegic.

Written Submissions

Mr. Herkert and his wife Patricia A. Walker submitted affidavits. Ms. Walker states on July 28, 1997 that after his fourth DTP at approximately 7:00 p.m. on August 4, 1994, John Henry cried intensely for some time and ultimately appeared quite lethargic until he was put to

bed. P. Ex. 4. Mr. Hans J. Herkert states in his affidavit dated July 22, 1997 that he awoke on August 5, 1994, at 8:00 a.m. Usually John Henry was up at that time, wanting to get out of his crib. But that morning, John Henry was not up. Mr. Herkert went to the crib and John Henry was on his back. His color was gray, his eyes half open, and he signed a low moan. There was dried milk vomit caked around his mouth and on his nightshirt. John Henry did not move. Mr. Herkert lifted John Henry out of the crib after calling the pediatrician's office and John Henry was completely limp. His arms and legs hung loosely and he did not support his head. P. Ex. 5.

Ms. Walker submitted a supplemental affidavit, dated August 3, 1999, stating that the week prior to Thursday, August 4, 1994, John Henry had had a cold. His condition had substantially improved by the weekend preceding August 4th. At the doctor's office, John Henry appeared to be fine, but when the doctor came in to start the examination, John Henry started crying. Dr. Gergely told Ms. Walker that John Henry was perfectly normal. When John Henry received the DPaT vaccine, he started to scream. John Henry's parents went home about 7:00 p.m. Ms. Walker put a nature film on the television, John Henry watched, and he became quiet. Mr. Herkert told Ms. Walker that John Henry had not napped that afternoon, which was unusual for him.

During the night, because the power went out, John Henry's parents took him while he was asleep to a lake house. At around 11:00 p.m., they put John Henry to bed. He opened his eyes and said, "Mama." The next morning, at 5:00 a.m., Ms. Walker left for work. P. Ex. 19.

Mr. Herkert submitted a supplemental affidavit, dated August 3, 1999, stating that he had taken care of John Henry on August 4th. John Henry had recovered from a cold he had had the prior week. During August 4th, John Henry played normally and went swimming with his father

at his paternal grandmother's house on the lake. They returned to their house in late afternoon and John Henry seemed normal. During Dr. Gergely's examination of John Henry, Ms. Walker told him that John Henry still had some hoarseness in his voice from his prior cold. The doctor checked John Henry's ears, nose, throat, and chest and said everything was okay. During this time, John Henry was crying.

After coming home from the doctor, John Henry seemed very tired, occasionally sobbing. He did not finish his bottle and his parents put him to bed. They went to the lake house after the power outage. The next morning, at about 8:00 a.m., Mr. Herkert woke up and went to the crib. John Henry was gray. He had dried, caked vomit around his mouth and on his nightshirt. His eyes were half open and he sighed softly. He did not move. When Mr. Herkert lifted John Henry out of the crib, he was completely listless. His arms and legs hung loosely, and he could not hold his head up. P. Ex. 18.

Dr. Peter E. Gergely submitted an affidavit, dated July 19, 1999, stating that during August 4, 1994, John Henry was crying and fighting, becoming very quiet and limp for approximately three to five seconds during the examination of his back. Dr. Gergely lifted him and he responded immediately with normal affect. Dr. Gergely's impression at the time was that this episode was related to John Henry's fatigue. P. Ex. 20.

Dr. Mark R. Geier submitted an affidavit, dated July 29, 1997, on behalf of petitioner, stating that John Henry had encephalopathy and TM to which the DPT vaccination contributed significantly. He opined that John Henry's TM probably involved an infection with CMV which the DPT vaccine worsened through an adjuvant effect. P. Ex. 17.

Petitioner submitted 31 medical articles in support of the allegations of his petition. These articles are grouped around various themes: (1) CMV does not cause TM in patients whose immune systems are competent (P. Exs. 23-25); (2) pertussis toxin has immuno-suppressant properties, shown in mouse experiments (P. Exs. 26-32); (3) the effect of the immunosuppressant property of pertussis toxin is to increase vulnerability to viral infection shown in experiments with mice (P. Exs. 33-36); (4) the effect of the immunosuppressant property of pertussis toxin is to increase vascular permeability in brain and spinal cord, leading to myelitis of both, resulting in paralysis, shown in experiments with mice and rats (P. Exs. 37-42); (5) pertussis toxin suppresses the immune system by stimulating lymphocytes (a process called lymphocytosis), resulting in inhibition of the primary antibody response (P. Exs. 43-44); (6) pertussis toxin causes lymphocytosis in people (P. Exs. 45-49); (7) pertussis toxin also suppresses the immune system by inhibiting recirculation of lymphocytes between the bloodstream and lymphoid organs, impairing immune surveillance (P. Exs. 50-51); and (8) pertussis toxin increases the virulence of viral infection in children (P. Exs. 52-53).

Respondent submitted nine articles in support of its defense that CMV was the sole cause of John Henry's TM. The themes of these articles are essentially that CMV can cause TM in previously normal people, that CMV does not need the adjuvant effect of another factor to cause TM, and that sudden onset of TM results in serious sequelae. R. Exs. F-N.

TESTIMONY

Dr. Kevin C. Geraghty testified first for petitioner. Tr. at 11. He is board-certified in pediatrics and in allergy and immunology. *Id.* His opinion is that John Henry had an adverse

reaction to the pertussis component of his fourth DPT, which caused his TM. *Id.* This adverse reaction affected the onset, intensity, and outcome of his TM. Tr. at 12-13.

John Henry was a normal infant. Tr. at 13. He received three prior DPTs without significant adverse effects. *Id.* Within 24 hours of his fourth DPT, he had a hypotonic-hyporesponsive episode (HHE) and TM. *Id.* Prompt medical intervention saved him from death. *Id.* An MRI showed he had TM. *Id.* CMV was the etiologic agent of John Henry's TM. Tr. at 13-14. CMV does not cause such an outcome in a normal child. Tr. at 14.

John Henry's CMV on August 4, 1994 before the vaccination was that it had been successfully resolved. Tr. at 15. Myelitis is an adverse outcome associated with trauma, infection, and vaccines. *Id.* A cellular reaction in the tissues compromises the spinal column. *Id.* CMV has a predilection to invade tissues. *Id.* It is neurotropic, meaning it attacks tissues. Tr. at 15-16. It is an opportunistic infection. Tr. at 16. A normal immune host would deal with it very successfully. *Id.* Only when there is immune compromise does this virus express itself. *Id.* That is the prevalent view in the immunological community. *Id.*

Dr. Geraghty interpreted the limpness John Henry experienced in Dr. Gergely's office on August 4, 1994 as a breath-holding spell which he has seen often. Tr. at 17-18. It is not a manifestation of TM. Tr. at 18-19.

Dr. Geraghty found in the medical literature clinical case reports of CMV causing TM only where vaccines were administered or the child was clearly immunocompromised. Tr. at 19. A number of the exhibits deal, however, with CMV causing TM only in adults, not children. Tr. at 20. A previously immunocompetent child can become immunocompromised. Tr. at 21.

CMV is an immunocompromising factor in itself. Tr. at 22.

In acellular DPT, the pertussis is purified and there is a marked reduction of endotoxin by a thousandfold. Tr. at 26. However, there are other toxins in DPaT besides the trace of endotoxin. Tr. at 26-27. Only pertussis toxin can be considered a toxin in the acellular vaccine. Tr. at 28. Because of the use of acellular DPT, there has been a ninety percent reduction in HHE. Tr. at 29. There is also a decrease in encephalopathy. Tr. at 30.

Dr. Geraghty testified that pertussis toxin induces shock by changing the blood sugar. Tr. at 31. Pertussis toxin also causes vascular permeability. *Id.* The blood vessels then leak, affecting the tissues and causing TM. Tr. at 31-33. As the vaccine ages, the pertussis toxin increases. Tr. at 34. The batch that John Henry received was ten months old. Tr. at 35. It was biologically active and had a biochemical propensity for certain proteins to go back to their original more potent state. Tr. at 39-40. In mice, the toxic component has immunosuppressant properties. Tr. at 44. The hallmark of toxin is rapidity of action. Tr. at 54. Dr. Geraghty testified that if John Henry had not had CMV, he would not have had TM, and if he had not had DPaT, he would not have had TM. Tr. at 38.

Dr. Geraghty stated that John Henry experienced a change in the permeability of his blood vessels over 18 hours, resulting in a change in the swelling of his spinal column, a major compromise of his body. Tr. at 49-50. Shock also made an impact on him after vaccination. Tr. at 50. Endotoxin is promptly absorbed. *Id.* But pertussis toxin has a minimal duration of 8 to 12 hours, and goes to many areas of the body. Tr. at 50-51.

Dr. Geraghty testified that John Henry had clinical findings of immune compromise by DPaT. Tr. at 52. One does not find TM after CMV in an immunologically-normal child. *Id.* John Henry, once he was out of shock, had persistent fever. *Id.* CMV is notorious for prolonged

fever. *Id.* He had 102 to 105 degrees through his first hospitalization, and at Columbia Presbyterian, evening spikes and fever of unknown origin. Tr. at 53. John Henry had persistent pneumonitis and interstitial to frank pneumonia, also part of his CMV. *Id.* He had a rash, which is more a finding of CMV than immunocompromise. *Id.* He had a cold seven days prior to his visit to Dr. Gergely on August 4, 1994. Tr. at 54. CMV incubation varies wildly. *Id.*

Animal experiments establish that DPaT vaccine causes vascular permeability. Tr. at 55-56. Pertussis toxin ties up the lymphocytic response which otherwise would have assisted in recovery because lymphocytes make antibodies. Tr. at 57-58. The G receptor in pertussis toxin changed John Henry's vascular permeability, permitting lymphocytes to enter the spinal cord and release cytokines, increasing the swelling. Tr. at 59-60.

The mechanisms of DPaT vaccine's effect on John Henry were: (1) suppression of his immune system; (2) increased vascular permeability; and (3) inhibition of antibody response. Tr. at 85-86. One can expect to see HHE and acute encephalopathy associated with pertussis toxin. Tr. at 86. They are marked by change in behavior. *Id.* HHE is shock-like and more dramatic. *Id.* It arose earlier due to endotoxin in the vaccine. Tr. at 87. Pertussis toxin has later effects. *Id.* Dr. Geraghty cannot separate John Henry's shock from his TM. *Id.*

Dr. Geraghty admitted that acellular DPT was developed because it causes fewer reactions including death and permanent brain damage. Tr. at 92. The offending agent is reduced one thousandfold. Tr. at 93. Pertussis toxin is in acellular DPT, but reduced 50 to 80 percent in active toxin. Tr. at 107. Dr. Geraghty stated one should not immunize a child within thirty days of his having a virus. Tr. at 109.

If John Henry had not had a DPT vaccination, he would have recovered from the CMV based on epidemiology. Tr. at 114. Instead, his cold came back to life, and he developed interstitial pneumonia and fever. Tr. at 118. He was an immunocompromised host. Tr. at 118-19. Normally CMV does not wax and wane. Tr. at 120. It is monophasic and goes away. *Id.*

Dr. Geraghty is unaware of any biological markers for immunosuppression. Tr. at 122. John Henry's spinal cord had a permeability event in the blood vessels. Tr. at 126. Spinal edema is always the result of vascular permeability. Tr. at 126-27. Dr. Geraghty does not deal with spinal cord injuries in his medical practice. Tr. at 127. Pertussis toxin opened up the channels and fluid entered. Tr. at 128. Many of John Henry's treating doctors, in considering causation, focused on his vaccination. Tr. at 132. His dermatologist previously saw one case like his after DPT vaccination. *Id.* DPaT does have biologically reactive pertussis toxin and can enhance viral virulence. Tr. at 133. Dr. Geraghty has not treated a child with TM. Tr. at 55.

Dr. Ronald Gabriel, a pediatric neurologist, testified next for petitioner. Tr. at 64. He interpreted the August 4, 1994 episode in Dr. Gergely's office (when John Henry became limp) as benign. Tr. at 66-67. It was a very brief period of apnea and hypotonia due to breath holding. Tr. at 67. John Henry was on Dr. Gergely's knee, which compressed his diaphragm causing it to contract. *Id.* The abrupt onset and cessation are typical of a compressed diaphragm. Tr. at 67-68. This could not have been a neurologic event (i.e., the beginning of TM) because John Henry rapidly recovered. Tr. at 68. In children up to the age of two and one-half years, breath-holding is one of the most common events. *Id.*

Dr. Gabriel testified that DPaT caused John Henry's HHE. Tr. at 68-69. His shock was possibly but unlikely due to TM because systemic shock does not normally accompany TM. Tr.

at 69. The text (Adams and Victor) to which respondent's expert, Dr. Raymond, refers in his report relates to traumatic cord transection, where systemic shock is common. *Id.* But one does not see this in infection or ischemia of the spinal cord. Tr. at 70. HHE can occur from hours to two to three days after vaccination. Tr. at 74.

Some time in the evening of his vaccination, John Henry had encephalopathy, which was the early sign of inflammation. Tr. at 75. Inflammation proceeds at a slower pace than traumatic injury to the spinal cord. Tr. at 72-73. John Henry's CMV was localized in his throat. Tr. at 76. The effect of the DPaT's immunosuppression was to spread CMV to the spinal cord and the foramen magnum (the base of skull). *Id.* The brain medulla is connected to the spinal cord. The foramen magnum is that part of the skull base through which the connections from the medulla to the spinal cord run. Tr. at 76.

Dr. Gabriel testified that he sees a fair number of TM patients. Tr at 78-79. It is rare to see TM in a child who is not immunosuppressed. Tr. at 79. The lack of recovery in John Henry is to be expected because of the TM's sudden onset. *Id.* A number of doctors at Columbia Presbyterian thought the vaccination contributed to John Henry's TM, which impressed Dr. Gabriel. Tr. at 79-80. In their minds as well as in his, the vaccine contributed to the acute onset, resulting in a far worse outcome from the vaccine. Tr. at 80.

Dr. Gabriel stated he was surprised that John Henry's vaccine was acellular. Tr. at 82. But he opined that he should not be surprised about anything relating to the immune system. *Id*.

Dr. Gerald Raymond, a pediatric neurologist, testified for respondent. Tr. at 138. He is also board-certified in clinical genetics, and treats patients with acute TM and with CMV. Tr. at

139. His opinion is that CMV caused John Henry's course and outcome and DPaT had no effect. Tr. at 140-41.

Dr. Raymond testified that John Henry had typical acute TM. Tr. at 141. It occurs in children and is caused by viruses as well as bacteria, other inflammatory diseases, and granuloma diseases. *Id.* There is medical literature linking vaccinations with TM, but they are the older vaccines for smallpox and rabies as well as for MMR and DPT, but it is never clear that the vaccine is the cause rather than just a coincidence. Tr. at 141-42. Most of the literature petitioner submitted deals with whole cell DPT vaccine, the immune effects of bordetella pertussis and pertussis toxin, and the injecting of high dosages of pertussis toxin into animals. *Id.* Lederle's HSF test results further confirm that DPaT contains much lower pertussis toxin thatn whole cell vaccine. Tr. at 143. The HSF test measures toxicity of pertussis toxin. Tr. at 146.

Dr. Raymond testified that CMV was the sole cause of John Henry's acute TM. Tr. at 147. His persistent fever is completely consistent with CMV. Tr. at 148. Dr. Raymond testified that CMV does wax and wane. Tr. at 149, 188. John Henry had a CMV infection, but the persistent fever showed it was not resolving. Tr. at 150. John Henry's limp episode on August 4, 1994 at the doctor's office does not affect his opinion, and he does not know its significance (a change in opinion from his written report in which he opined the limp episode was part of John Henry's TM, which if true, would mean the onset preceded the DPaT vaccination). *Id.* Dr. Raymond agrees that John Henry was in shock when he reached the doctor's office the next day because his blood pressure was 50 over 20. Tr. at 156.

John Henry's hypotension was secondary to dysautonomia (failure to respond autonomically). Tr. at 153. Dr. Raymond thinks that John Henry had a component of encephalopathy, but he cannot discern between John Henry's hypotension and the possibility that CMV was giving him some mild meningeal encephalitis affecting his brain to some extent. Tr. at 159. Dr. Raymond has seen one and one-half-year-old children with TM due to CMV. Tr. at 160. On August 5, 1994, John Henry had a white blood count of 14,900, showing a mild to moderate elevation. Tr. at 161. His cerebrospinal fluid (CSF) showed 13 percent lymphocytes, showing that he was mounting an inflammatory response. *Id.* His protein count was 127. *Id.* There were 55 white blood cells. *Id.* Normally there are no cells in the CSF. *Id.* He had acute inflammation of the meninges, or myelitis. Tr. at 162. He had complete inflammation of the spinal cord. *Id.*

Dr. Raymond testified that there is no indication that John Henry was immunocompromised. *Id.* He was mounting an immune reaction and having a fever. *Id.* Dr. Raymond queried how John Henry could be immunosuppressed when the DPaT injection was in his arm but there was no overwhelming infection in parts of his body other than his spinal cord. Tr. at 162-63. Immunosuppression takes time. Tr. at 163-64. A lymphocyte reaction takes days, too. Tr. at 164-65. Dr. Raymond cautioned that he is not an immunologist. Tr. at 165.

Dr. Raymond could not understand how if the cause arose from the deltoid (the place of vaccination), the locus of the inflammation would be the spinal cord. Tr. at 166-67. But he admitted that in non-allergic or non-immunologic causes of TM, he did not understand all the precipitants or why the sensitivity is at the spinal cord. Tr. at 167.

In bone marrow transplantation, immunosuppression takes five to six days. Tr at 168. This was the difference between an allergic reaction and immunosuppression. Tr. at 169. Dr. Geraghty then opined that John Henry was not totally bodily immunosuppressed, but rather immunomodulated. Tr. at 169-70. John Henry did not have an allergic reaction. Tr. at 169-70. Dr. Raymond responded that immunomodulation still takes time. Tr. at 171. Dr. Geraghty replied that the time necessary depends on the type of immunomodulation. Tr. at 172.

Dr. Raymond stated that he did not have the knowledge to answer whether a vaccine presents an antigenic challenge to a recipient of it who is already fighting an unrelated virus. Tr. at 175-76.

CMV affects a variety of tissues and can affect the immune system. Tr. at 176. There is no way to tell if John Henry were reacting to the DPaT rather than to the CMV, according to Dr. Raymond. Tr. at 176-77. Dr. Rraymond testified that if John Henry had not received DPaT, he still would have had TM. Tr. at 177. Acellular DPT can cause reactions such as arm swelling, fussiness, irritability, HHE, neuropathies, and encephalopathy. Tr. at 178-79. Dr. Raymond testified he does not know if that is due to an immunosuppressive effect or not. Tr. at 197. Dr. Raymond testified that it has never been shown that even whole cell DPT causes TM. Tr. at 179. The supposition is that the "bad actor" is pertussis toxin, but endotoxin could be as well. Tr. at 180. Even though the amounts of endotoxin and pertussis toxin are reduced in DPaT, these toxins can still be bad actors in particular vaccinees who receive DPaT. *Id*. However, Dr. Raymond does not believe that DPaT could cause TM in hours. Tr. at 181-82.

Dr. Raymond admitted that one can remove the toxic effect of acellular DPT and it would still be immunogenic. Tr. at 184-85. He believes that John Henry's CMV was not resolving

when he received DPaT. Tr. at 188. The medical literature on TM secondary to CMV deals exclusively with adults. Tr. at 204. There were two papers that petitioner submitted dealing with severe neurologic consequences in children secondary to CMV who had pertussis or received DPT vaccine. Tr. at 206-09.

DISCUSSION

Petitioner is proceeding on a theory of causation in fact. To satisfy his burden of proving causation in fact, petitioner must offer "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury. A reputable medical or scientific explanation must support this logical sequence of cause and effect." Grant v. Secretary, HHS, 956 F.2d 1144, 1148 (Fed. Cir. 1992). Agarwsal v. Secretary, HHS, 33 Fed. Cl. 482, 487 (1995); see also Knudsen v. Secretary, HHS, 35 F.3d 543, 548 (Fed. Cir. 1994); Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579 (1993).

Without more, "evidence showing an absence of other causes does not meet petitioners' affirmative duty to show actual or legal causation." Grant, supra, 956 F.2d at 1149.

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

In essence, the special master is looking for a reputable medical explanation of a logical sequence of cause and effect (Grant, supra, 956 F.2d at 1148), and medical probability rather than certainty (Knudsen, supra, 35 F.3d at 548-49). To the undersigned, medical probability means biologic credibility or plausibility rather than exact biologic mechanism. As the Federal Circuit stated in Knudsen:

> Furthermore, to require identification and proof of specific biological mechanisms would be inconsistent with the purpose and nature of the vaccine compensation program. The Vaccine Act does not contemplate full blown tort litigation in the Court of Federal Claims. The Vaccine Act established a federal "compensation program" under which awards are to be "made to vaccine-injured persons quickly, easily, and with certainty and generosity." House Report 99-908, *supra*, at 3, 1986 U.S.C.C.A.N. at 6344.
>
> The Court of Federal Claims is therefore not to be seen as a vehicle for ascertaining precisely how and why DTP and other vaccines sometimes destroy the health and lives of certain children while safely immunizing most others.

35 F.3d at 549.

Although the United States Supreme Court in Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579 (1993), listed various criteria for the federal district court judges to follow in their role as gatekeeper for the admission of scientific and medical evidence, such criteria are merely aides in evaluation, rather than prescriptions, for the Office of Special Masters. Even in federal district courts, "Daubert's list of specific factors neither necessarily nor exclusively applies . . . in every case . . . [and its] list of factors was meant to be helpful, not definitive." Kumho Tire Co., Ltd. v. Carmichael, 526 U.S. 137, ___, 119 S. Ct. 1167, 1171, 1175 (1999).

In the Office of Special Masters, even the Federal Rules of Evidence are not required.[2] Invariably, consistent with the legislative intent in creating the Vaccine Program, the special masters admit most evidence. But see, Domeny v. Secretary, HHS, No. 94-1086V, 1999 WL 199059 (Fed. Cl. Spec. Mstr. March 15, 1999), aff'd, (Fed. Cl. May 25, 1999) (unpublished), on appeal, No. 99-5130 (Fed. Cir.) (proffer of dentist's testimony for diagnosis of a neuropathy rejected).

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[2] CFC Rules, Vaccine Rule 8(b) Evidence. "In receiving evidence, the special master will not be bound by common law or statutory rules of evidence. The special master will consider all relevant, reliable evidence, governed by principles of fundamental fairness to both parties."

As the Federal Circuit stated in Knudsen, supra, 35 F.3d at 548, "Causation in fact under the Vaccine Act is thus based on the circumstances of the particular case, having no hard and fast *per se* scientific or medical rules." Thus, the task before the undersigned is not to delineate how petitioner's evidence of immunomodulation does or does not satisfy the Daubert litany of support in peer-reviewed medical literature, concurrence among a majority of physicians in the field of immunology and/or neurology, and confirmative testing of methodology. Rather, the task is to determine medical probability based on the evidence before the undersigned in this particular case.

Petitioner asserts that John Henry's acellular DPT immunomodulated his ability to fight CMV, which had practically resolved, enabling the CMV to become virulent again, leading to his contracting TM after transient HHE and encephalopathy. The effect of the vaccine was to increase John Henry's vascular permeability, leading to spinal edema due to the opportunistic infection of CMV. Thus, DPaT was a substantial factor in John Henry's contraction of TM.

Petitioner's expert Dr. Geraghty, buttressed by the testimony of Dr. Gabriel and the affidavit of Dr. Geier (as well as several opinions in the medical records of John Henry's treating doctors), opined that regardless of the diminution of the toxins (endotoxin and pertussis toxin) in acellular DPT, it still has an immunogenic effect which, on rare occasions, can result in immunomodulation in a host who is fighting off CMV. The court is impressed with Dr. Geraghty's credentials as an immunologist, plus the fact that his reasoning makes good sense in the context of the events of this case. The suddenness of John Henry's decline post-vaccination from a normal, combative toddler to a dying child afflicted with HHE, encephalopathy, and

subsequently quadriplegia, all in the space of one day, fits well into the theory of cause and effect that Dr. Geraghty, buttressed by Drs. Gabriel and Geier, has given.

Respondent's expert Dr. Raymond could not distinguish whether the vaccine or the CMV caused John Henry's HHE and acute encephalopathy within hours of his vaccination. He said there was no way to determine if John Henry were reacting to his vaccination rather than to the CMV, and admitted that acellular DPT can cause reactions, such as arm swelling, fussiness, irritability, HHE, neuropathies, and encephalopathy. When pressed about immunologic theory, he responded that he was not an immunologist. When confronted with a medical article pertaining to a child with severe neurologic injury secondary to CMV who had received DPT, he opined that there was no way to determine if the DPT had an immunosuppressant effect. He also could not answer if the acellular vaccine, when injected into a host who is already fighting a virus, will antigenically challenge the host.

Examining the facts and weighing the testimony, the undersigned notes that John Henry had recently and successfully recovered from a cold, with only a slight symptom of hoarseness, when he received his acellular DPT vaccination. The evening after vaccination, he was not well. By the next morning, he was in effect dying. He was encephalopathic, in shock, and limp. The hospital cultured CMV, yet in the three hospitals in which he was a patient, doctors queried whether the vaccination had been responsible for his condition. He also had persistent fevers and rashes on his palms, hallmarks of CMV infection together with the viral cultures.

Dr. Geraghty posited, as an immunologist, that while John Henry successfully fought off the CMV, hence his clinical recovery, acellular DPT challenged his body immunologically, making him less capable to withstand the effect of the CMV, particularly as his blood vessels

became more permeable, and he developed a lesion along his cervical spine, rendering him a quadriplegic. Thus, the vaccine was a substantial factor in John Henry's contraction of TM.

Dr. Raymond could not distinguish between the DPaT or the CMV as the cause of John Henry's initial symptoms of HHE and encephalopathy. Even though he attributes John Henry's TM solely to his CMV, Dr. Raymond believes the vaccine played a factor in John Henry's illness. Dr. Raymond's reluctance to enter the immunologic field in discussing the effects of vaccination and the concept of immunomodulation is understandable (and laudable) since he is not an immunologist. But, petitioner's experts were more persuasive in explaining the cause and effect of John Henry's condition through the immunomodulation of the antigen he received while recovering from CMV. DPaT was a substantial factor in John Henry's contraction of TM.

As in Shyface, supra, there are two substantial factors in this case: the first is the vaccine and the second is the infection. In Shyface, the vaccinee suffered an extremely high fever due to receipt of DPT while at the same time having an E. coli infection. Testimony showed that the infection was not at a sufficient level to have alone caused such a high fever, which led to Cheyenne Shyface's encephalopathy and death. The Federal Circuit held that because the vaccine played a substantial factor in Cheyenne Shyface's encephalopathy and death, petitioners must prevail. Because the special master had held that the two factors were in equipoise, the Federal Circuit did not hold that either the vaccine or the infection was the predominant factor. But, legally, the vaccine's being a substantial factor is sufficient to entitle petitioners to compensation. 165 F.3d at 1353.

In the instant action, John Henry had almost successfully fought off the CMV when he received DPaT vaccine, which caused HHE, encephalopathy, and vascular permeability leading

to spinal edema. As Dr. Geraghty testified, had he not been exposed to a further immunologic challenge from the vaccination, John Henry would have successfully fought off the remains of the CMV without succumbing to TM. The vaccine here, as in Shyface, was a substantial factor in causing his TM from which he still suffers today.

It may be argued that TM cases have not prevailed in this court. But, there are cases dealing with other vaccines in other courts in which TM plaintiffs have prevailed: Toner v. Lederle Laboratories, a Division of American Cyanamid Co., 828 F.2d 510, modified, 831 F.2d 180 (9th Cir. 1987), cert. denied sub nom., Lederle Laboratories, Division of American Cyanamid Co. v. Toner, 485 U.S. 942 (1988) (vaccine manufacturer's negligence proximately caused infant's TM); Unthank v. U.S., 732 F.2d 1517 (10th Cir. 1984) (swine flu vaccination caused adult's TM); Guillory v. St. Jude Medical Center, 675 So.2d 1198 (5th Cir. Ct. App. LA 1996) (amended to increase attorney's fees and affirmed workers' compensation decision that hepatitis B vaccine triggered adult TM); cf. Wyeth Laboratories, Inc. v. Fortenberry, 530 So.2d 688 (Sup. Ct. MI 1988) (decision against vaccine manufacturer in adult TM reversed because package warning was adequate)

The issue here, unlike the cases supra, is not whether the vaccine alone caused John Henry's TM, but whether it was a substantial factor in causing his TM. Dr. Geraghty's testimony about the effect of the vaccine on John Henry's already-burdened immune system is medically probable, being a logical sequence of cause and effect based on a reputable immunological explanation.

Dr. Geraghty admitted that acellular DPT reduces reactogenicity by a hundredfold, the very reason for its development and use. But, as Dr. Geraghty also stated, it still contains

pertussis toxin which can have harmful effects. (Dr. Raymond called pertussis toxin the "bad actor.") But that there should be fewer reactions does not mean that there are no reactions. Cf. Knudsen, supra, in which the Federal Circuit stated that the fact that viral infections more often cause encephalopathies than do vaccines was not proof in an individual case that a virus and not the vaccine was the cause of encephalopathy:

> The bare statistical fact that there are more reported cases of viral encephalopathies than there are reported cases of DTP encephalopathies is not evidence that in a particular case an encephalopathy following a DTP vaccination was in fact caused by a viral infection present in the child and not caused by the DTP vaccine.

35 F.3d at 550.

The bare statistical fact that the incidence of reactions following acellular pertussis vaccination is reduced one hundredfold and that TM is a rare but accepted complication of CMV is not evidence that, in a particular case, TM following a DPaT vaccination was in fact caused solely by the CMV and not substantially by the DPaT vaccine.

Petitioner's evidence meets the requirement the Federal Circuit enunciated of showing a logical sequence of cause and effect based on reputable medical opinion. That Dr. Geraghty is an immunologist assists the court in understanding what is after all an immunologic explanation supporting the conclusion that petitioner's theory of causation is medically probable.

Petitioner has proved a prima facie case of causation in fact.

CONCLUSION

Petitioner is entitled to reasonable compensation. The undersigned hopes that the parties may reach an amicable settlement, and will convene a telephonic status conference soon to

discuss the filing of life care plans, unless the parties agree on a joint life care plan. Should the parties not be able to settle this case, the undersigned will hold a damages hearing.

IT IS SO ORDERED.

_____ _____

DATE Laura D. Millman
Special Master