OFFICE OF SPECIAL MASTERS

December 18, 2002

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| TAYLOR MORRIS, by her parents and natural guardians, SONYA and DAVID MORRIS, | * | |
| Petitioners, | * | **No. 99-412V** **PUBLISHED** |
| v. | * | |
| SECRETARY OF THE DEPARTMENT OF HEALTH AND HUMAN SERVICES, | * | |
| Respondent. | * | |

Clifford J. Shoemaker, Vienna, VA, for petitioners.
Joan E. Coleman, Washington, DC, for respondent.

## DECISION

**MILLMAN, Special Master**

On June 28. 1999, petitioners filed a petition on behalf of their daughter, Taylor Morris (hereinafter, "Taylor"), under the National Childhood Vaccine Injury Act of 1986[1] (hereinafter the "Vaccine Act" or the "Act"). Petitioners have satisfied the requirements for a prima facie case pursuant to 42 U.S.C. § 300aa-11(c) by showing that: (1) they have not previously collected an

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

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

Petitioners allege that OPV vaccine caused Taylor's transverse myelitis (TM). Respondent denies causation from the vaccine.

The court held a hearing in this case on November 29, 2001. Testifying for petitioners were David H. Morris, Sonya Morris, and Dr. Carlo Tornatore. Testifying for respondent was Dr. Gerald V. Raymond.

Because petitioners did not file Taylor's most current medical records prior to the hearing, the court issued an Order, dated November 30, 2001, ordering petitioners to file all current records from Wake Forest Medical Center, originals of the 2000 MRIs performed on Taylor, an affidavit from Patty Wagener, Taylor's babysitter on August 8, 1996, describing Taylor's condition on August 8, 1996, and the photograph Mrs. Morris took of Taylor on or about August 10, 1986. The court also ordered petitioners to file a medical article and a medical text chapter (P Exs. 14 and 15), and set a status conference for January 30, 2002.

Petitioners filed their affidavits on December 3, 2001, stating that Taylor seemed fine when she went to the babysitter on August 8, 1996. On January 18, 2002, petitioners filed the medical article and chapter previously ordered, a letter stating Taylor has a medical exemption from receiving MMR vaccine, and an affidavit from the babysitter, Patricia Wagener, that Taylor became sick after she had entered Ms. Wagener's home on August 8, 1996. Ms. Wagener put Taylor down for a nap at 11:00 a.m. and later heard her "breathing funny." She went and got her, brought her downstairs, and telephoned Mr. Morris. Taylor became sick very quickly and Ms. Wagener stated she did not know what was wrong. Mr. Morris arrived 15 minutes later and Taylor was so sick that she was

limp in Ms. Wagener's arms and could not raise her head. It happened so fast that it scared Ms. Wagener who was concerned for Taylor's life. P. Ex. 16. Petitioners also filed a baby picture of Taylor at 4 months of age. P. Ex. 17.

After the status conference on January 30, 2002, the undersigned issued an Order, dated January 31, 2002, for petitioners to file all current records from Wake Forest Medical Center, and for respondent to file Dr. Raymond's supplemental medical report dealing after reviewing Taylor's MRIs and the Wake Forest medical records. A status conference was set for March 19, 2002.

On February 8, 2002, petitioners filed medical records from Wake Forest. The status conference scheduled for March 19, 2002 was rescheduled for April 2, 2002. This conference was then rescheduled for April 10, 2002. Respondent orally moved for an extension of time until April 17, 2002, which was granted, and the status conference was set for April 17, 2002. On April 17, 2002, petitioners filed Dr. Tornatore's revised curriculum vitae. Also on April 17, 2002, respondent filed Dr. Raymond's supplemental report. R. Ex. U.

At the status conference on April 17, 2002, the undersigned ordered that Dr. Tornatore's response to Dr. Raymond's supplemental report be filed no later than May 17, 2002, and set a status conference for May 24, 2002. Petitioners did not file Dr. Tornatore's supplemental report for six months (on October 11, 2002) due to a medical problem in Dr. Tornatore's family. P. Ex. 21. Respondent filed Dr. Raymond's response to Dr. Tornatore's supplemental report on December 10, 2002. R. Ex. V.

## FACTS

Taylor was born on March 10, 1996. She received her second set of DPT, oral polio, and HiB vaccines on July 12, 1996 at the age of 4 months. (Her first set had been administered on May 14, 1996.) Med. recs. at Ex. 3, p. 3.

Twenty-seven days later, on August 8, 1996, she was brought to Rowan Regional Medical Center where physician's assistant Jeff Taylor dictated notes for Dr. L.E. Nickerson, Taylor's pediatrician. He recorded that Taylor had a fever of unknown origin on admission with mild reactive airway disease and air trapping. She had a sudden onset of fever, lethargy, respirations in the 80s, and expiratory wheeze. Cerebrospinal fluid (CSF) cultures were negative at 24 and 48 hours. A chest x-ray showed mild hyperinflation with air trapping. A CT scan of her brain on August 11, 1996 showed slight prominence of her ventricles without acute findings. Med. recs. at Ex. 6, pp. 43, 55.

Taylor was placed in a croup tent and given medications. She showed good, early improvement in her wheezing and expiratory congestion. She had a marked decrease in her respiratory rate. On August 9, 1996, she did not grasp as well and her right pupil was somewhat dilated in comparison with her left. She also had some flaccidity in her left upper and lower extremity, more pronounced in the lower extremity. Med. recs. at Ex. 6, pp. 43-44. They consulted a Children's Clinic which recommended discontinuation of steroids and antihistamine decongestants to see if Taylor improved, but the following morning, she did not improve. An immediate CT scan of her head was ordered, which showed enlarged ventricles, but no acute process. A spinal tap was negative. She was transferred to Baptist on August 12, 1996 to come under the care of Dr. Santos. Med. recs. at Ex. 6, p. 44.

In the history of present illness that Jeff Taylor wrote on August 8, 1996, he recorded as Taylor's chief complaint respiratory distress. The history given was that Mr. and Mrs. Morris brought Taylor into Dr. Nickerson's office with complaints of new onset increasing respiratory distress, tachypnea (quick, shallow breathing), lethargy, and fever of 101°. Taylor was slightly lethargic, but responsive to verbal and noxious stimuli. Her parents denied any nausea, vomiting, diarrhea, rash, mental status changes, or signs or symptoms consistent with a more systemic type of toxicity. Because of Taylor's expiratory wheezing, she was given Albuterol nebulizer. Her temperature at Rowan was 100.8° and her vital signs were stable. She was lethargic but alert and responsive, perfusing without difficulty, and sucking a bottle well.

Her pupils were equal, round, and reactive to light and accommodation. Her anterior fontanelle was flat, not bulging. Her lungs had scattered rhonchi with slight expiratory wheeze and mild retractions. Her extremities had full range of motion. She perfused well. Her neurological examination was grossly intact without any focal or neurologic deficits. The impression was reactive airway disease with exacerbation with air trapping and questionable early organizing pneumonia. Med. recs. at Ex. 6, p. 45. A chest x-ray done on August 8, 1996 showed some mild air trapping. Her spleen might have been slightly prominent in size. Med. recs. at Ex. 6, p. 56.

Dr. Joseph D. Corpening consulted on the case on August 10, 1996. He noted that Taylor had been admitted on August 8, 1996 with a complaint of coughing and fever. She became ill approximately 24 hours prior to that time. She began to have some congestion and coughing, and questionable wheezing. She had fever with irritability. She had been in excellent health, with no serious illnesses or reported drug or food allergies until the onset of her illness. On physical examination, her temperature was 100.4°. She was husky, well-developed, fairy alert, and apparently

non-toxic in appearance. Both her fontanelles were flat. The right pupil appeared slightly more dilated than the left. No wheezes were heard. Abduction of the legs was almost 90°. His impression was fever of unknown cause (acute bronchitis) and observe for possible CNS disease. Med. recs. at Ex. 6, p. 47.

On August 10, 1996, she was breathing better but still had upper airway congestion. The parents reported decreased movement and slight lethargy. She fed well and sucked well. On physical examination, her anterior fontanelle was flat, she had scattered rhonchi. Med. recs. at Ex. 6, p. 62.

On August 11, 1996, Dr. Wayne C. Koontz wrote that Taylor had been taken to her sitter since she was two months old. She went to her sitter with a mild head cold, but she was playful and active. At her sitter, she awoke from her nap with a fever of 101° and was limp. She did not use her left hand well. Her right pupil was bigger than her left. She vomited during the first two days, but not since. She had been alert and eating well. She was alert on physical examination and had a vigorous suck. Med. recs. at Ex. 6, p. 60.

Dr. Koontz wrote on August 12, 1996 that Taylor was at her sitter when she was found limp, febrile (101°), and was later noted to have a dilated right pupil with weakness of her left arm, hand, left foot, and leg. He first saw Taylor on August 11, 1996 and did a CT scan and lumbar puncture, both of which were normal except for slight prominence of her ventricles on CT. Presently, she was happy, afebrile, and eating well. Dr. Koontz thought she might have had a CVA (cerebrovascular accident) and called the neurologist Dr. Santos who felt this was the most likely possibility. He informed the Morrises and Dr. Nickerson of this and the decision was made to transfer her to Dr. Santos' care. Dr. Koontz's impression was to rule out CVA. Med. recs. at Ex. 6, p. 57.

Other notes for August 12, 1996 are that Taylor slept well, and was alert and active with her eyes. Her parents reported increased limb movement, but the doctor perceived little change. On physical examination, her pupils were apparently equal bilaterally that morning, but her fontanelle was slightly bulging compared to August 11th and somewhat tense. Her lungs were clear. He questioned whether she had Todd's paralysis (after epileptic seizure), post-viral encephalitis, or early urinary tract infection. She was out of the croup tent. Med. recs. at Ex. 6, p. 58.

Dr. Koontz ordered an MRI on August 12, 1996 to see if a CVA was present. Taylor's anterior fontanelle was slightly full but not bulging. Her right pupil was still dilated. Her left arm and left leg had minimal movement. Otherwise, she was happy and eating well. Id.

Taylor was hospitalized until August 12, 1996 when she was transferred to Carolinas Medical Center where she remained until August 15, 1996.

At Carolinas Medical Center, on admission to the PICU, she had a left pupil 1 mm. smaller than the right but both were reactive. She had very mild facial droop, and decreased use of her left arm. Her cerebrospinal fluid was negative as was a repeat CT scan. An MRI on August 12, 1996 showed a swollen cervical cord suggesting TM. [Petitioners did not file the written interpretation of the MRI of Taylor's cervical spinal cord.] Med. recs. at Ex. 1, pp. 3, 4.

A consultation report by Dr. Paul D. Knowles, a pediatric neurologist, dated August 12, 1996, states on physical examination, Taylor's neck was supple, her fontanelle was soft, her right pupil was 1.5 cm. larger than her left pupil. She had very mild asymmetry of her face. Her lungs were clear. Neurologically, she had a flaccid left arm. The lower extremities seemed to move symmetrically, but there was a suggestion of some mild decrease in tone bilaterally. She withdrew her lower extremities bilaterally very well. His impression was asymmetric face with upper extremity

flaccidity. He questioned whether Taylor's condition could be attributed to Horner's syndrome[2] with brachial plexopathy versus a brain stem injury. He ordered MRIs of the brain and cervical spine. Med. recs. at Ex. 1, p. 6; Ex. 2, p. 5. Dr. Knowles states in a letter to Dr. Nickerson dated March 7, 1997 that an MRI showed an enlarged cervical cord thought to be consistent with TM. Med. recs. at Ex. 2, p. 3.

On June 19, 1997, Taylor had surgery to insert bilateral myringotomy tubes because of chronic mucoid otitis media. Med. recs. at Ex. 6, p. 37.

On August 1, 1997, Taylor saw Dr. Anthony L. Burke for evaluation of an umbilical hernia[3] which Mrs. Morris noticed when Taylor was 11 months old and thought was increasing in size. On examination, Taylor had pupils which were equal, round, and reactive to light and accommodation. She had an umbilical hernia and rectus diathesis.[4] Her extremities had a full range of motion. Med. recs. at Ex. 6, p. 32. On August 1, 1997, Taylor had surgery to repair her umbilical hernia. Dr. Burke decided that the separation of the rectus muscles was not marked and Taylor probably did not need to have the rectus diathesis repaired. Med. recs. at Ex. 6, p. 34.

On August 7, 1998, Taylor saw a physical therapist at the Shriners Hospital. [Petitioners filed only the first page of that report.] Med. recs. at Ex. 5, p. 3. The history of the present illness was

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[2] Horner's syndrome is "caused by paralysis of the cervical sympathetic nerves." Dorland's Illustrated Medical Dictionary 27th Edition (1988) at 1636.

[3] An umbilical hernia is "protrusion of part of the intestine at the umbilicus, the defect in the abdominal wall and protruding bowel being covered with skin and subcutaneous tissue...." Dorland's, supra, at 758.

[4] "Diathesis" means "a constitution or condition of the body which makes the tissues react in special ways to certain extrinsic stimuli and thus tends to make the person more than usually susceptible to certain diseases." Dorland's, supra, at 465.

that Taylor had a diagnosis of transverse myelitis at the L3 level.[5] She was born two weeks post date by caesarean section for failure to progress. She did well until at about five months of age, she developed a febrile illness, became poorly responsive, and was seen at a local hospital with a temperature of 101°. She was felt to have early pneumonia, was placed on antibiotics and steroid treatment, and had decreased motion in the lower extremities and poor improvement. Upon transfer to Carolinas Medical Center, she received an MRI which led to a diagnosis of TM at the L3 level. Following that, her developmental milestones were somewhat delayed. She has frequent urinary tract infections. Id.

According to the history given on August 25, 2000 to Dr. L. Andrew Koman, an orthopedist, at Wake Forest University Baptist Medical Center, Taylor was normal until she was four months old when she suddenly became lethargic and had fever one week [sic] after her four-month immunizations. She has lower extremity contractures and spasticity, and a neurogenic bladder. P. Ex. 18, p. 9. Dr. Koman's impression was that Taylor's lesion likely localized to the high lumbar region. He did not have films or lumbar puncture results and based his diagnosis on history. P. Ex. 18, p. 12. Taylor was also examined by Dr. Cesar C. Santos, a pediatric neurologist, and his resident, Dr. Tareck A. Kadrie, on August 25, 2000. Taylor's hips had decreased abduction to 35°. Tone was markedly increased in her lower extremities. Id. at 19.

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[5] The L3 reflects the lumbar spine, which is far from the cervical spine. The cervical spine (at the neck) has 7 levels. Then comes the thoracic spine, which has 12 levels. Following the thoracic spine is the lumbar spine with 5 levels. At the bottom of the spine is the sacrum, with five levels, followed by the coccyx. Although the Rowan and Carolinas medical records do not give a level for Taylor's swollen cervical cord, Dr. Tornatore testified that she has a lesion at the C2 or C3 level of her spine. At the very least, the L3 level noted in the 1998 and 2000 records as the level for Taylor's TM is at least 15 levels below the cervical spine noted in the 1996 records, and it is 19 or 20 levels below the level that Dr. Tornatore identified as where Taylor's lesion is. Atlas of Human Anatomy, 2nd Ed., by F.H. Netter (1997), at Plate 142.

On August 30, 2000, Taylor had an MRI of her lumbar spine with and without contrast. The MRI was normal. P. Ex. 18, p. 41.

On September 1, 2000, Taylor had a radiologic procedure on her pelvis which showed pedicular widening at her lower lumbar and sacral vertebrae, consistent with myelomeningocele.[6] P. Ex. 18, pp. 40, 50.

In a supplemental report filed by respondent's expert, Dr. Gerald V. Raymond, a pediatric neurologist, he refers to receiving P. Ex. 12, which are neuroimaging studies, including a brain MRI, magnetic resonance angiography and cervical cord examination from August 12, 1996, and lumbosacral MRI from NC Baptist Hospital from August 30, 2000. These are the actual films, not interpretations of them. R. Ex. U. Petitioners filed the Rowan Memorial Hospital interpretation of the CT scan of Taylor's head dated August 11, 1996 (P. Ex. 6, p. 55) and the Wake Forest written interpretation of Taylor's August 30, 2000 MRI of her lumbar spine (P. Ex. 18, p. 41), but never filed

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[6] "Myelomeningocele" is a "hernial protrusion of the cord and its meninges through a defect in the vertebral canal." Dorland's, supra, at 1088. "Paraplegia from the myelodysplasia typically causes some impairment of mobility along with neurogenic bowel and bladder. Other neurologic deficits may present acutely or chronically at birth or later." "The musculoskeletal deformities that occur are related to the functional level of the lesion. Thoracic and high-lumbar groups tend to have increased incidence of lumbar lordosis, hip abduction and external rotation contractures, knee flexion, and equinus contractures of the ankles. Unopposed hip flexion and adduction contractures in the high-lumbar group frequently result in dislocated hips." "Myelomeningocele is the most common cause of neurogenic bladder dysfunction in children. The nature of the urinary tract dysfunction in myelomeningocele depends on the level and extent of the spinal cord lesion." "Myelomeningocele," by K. Kolaski, MD, http://www.emedicine.com/pmr/topic83.htm. "Protrusion of the spinal cord and meninges damages the spinal cord and nerve roots, causing a decrease or lack of function of body areas controlled at or below the defect. Symptoms are related to the anatomic level of the defect. Most defects occur in the lower lumbar or sacral areas of the back (the lowest areas of the back) because this area is normally the last part of the spine to close. Symptoms include partial or complete paralysis of the legs, with partial or complete lack of sensation, and may include loss of bladder or bowel control." Myelomeningocele (children). http://www.nlm.nih.gov/medlineplus/ency/article/001558.htm.

interpretations of her brain MRI, magnetic resonance angiography and cervical cord MRI done August 12, 1996. Thus, the undersigned has not had any opportunity to compare the treating doctors' interpretations with the interpretations of Dr. Raymond (R. Exs. U, V) and Dr. Tornatore (P. Ex. 21).

**Other Submissions**

Petitioners filed "Acute Poliomyelitis Beginning as Transverse Myelopathy," by K.M. Foley and H.R. Beresford, 30 *Arch Neurol* 182-83 (1974). P. Ex. 13. A 16-year-old boy, who had never received immunizations due to religious convictions, came down with myelopathy during a school outbreak of poliomyelitis in a largely unvaccinated population. He had back pain, leg weakness, and was unable to walk or urinate. He had no recent fever, chills, diarrhea, abdominal pain, neck stiffness, or contact with ill persons. Cultures of spinal fluid, stool, and nasopharyngeal washings demonstrated no growth of virus. However, serial titers indicated recent infection with type I poliovirus. During the 18 days before his hospitalization, ten other students at his school had paralytic illnesses. None of them had received immunizations for religious reasons. Type I poliomyelitis was isolated from nine of these students. Id. at 182. The authors state that the case exemplifies the uncommon presentation of acute poliomyelitis as TM. What made this and other cases of TM in acute poliomyelitis unusual was the sensory loss component. Id. at 183.

Petitioners filed chapter 6, entitled "Poliomyelitis," by R.W. Price and F. Plum, from the second volume of Infections of the Nervous System, edited by P.J. Vinken and G.W. Bruyn (1978), pp. 93-132. P. Ex. 14. On page 106, the authors state, "There is little evidence to suggest that poliovirus can persist for prolonged periods in the nervous system." They continue:

The incubation period (from viral contact to the onset of minor symptoms) is usually 1-3, and rarely more than 4-5 days. ...
The major illness represents an extension of the enteric infection and viremia of the minor illness. The incubation period (including the minor illness, if present) varies from 3 to 35 days but usually lasts 4-10 days. [citation omitted]. ...

The major illness begins with fever, malaise and, within hours, generalized headache and vomiting, followed 12-24 hours later by the development of neck and back stiffness. ...
Children, in general, tend to have less intense systemic symptoms...: the preparalytic phase in children tends to be shorter, and muscle weakness may even coincide with the point when the temperature falls and the patient begins to feel better. ...

Acute transverse myelitis can be confused with poliomyelitis but in the former, a sensory and motor level at the involved spinal cord segment usually serves to separate an inflammatory cord transection from diffuse anterior horn cell involvement.
[In the oral polio vaccine], [t]he attenuated strains possess reduced neurovirulence but retain the ability to infect and replicate in the nasopharynx and lower gastrointestinal tract, thereby inducing both local (secretory) and systemic immunity.

After oral administration, the attenuated virus strains undergo replication in the oropharynx and lower gastrointestinal tract, and excreted virus can be detected in oral secretions or feces in 24-28 hours. As with natural poliovirus infection, fecal excretion may continue for 6-8 weeks. Unlike virulent poliovirus, however, replication of attenuated strains occurs almost entirely within the gastrointestinal and perhaps the lymphatic systems. Viremia does not occur with types 1 and 3 vaccine viruses. After type 2 attenuated virus administration, low levels of virus have been detected in the blood, but this appears to be entirely benign and not associated with CNS invasion. ...
Immunization with inactivated poliovirus has proved to be generally safe, with two notable exceptions. In 1955, soon after the [Salk inactivated] vaccine was licensed, an outbreak of 65 cases of paralytic poliomyelitis occurred within 4 weeks following inoculation of about 400,000 persons [the Cutter incident]. This was traced to a single source of vaccine in which type 1 poliovirus was inadequately inactivated so that the vaccine contained residual amounts of infectious virus.... [citations omitted.] The complication has subsequently been eliminated by more stringent regulation of vaccine production. The second complication was the contamination of vaccine with live SV 40 virus.... [citation omitted.] This simian papovavirus is a natural contaminant of the monkey kidney cells used to produce vaccine and is more resistant to formalin inactivation than poliovirus. Although SV 40 is oncogenic in animals and is one of the two viruses isolated from the brains of patients afflicted with the demyelinating disease, progressive leukoencephalopathy..., present evidence does not implicate vaccination as a cause of cancer or demyelinating disease in man. [citation omitted.]

> The conversion to WI-38 cells from monkey kidney cells in recent years eliminates the hazard of introducing into virus cultures simian viruses such as the SV 40 or simian foamy viruses ... which contaminate monkey cells. [citation omitted.] In addition, administration of vaccine by the oral route rather than by injection means that natural host barriers are still available to prevent systemic infection.
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> **Live-attenuated virus vaccine is ... highly effective in preventing poliomyelitis**.... Unlike virulent poliovirus, ...replication of attenuated strains occurs almost entirely within the gastrointestinal and perhaps the lymphatic systems. **[T]he immune response to a single dose of an attenuated strain is brisk and sustained**. [citation omitted.] **These serum antibodies protect against paralytic poliomyelitis by preventing viremia on subsequent challenge with wild virus**. [T]he presence of circulating antibody alone is adequate to prevent the crucial viremic state.

Pages 108, 109, 114, 121, 123, 124, 125.

Respondent filed Principles of Neurology, 6th ed., by R.D. Adams, M. Victor, and A.H. Ropper (1997). R. Ex. C. On page 1243 of chapter 44, "Diseases of the Spinal Cord," is the statement, "Several partial cases [of TM] have had normal [MRI] studies."

On page 12444, the authors state:

> A pharyngitis, **respiratory infection**, conjunctivitis, etc., **with** or without **fever**, are likely related [to TM] if they occurred **several days** or in a 2- to 3-week period before the onset of neurologic symptoms. A transverse myelitis that emerges **during or within a few days of a febrile illness** is more likely related to **a neurological viral infection such as zoster or Epstein-Barr virus infection** or to a pyogenic bacterial infection such as epidural abscess. [emphasis added].

Respondent filed "Unusual Viral Causes of Transverse Myelitis: Hepatitis A Virus and Cytomegalovirus," by K.L. Tyler, et al., 1986 *Neur* 36(6):855-58. R. Ex. O. On page 855, the authors state that "20 to 40% of [TM] patients have evidence of **preceding or concurrent viral illness, but the specific viral diagnosis is rarely made**." [citations omitted.] [emphasis added.] The authors state at page 856 that poliovirus causes myelitis. In one of the two cases of TM following either hepatitis A virus or cytomegalovirus, the "infection occurred **coincident** with the symptoms of transverse myelitis." Id. at 856.

Respondent filed "Neurologic Complications of Immunization," by G.M. Fenichel, chapter 59 in K.F. Swaiman's Pediatric Neurology. Principles and Practice (1994). R. Ex. P. Dr. Fenichel states at 930, "The only definite neurologic complication of [OPV] is paralytic poliomyelitis."

Respondent filed "Transverse Myelitis Following Hepatitis B Vaccination," by F. Trevisani, et al., 1993 *J Hepatol* 19(2): 317-18. R. Ex. S. The authors discuss the case of a girl having TM three weeks after receiving hepatitis B vaccine. Her cerebral and spinal cord MRIs were normal. Id. at 317. They state they cannot prove that hepatitis B vaccine caused her TM because the patient had an increase in antibody titers against influenza virus. It could be, however, that the vaccine and the viral infection acted in synergy to evoke immune responses causing severe neurologic damage. Id.

Respondent filed "Transverse Myelitis after Diphtheria, Tetanus, and Polio Immunisation," by E.Whittle, et al., 1997 *Br Med J* 1 (607): 1450. R. Ex. T. A 7-month-old girl had TM 6 to 7 days after receiving her first Dt and OPV vaccines. She had a 4-day history of a slight cough and a hoarse cry associated with mild irritability and lassitude, a temperature of 38.2° C [100.7° F], and complete flaccid paralysis of her legs and lower trunk. Her spinal fluid and throat were normal. The authors did not know if Dt or OPV were the cause of her illness. They stated that no cases of myelitis in over 30 million doses of live oral polio vaccine had been reported. Id. at 1450.

**TESTIMONY**

David H. Morris, Taylor's father, testified first for petitioners. The oral polio vaccine Taylor received on July 12, 1996 was her second one. Tr. at 8. She did not have any problems with her first series of vaccinations at the age of two months. Tr. at 9, 38.

The onset of Taylor's illness was August 8, 1996. Tr. at 10. He took her to the babysitter from 7:30 to 8:00 a.m. Id. The babysitter paged him between 1:00 and 2:00 p.m. that Taylor was breathing rapidly and was limp. He took Taylor to Dr. Nickerson and his P.A. Jeff Taylor. Taylor was breathing extremely fast and she was limp. Tr. at 10-11. Rowan Regional Hospital is adjacent to the pediatrician's office. They started her on steroids. Tr. at 12. He did not remember that Taylor vomited her first two days in the hospital. Tr. at 35.

Mr. Morris denied that Taylor had coughing, congestion, or fever. Tr. at 36-37. If Taylor had been sick that morning, he would not have taken her to the babysitter. Tr. at 12. Taylor was transferred to Carolinas Medical Center, where they increased her dosage of steroids for five days. Tr. at 14. Dr. Knowles said that polio vaccine was a possible cause of Taylor's condition. Tr. at 15, 34. She possibly had a stroke since her pupils were uneven (they still are). Tr. at 16, 17. Taylor's left arm got better. Tr. at 17. She drags her left leg a little more than she drags her right leg. Her heel cords and hamstrings are tight. Tr. at 18. She receives physical therapy. Tr. at 19. She has a spastic bladder and is catheterized four times a day. Tr. at 22.

Sonya Morris, Taylor's mother, testified next for petitioners. Taylor was never sick as a baby. Tr. at 41. On July 12, 1996, she had her four-month vaccinations. Tr. at 42. The onset of her difficulty was August 8, 1996. Tr. at 43. The babysitter called David to say something was wrong with Taylor after her nap. Tr. at 43-44. Usually, Taylor woke herself from her nap, but on August 8, 1996, her babysitter went to wake her up. Tr. at 48. She was limp, dazed, and breathing rapidly, and did not fix her eyes on anything, but she had been fine that morning (although she did not see Taylor awake that morning because she left for work). Tr. at 44, 46. Mrs. Morris does not remember her having a fever, congestion, cough, or runny nose. Taylor looked scared. Tr. at 46, 48. She

vomited twice on August 11, 1996 because she is allergic to Tylenol. Tr. at 62. Dr. Knowles said that Taylor had a polio-like illness. Tr. at 53. Spasticity started after seven months. Tr. at 54.

Dr. Carlo Tornatore testified next for petitioners. He is an adult neurologist who has interests in viral molecular pathogenesis. Tr. at 79, 81, 82. He has seen patients with polio in the past and post-polio syndrome, but not an acute polio case. Tr. at 80. He does not treat children. Tr. at 82. He examined Taylor the day before trial. Tr. at 85.

Dr. Tornatore testified that Taylor did not have antecedent symptoms before August 8, 1996, and ascribed the medical recording of such symptoms as errors. Tr. at 86. Dr. Tornatore, on examination of Taylor, found her arms and legs strong, but spastic paraparesis (both her legs are very stiff). Her left pupil is a little smaller than her right. Tr. at 87-88. Taylor has a lesion at the C2-C3 level of the spine with weakness in the upper extremities. Tr. at 89. Dr. Tornatore felt that the 1998 physical therapist record which noted the level of Taylor's transverse myelitis was L-3 was probably dictated wrong. Everything affecting Taylor was at the cervical level. Id.

When she became ill, she was breathing very rapidly, had a temperature of 100.8 degrees, and a slight bulging of her fontanelle. Tr. at 89-90. Taylor had transverse myelitis, characterized by weakness and limpness. Tr. at 90. She had a profound event at the babysitter. Tr. at 91. Taylor's slight fever and rapid breathing were due to the transverse myelitis. Id.

Poliovirus is one of the few viruses that infect the nerve. It has a propensity to infect the spinal cord including the cervical spine, and affect respiration. Tr. at 92. Taylor had received attenuated poliovirus. Id. Even attenuated poliovirus has the propensity to get to nerves. Tr. at 93. The hope is it will not get to the nerves before the vaccinee mounts an immune response to it. Id. Taylor had some immunity from her first polio vaccine. Tr. at 94. In the second polio vaccine that

she received, one virus was somehow not attenuated and got into her spinal cord, causing an immune response which caused transverse myelitis. Taylor does not have polio because she does not have muscle atrophy. She had a monophasic illness. Id.

Myelitis is inflammation of the spinal cord. Tr. at 96. Transverse myelitis is very patchy. The immunogen or epitope[7] seems to be more on one side than the other (in the anterior or front of the spinal cord). Id. In transverse myelitis, a second nerve coming from the brain (the upper motor neuron) correlates with the first nerve (the anterior horn cell, the lower motor neuron). Tr. at 97. Polio affects the lower motor neuron and never causes spasticity. Id. If the upper motor neuron is affected, there is spasticity. Tr. at 97-88, 99. The upper motor neuron can affect the bladder, causing it to become spastic. Tr. at 100.

During the 27 days between vaccination and onset, the poliovirus replicated in the gastrointestinal tract to get into numbers high enough to enter the central nervous system. It took this amount of time because, being an attenuated virus, it probably was not replicating very well. Tr. at 101. A cold would not have caused transverse myelitis as quickly as the symptoms were described with the onset of Taylor's transverse myelitis. Tr. at 102. An x-ray showed that Taylor did not have pneumonia. She had reactive airway disease that caused wheezing. Tr. at 103. The loss of spinal cord function caused Taylor's respiratory problems. Id. She did not have axonal destruction. Tr. at 105. If Taylor had had cytomegalovirus or herpes virus, steroid treatment would have made her worse. Tr. at 105-06. People who get polio from polio vaccine actually have polio, which Taylor did not. Tr. at 106. Predominately, Taylor had a cellular response but she also had recruitment of her humoral or antibody response. Tr. at 105. She had a polio infection, but not

[7] An epitope is an "antigenic determinant." Dorland's, supra, at 572.

polio. Tr. at 110. Her upper motor nerves were more affected than her lower motor nerves. Tr. at 111.

Taylor did not have any white blood cells in her cerebrospinal fluid. Therefore, she had no inflammation from cytomegalovirus or herpes. Tr. at 113. Transverse myelitis is the right diagnosis for Taylor. Her glucose and protein were normal in her cerebrospinal fluid. She did not have herpes or other viruses. Tr. at 114-15. Spasticity is the sequela of her transverse myelitis. Dr. Tornatore does not believe that Taylor had an antecedent infection. Tr. at 116.

Dr. Tornatore testified that it is exceedingly rare for infants under six months of age to have transverse myelitis. There is always some antecedent episode. Tr. at 129. The reason Taylor did not develop polio is that the attenuated poliovirus was not as virulent as it could have been although it was not as attenuated as it should have been. That is one possible reason she did not have poliomyelitis. Tr. at 135. A second possibility is that she also had some protection from her first oral polio vaccine. Id. A third possibility is that just those who are immunodeficient contract polio from oral polio vaccine. Tr. at 137.

Poliovirus affects and kills anterior horn cells and can cause atrophy. Tr. at 166. Taylor's high cervical area affects the muscles in her neck. Id. She had neuromuscular problems in ventilation. Tr. at 168. If the muscles are weak, there is shallow breathing and an increased respiratory rate (in the 80s is too rapid; 12 to 18 per minute is the normal rate). Tr. at 169. Her spinal tap was relatively normal and there was no elevated white blood cell count or protein. Tr. at 171. He admitted that viral testing is very difficult. Tr. at 172.

Dr. Gerald V. Raymond testified for respondent. He is a pediatric neurologist. Tr. at 182. He has experience in developmental neuropathology, dealing with cerebral palsy, and teratology or

brain malformations. Tr. at 183. He has seen individuals who have had polio. He sees one case of transverse myelitis per year and has seen a total of 16 to 20 transverse myelitis cases. He also sees adult transverse myelitis cases. Tr. at 184-85.

Dr. Raymond's opinion is that OPV did not cause Taylor's transverse myelitis. Tr. at 186. She has no evidence of the features of polio. One could argue whether her transverse myelitis is complete or patchy. Taylor had a viral illness at the time she presented with fever, wheezing, reactive airway disease, and questionable pneumonia. She also vomited. Tr. at 187. A lesion at the cervical spine would decrease, not increase, her respiratory symptoms. She would not have had reactive airway disease from transverse myelitis at the cervical section. Tr. at 188, 189. (Dr. Tornatore disagreed, saying that the tidal volume declines and one would breathe faster. Tr. at 190.)

Dr. Raymond reiterated that Taylor had a viral illness leading to reactive airway disease and transverse myelitis, this being a reasonable hypothesis. Tr. at 193. A virus can replicate long before the patient manifests symptoms. The incubation period for a virus can last from 3 to 7 days before symptoms show. Tr. at 195. (Dr. Tornatore disagreed, stating that a cold virus or adenovirus replicates quickly, within hours. Id. He said that if Taylor had had an infection, she would have had a runny nose. Tr. at 196. Rapid breathing can cause wheezing. Her fever could have been from the transverse myelitis.)

Dr. Raymond reiterated that replication of a virus takes days and wheezing is not a manifestation of tachypnea. Children do not breathe at 10 to 12 times a minute but more likely 20 to 24 times a minute. Tr. at 199. Taylor responded to Alubuterol, a medication used to treat reactive airway disease. The next morning, she was better. Tr. at 200.

Dr. Raymond testified that manufacturing the OPV alters the poliovirus, creating mucosal immunity as well as circulating immunity. Tr. at 201. That Taylor already had received her first OPV at two months probably means she had some circulating immunity. Tr. at 201-02. The onset of a vaccine-associated paralytic polio is in the first month after receiving OPV. The peak incidence is around two weeks. Tr. at 202. Transverse myelitis is diagnosed both clinically as well as with radiographic evidence of cord inflammation. Id. There are multiple causes of transverse myelitis: viral, bacterial, protozoan, inflammatory agents, MS, lupus, etc. Id. OPV has never been associated with transverse myelitis. Id.

Very little was done to look for the cause of Taylor's transverse myelitis. Tr. at 213. They looked only for a bacterial infection. Tr. at 214. They did not look for a viral cause. Id. (Dr. Tornatore stated that viral analysis is very difficult. To isolate a virus from cerebrospinal fluid and grow it is almost impossible because there are so many other things in the spinal fluid that almost inhibit viral growth. Tr. at 215. He does these studies and it is very difficult to isolate virus. Id.)

Taylor first had reactive airway disease, fever, and possible pneumonia, but did not develop neurological signs of weakness in her left arm until two days after being hospitalized. This is consistent with the course Dr. Raymond has seen in other cases of acute transverse myelitis secondary to viruses. One often has a prodromal phase. The classic used to be measles and some of the other viral exanthems. As the rash cleared, the transverse myelitis would develop. We do not see these any more because of the vaccinations. However, that is the classic picture if there is a prodromal illness. Tr. at 218. One does not always see a prodromal illness.

Since Taylor did not have atrophy or muscle wasting, she did not have polio. Tr. at 219. The use of steroids is to reduce the inflammation. Id. His understanding is that the one or two cases

reported in the literature of transverse myelitis following OPV are felt to be coincidental and there has never been a clear causal relationship between them. Tr. at 221. He agrees that OPV has been causally associated with Guillain-Barre Syndrome (GBS). Tr. at 222. He also agrees that hepatitis A virus and cytomegalovirus have been causally associated with transverse myelitis. Respondent's exhibit O states that 20 to 40 percent of cases of acute transverse myelitis are attributed to viral infections, although the specific viral etiology is only rarely identified. Tr. at 224. We know that there are lots of viruses that can cause transverse myelitis. Tr. at 228. The most likely viruses that would have caused Taylor's transverse myelitis are adenovirus and enterovirus. Id. Polio is an enterovirus, but there are dozens more. Echovirus in an enterovirus. Id. Epstein-Barr virus and cytomegalovirus often present as a very mild viral illness. There is no evidence that the transverse myelitis they may cause will be any different that what Taylor had. Tr. at 229.

Poliovirus can cause transverse myelitis, but not the polio vaccine. Tr. at 230. The clinical event that Dr. Tornatore described of a component in the polio vaccine being more virulent and forming a nidus of infection in the cervical section of Taylor's spine, causing transverse myelitis, has never been documented. Tr. at 230-31, 234. Dr. Raymond thinks that the literature that says poliomyelitis causes transverse myelitis would probably on examination show that most of the cases are not documented with clear evidence of polio infection. In those cases that are indisputably related, probably the patients would also have polio as part of their symptoms. Tr. at 231.

There is a difference between the attenuated polio vaccine and the wild-type virus. The vaccine changes the virus so that it does not cause disease. Tr. at 233. Dr. Tornatore was impressed with the medical article (P. Ex.13) describing a young man who had never received polio vaccine who developed transverse myelitis and then polio myelopathy because it shows that poliovirus can

induce transverse myelitis. It does not matter that the young man had myelopathy rather than myelitis. Tr. at 234.

Mr. Morris resumed testifying. He said that during Taylor's first two hospitalizations, she never moved any of her extremities, but remained limp. Also, she did not acknowledge that her parents were there. Tr. at 238. Mrs. Morris resumed testifying. When Taylor was brought in, the hospital personnel put her in a croup tent. Tr. at 243. Taylor moved her right hand maybe an inch. Tr. at 242, 244. Her left hand had an IV in it. Tr. at 249.

Dr. Raymond stated that the records note that Taylor was moving on both sides. The observers picked up her flaccidity two to three days after admission, buttressing his testimony that Taylor had a prodromal viral illness with the onset of her transverse myelitis several days later. Tr. at 251.

Dr. Tornatore's view is that we cannot interpret Taylor's condition in the first few days because she had an IV board on her left arm. Clearly, she was lethargic and she did not have normal movement. He does not think a day or two makes a huge difference. Even if there were a cold that started the same day, one cannot get transverse myelitis from an infection that started that day that theoretically caused reactive airway disease. It does not make sense that Taylor would get transverse myelitis two days later. Tr. at 252.

After petitioners filed medical records from Wake Forest and produced the MRI films for respondent's expert to examine, respondent filed a supplemental report from Dr. Raymond, dated April 16, 2002. R. Ex. U. Dr. Raymond states that his opinion of the brain image of August 12, 1996 is that it demonstrates ventricular dilatation and asymmetry of the ventricles, with the left being larger than the right, and a decrease in apparent white matter. This is consistent with a congenital

abnormality resulting in a fixed motor deficit such as cerebral palsy. Dr. Raymond also wrote that the August 12, 1996 MRI of Taylor's cervical spinal cord is normal without any evidence of swelling or increased signal.

Dr. Raymond states that if one were to accept the documented physical and radiographic examinations of Taylor, there is no evidence to suggest she has transverse myelitis. At her initial presentation, she had weakness of her left arm, but no leg involvement and her CSF examination was unremarkable. The transverse myelitis diagnosis was based on history. But Dr. Raymond adds, based on the MRIs, the original diagnosis of transverse myelitis in Taylor is unsupported. Her history, physical examination, and MRIs are inconsistent with a diagnosis of transverse myelitis. Taylor does have spastic diplegia, and Dr. Raymond recommends further testing to determine what she has. Id.

Dr. Tornatore, in response to Dr. Raymond's supplemental report, wrote a supplemental report dated October 3, 2002. P. Ex. 21. He reviewed the MRI scans done August 1996, including the MRI of Taylor's cervical spine. He finds Taylor's course consistent with a diagnosis of transverse myelitis. He rejects any diagnosis of cerebral palsy because cerebral palsy's onset is not acute. Horner's sign (the documented change in Taylor's pupils) is not seen in cerebral palsy, and demonstrates an acute injury to the spinal cord. Id.

Dr. Raymond, in response to Dr. Tornatore's supplemental report, wrote in a report dated December 9, 2002, that the medical records do not support Dr. Tornatore's statement that Taylor had decreased tone in her legs. He still feels Taylor's cervical MRI was normal. Dr. Raymond notes that Drs. Kadrie and Santos on August 25, 200 localized Taylor's area of involvement to her lumbosacral rather than her cervical spinal cord. R. Ex. V.

## DISCUSSION

Petitioners have two options under the Vaccine Program: (1) to proceed under the theory of a Table injury or (2) to proceed on a causation in fact theory. Because petitioners are not alleging that Taylor contracted polio following her OPV, there is no Table injury alleged in this case. Petitioners are therefore proceeding on a theory of causation in fact.

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

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

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

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).

This case is replete with medical conflict. The experts for both parties disagreed with numerous parts of the contemporaneous medical records (although everyone agrees that Taylor's health was excellent for 27 days after her receipt OPV). Dr. Tornatore, plaintiff's expert, disagreed that Taylor had an upper respiratory infection when she became ill on August 8, 1996. His explanation for why the level of her spinal cord affected by transverse myelitis (TM) was listed as the lumbar, rather than the cervical, spine in later medical records was that it was probably an error.

Dr. Raymond, respondent's expert initially agreed that Taylor had TM at the cervical level, but changed his mind after seeing the actual 1996 MRI of her cervical spine which he said was normal. (Dr. Tornatore responded, after seeing the same, that it was not normal.) He also had no explanation for the difference in cord level (cervical vs. lumbar) in the medical records. But Dr. Raymond did accept that Taylor had an upper respiratory infection, probably of viral origin, when she was first admitted to the hospital on August 8, 1996.

Neither Dr. Tornatore nor Dr. Raymond discussed in their supplemental reports the September 1, 2000 radiologic examination of Taylor's pelvis which showed pedicular widening at her lower lumbar and sacral vertebrae, consistent with myelomeningocele. This condition is a hernial protusion of the spinal cord and its meninges at Taylor's lower lumbar and sacral levels because of a defect in the vertebral canal. That, in itself, would cause her paraplegia and spastic bladder. This condition is presumably congenital. The protrusion of the spinal cord and meninges damages the spinal cord and nerve roots at that level, causing a decrease or lack of function of body areas controlled at or below the defect.

Taylor's symptomatology matches the level of this myelomeningocele, which is undoubtedly why her doctors (according to the 1998 and 2000 medical records) opined that the level of her TM

was in the lumbar region. An MRI of Taylor's lumbar spine taken on August 30, 2000, two days before the detection of her myelomingocele, was normal. The medical literature does state that someone may have TM with a normal MRI, but it would be extraordinarily coincidental that Taylor had both a lesion in her lumbar spine as well as a myelomeningocele at the very same spinal level.

What seems most likely in this case is that Taylor's swelling of her cervical spinal cord healed and her subsequent medical history is due to her myelomeningocele. But this is pure speculation on the undersigned's part since neither expert offered an opinion about this. (Of course, they could not have offered an opinion at the hearing because petitioners did not file the September 1, 2000 record of the myelomeningocele until after the hearing. But even in their supplemental reports, both experts ignored the findings consistent with a myelomeningocele.) Therefore, the court will analyze this case solely on the allegations petitioners have made and the testimony Dr. Tornatore gave, compared with the medical records, the testimony of Dr. Raymond, and the medical literature.

Petitioners allege that Taylor's second OPV caused her TM 27 days later. Dr. Tornatore, who is an adult neurologist, testified that he did not believe the contemporaneous medical records were correct when they state that she had an upper respiratory infection at the time of admission. In addition, he thinks they were incorrect when they did not diagnose her as having a neurologic condition on August 8, 1996. His view is that she had a sudden onset of TM on August 8, 1996, that the TM caused her fever and wheezing, and that a virulent component of the OPV resulted in her TM, which has led to her spastic bladder and paraplegia.

The parents either do not remember or dispute that Taylor had a fever and upper respiratory infection on August 8, 1996. Well-established case law holds that information in contemporary medical records is more believable than that produced years later at trial. United States v. United

States Gypsum Co., 333 U.S. 364, 396 (1948); Burns v. Secretary, HHS, 3 F.3d 415 (Fed. Cir. 1993); Ware v. Secretary, HHS, 28 Fed. Cl. 716, 719 (1993); Estate of Arrowood v. Secretary, HHS, 28 Fed. Cl. 453 (1993); Murphy v. Secretary, HHS, 23 Cl. Ct. 726, 733 (1991), aff'd, 968 F.2d 1226 (Fed. Cir.), cert. denied sub nom. Murphy v. Sullivan, 113 S. Ct. 263 (1992); Montgomery Coca-Cola Bottling Co. v. United States, 615 F.2d 1318, 1328 (1980). Contemporaneous medical records are considered trustworthy because they contain information necessary to make diagnoses and determine appropriate treatment:

> Medical records, in general, warrant consideration as trustworthy evidence. The records contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium. These records are also generally contemporaneous to the medical events.

Cucuras v. Secretary, HHS, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

When faced with a conflict between the parents' testimony and the history and observations of the treating medical staff, the undersigned selects the history and contemporary observations of the medical staff as more credible. (Dr. Tornatore testified to the opposite. He selects the parents' version of events when faced with a conflict between contemporanous medical records and parents' testimony. His position is not consistent with legal precedent.)

Dr. Raymond, respondent's expert who is a pediatric neurologist, testified that Taylor indeed had an upper respiratory infection on August 8, 1996, which resulted in her fever, wheezing, and limpness. He agrees with the medical records that the onset of her TM (at trial, he accepted that Taylor had TM) was days after her hospitalization.

The undersigned finds the medical records more credible than Dr. Tornatore's dismissal of them as error and the parents' disagreement with them. The undersigned holds that the doctors who

examined Taylor were accurate recorders of her medical history and accurate examiners and observers of her medical condition during her hospitalization from August 8, 1996 until her transfer on August 12, 1996. Taylor entered with upper respiratory infection symptoms: fever, lethargy, wheezing, and coughing. She was found to be grossly intact without any focal or neurologic deficits on neurologic examination on August 8, 1996. Her anterior fontanelle was flat, not bulging. (Dr. Tornatore's testimony implied that her fontanelle was bulging from the very beginning.) She was fairly alert and non-toxic in appearance. However, her lungs had scattered rhonchi with slight expiratory wheeze and mild retractions. She vomited on both August 8th and 9th. Because the doctors were concerned about early pneumonia and reactive airway disease, Taylor was placed in a croup tent and administered medications. They worked and she was better the next day.

However, she subsequently began to show neurologic symptoms. On August 9, 1996, she had unequal pupils and did not grasp as well with her left hand as with her right. She had some flaccidity in her left upper and lower extremities. She was breathing better but still had upper airway congestion on August 10, 1996. Her anterior fontanelle however was still flat. A CT scan of her head ordered on August 11, 1996 showed slight prominence of her ventricles without acute findings. She was alert on physical examination and had a vigorous suck.

On August 12, 1996, Taylor was happy, afebrile, and eating well. The doctors suspected she might have had a cerebrovascular accident and decided to transfer her to a neurologist Dr. Santos. Also on August 12, 1996, Taylor's fontanelle was slightly bulging and somewhat tense compared to August 11th. Her left arm and left leg had minimal movement.

On August 12, 1996, Taylor was transferred to Carolinas Medical Center from Rowan Medical Center. Dr. Koontz at Rowan had ordered an MRI which showed a swollen cervical cord

(petitioners did not file the written interpretation of this MRI). Her pupils were still unequal. On August 12, 1996, Dr. Knowles, a neurologist, examined Taylor and found her neck supple and her fontanelle soft. Neurologically, she had a flaccid arm. The lower extremities seemed to move symmetrically with a suggestion of some mild decrease in tone bilaterally. She withdrew her lower extremities bilaterally very well. She had an asymmetric face.

In subsequent medical records, on August 1, 1997, Taylor's extremities had full range of motion. A record on August 1, 1998 (of which petitioners filed only the first page) notes that she has frequent urinary tract infections and a spastic bladder.

An orthopedist, Dr. Koman, saw Taylor on August 25, 2000, and opined that her spinal lesion was likely in the high lumbar region, although he had no MRIs to substantiate this. Dr. Santos, the neurologist, concurred.

Dr. Raymond, respondent's expert, testified that viral infections are commonly the cause of TM. Medical literature supports him on this point. He also testified that it is unlikely that Taylor would have reacted to her second OPV, even if it had been virulent as Dr. Tornatore testified, because her first OPV would have immunized her against the poliovirus. Medical literature supports Dr. Raymond again.

The literature shows that when inactivated Salk polio vaccine was erroneously virulent, in the Cutter incident in the 1950s, it caused the occurrence of poliomyelitis in recipients. Very rarely, if at all, does polio vaccine cause TM. There is anecdotal mention that the poliovirus, not the vaccine, can cause transverse myelopathy. An unimmunized teenage boy contracted transverse myelopathy upon exposure to poliovirus. The article describing his case does not mention the interval between exposure to the poliovirus and the onset of his myelopathy.

But there is a great difference between the attenuated strain of poliovirus in OPV and the poliovirus itself, which is why Dr. Tornatore offered as the basis for his opinion of causation that Taylor's second OPV must have had an unattenuated (virulent) component to it. But, he does not know this. He speculated that the component of Taylor's second OPV was not virulent enough to cause her to contract poliomyelitis but not attenuated sufficiently so as to be harmless. He also speculates that the 27-day interval between Taylor's receipt of OPV and the onset of her symptoms (whether neurological or not) fits within his hypothesis that the attenuated poliovirus needed that longer period of time to replicate because it was not virulent enough to strike sooner. Dr. Tornatore's bases are too theoretical to be given weight. He has no support in the literature (which is extensive concerning polio). One would expect more people than Taylor to have contracted TM if there were virulent strains of OPV in use, just as the virulent strains of IPV caused what became known as the Cutter incident (and the illnesses there were poliomyelitis, not TM). Moreover, Dr. Raymond testified, and medical literature supports his statement, that the first OPV that Taylor received at the age of two months would have immunized her against poliovirus if her second OPV had somehow been insufficiently attenuated.

Dr. Tornatore testified that even if Taylor had had a respiratory infection, it occurred too close in time to Taylor's onset of TM for it to have been the cause of her TM. But Dr. Raymond testified that this is not true, and medical literature supports his statement. Dr. Raymond stated that the clinical symptoms of a respiratory infection manifest a few days after the individual already has the infection. Moreover, Taylor's neurological symptoms developed over days. The medical literature states that a respiratory infection, either simultaneous to or preceding the onset of TM, is likely causally related to the TM. That Rowan Medical Center did not culture out the specific virus

is not dispositive of whether Taylor had a virus. Dr. Tornatore agreed, and medical literature supports Dr. Tornatore here, that it is immensely difficult to culture out the virus responsible. Medical literature states that 20 to 40% of TM patients have evidence of preceding or concurrent viral illness, but specific viral diagnosis is rarely made.

The undersigned accepts that Taylor had a respiratory infection preceding the onset of her TM because that is the conclusion of her treating doctors. Her symptomatology and the course of her symptoms seem most consistent with that conclusion. Her TM did not develop all at once because her initial neurological examination on August 8, 1996 was normal. It took days for her neurological symptoms to manifest themselves, starting with unequal pupils the following day, and after that, bulging and tense fontanelles.

Dr. Raymond, a pediatric neurologist with extensive experience, testified that this respiratory infection caused Taylor's TM. Medical literature is highly supportive of a relationship between viral infections, even concurrent ones, but certainly ones occurring within a few days of onset of TM, being the cause of TM. The court holds that Taylor's respiratory infection, signifying a viral infection, is the cause of her TM, and not her OPV.

Petitioners have not prevailed in their allegation that OPV caused Taylor's TM.

## CONCLUSION

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

**IT IS SO ORDERED.**

_____ _____

DATE Laura D. Millman
Special Master