# In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 05-1024V
July 24, 2007
To be Published

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| SEAN VAN UUM and KATHLEEN VAN UUM, as parents and natural guardians of DEVON VAN UUM, a minor, | * | |
| Petitioners, | * | |
| v. | * | Entitlement; Down syndrome child received DPaT and IPV and had monoplegia |
| SECRETARY OF THE DEPARTMENT OF HEALTH AND HUMAN SERVICES, | * | |
| Respondent. | * | |

Donald P. Edwards, Atlanta, GA, for petitioners.
Lisa A. Watts, Washington, DC, for respondent.

**MILLMAN, Special Master**

## DECISION[1]

On September 23, 2005, petitioners filed a petition under the National Childhood Vaccination Injury Act, 42 U.S.C. §300aa-10 et seq., alleging that their daughter Devon Van

[1] Vaccine Rule 18(b) states that all decisions of the special masters will be made available to the public unless they contain trade secrets or commercial or financial information that is privileged and confidential, or medical or similar information whose disclosure would clearly be an unwarranted invasion of privacy. When such a decision or designated substantive order is filed, petitioner has 14 days to identify and move to delete such information prior to the document's disclosure. If the special master, upon review, agrees that the identified material fits within the banned categories listed above, the special master shall delete such material from public access.

Uum (hereinafter, "Devon"), who was born with Down syndrome and had a history of heart defect corrected by surgery, had an adverse reaction to DPaT and/or inactivated polio vaccine, resulting in dragging her left foot and suffered the Table injury of encephalopathy.

Attached to the petition was an expert report from Dr. Albert A. Cook, a neurologist, stating that pertussis vaccine impaired Devon's motor performance in the left leg, due to a focal encephalopathy. Due to the time interval, he opined that pertussis vaccine caused Devon's focal encephalopathy.

In a status conference with the parties on October 25, 2005, the undersigned informed petitioner's counsel that there was no Table encephalopathy here since the hallmark of a Table acute encephalopathy, according to the aids to interpretation, is a significantly decreased level of consciousness for at least 24 hours. 42 C.F.R. §100.3(b)(2). Petitioners then alleged causation in fact.

A hearing was held on March 29, 2007. Testifying for petitioners were Kathleen Van Uum, Sean T. Van Uum, and Dr. Ben Renfroe. Testifying for respondent was Dr. John B. Bodensteiner.

Petitioners filed a post-hearing brief on June 13, 2007. Respondent filed a post-hearing brief on July 5, 2007.

## FACTS

Devon was born on March 30, 2001 with Down syndrome. She was in the neonatal ICU for two weeks. She had an enlarged heart and was on a ventilator for three days. Med. recs. at p. 272.

On March 31, 2001, Devon had a pediatric echocardiogram, which showed an enlarged right ventricle in her heart. Med. recs. at p. 153. She had cyanosis and respiratory distress. Med. recs. at p. 155. Devon had a large atrioventricular septal defect, secondary to an atrial septal defect (ASD) with bi-directional shunt, and a large patent ductus arteriosus (PDA) with right to left shunt, as well as an enlarged right ventricle. *Id.*

On April 9, 2002, Devon was noted to belly crawl. Med. recs. at p. 253. On July 3, 2002, she was also noted to belly crawl. Med. recs. at p. 265.

On September 25, 2002, Devon received acellular DPT and inactivated polio vaccines. Med. recs. at pp. 252, 254.

On November 7, 2002, Devon saw Dr. R. Tracy Ballock, a pediatric orthopedic surgeon. Devon had begun dragging her left foot two months before, shortly after she received vaccinations in her left thigh. Med. recs. at p. 248.

On February 20, 2003, Devon was taken to the University Hospitals of Cleveland because she was dragging her left foot and not walking. Med. recs. at p. 272.

Also on February 20, 2003, Devon had a pediatric physical therapy evaluation. In summary, physical therapist Orphea Bass noted that she did not see any dragging. Med. recs. at p. 280. Devon's left heel cord was tight at neutral and seemed a bit stiffer than her right heel cord in tone. *Id.*

On May 21, 2003, Dr. Douglas Henry, a doctor specializing in physical medicine and rehabilitation, saw Devon. He noted that, at 18 months, she received her vaccinations. After this, she began dragging the toes on her left side. Now, she was walking on the toes of her left side. Med. recs. at p. 299. Devon had never walked independently, but now took steps well

holding someone's hand. It was not clear to Dr. Henry why she walked on her toes. He opined that if Devon suffered any vaccine reaction, it would have been a lower motor neuron problem. *Id.*

On August 26, 2003, Devon had surgery to repair the septal defect in her heart. Med. recs. at p. 65 (supp. recs.).

On October 2, 2003, Dr. Henry noted that Devon had increased tone in her left calf after vaccinations. Botox injections helped. Med. recs. at p. 301.

On November 13, 2003, Devon was taken to Children's Healthcare of Atlanta. Devon began walking at the end of September 2002. She dragged her left foot the day after she received vaccinations. She had increased spasticity of the left lower extremity, predominantly in the calf area. Med. recs. at p. 304.

On December 31, 2003, Devon saw Dr. Edward M. Goldstein, a pediatric neurologist. He diagnosed Devon with post-dysimmune reaction to vaccinations with residual left lower extremity monoparesis. Med. recs. at p. 312.

On June 8, 2004, Dr. Goldstein gave Devon a motor examination and found moderate, generalized hypotonia with superimposed dynamic hypotonia affecting her left lower extremity. Med. recs. at p. 314.

On May 27, 2005, Devon had a brain MRI done which showed possible periventricular leukomalacia.[2] Med. recs. at p. 319. Her corpus callosum was slightly atrophic as was the pons.

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[2] Periventricular leukomalacia is "bilateral necrosis of the white matter of the brain adjacent to the lateral ventricles, seen in the neonatal period, especially in premature newborns, and manifested by chalky, yellowish-white plaques in the white matter, with proliferation of astrocytes and microglia; cyst formation may lead to multicystic encephalopathy." Dorland's Illustrated Medical Dictionary, 30th ed. (2003) at 1022.

She had patchy areas of T2 hyperintensity in her deep periventricular white matter, on the right more than the left. Dr. Ariane Neish diagnosed Devon with brain cerebral palsy. *Id.*

On May 27, 2005, Devon also had an MRI done of her lumbar spine for a clinical history of a tethered cord. Dr. Neish's impression was a low-lying distal spinal cord at the top of L3. The distal spinal cord also lay slightly posteriorly within the spinal canal. Med. recs. at p. 35.

On December 14, 2005, Devon had another brain MRI done which showed no significant interval change in the size and configuration of her ventricular system. She had patchy periventricular areas of increased signal most likely consistent with gliosis,[3] unchanged from the prior study. Med. recs. at p. 122 (supp. recs.).

**Other Submitted Materal**

On November 20, 2006, petitioners filed a letter from Dr. William R. Balchunas, a radiologist. He reviewed Devon's brain MRIs of May 27, 2005 and December 14, 2005. He saw extensive periventricular deep white matter signal changes in addition to ventriculomegaly.[4] He did not see evidence of acute infarct in the diffusion-weighted images. There were no significant changes from the first to the second MRI. The MRI distribution of injury was not consistent with findings typically seen in viral or post-immunization injury patterns (i.e., extensive subcortical and deep white matter [central semi-ovale]) signal changes. The localized periventricular white matter pattern in combination with ventriculomegaly strongly suggested to Dr. Balchunas a prior

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[3] Gliosis is "an excess of astroglia in damaged areas of the central nervous system." Dorland's Illustrated Medical Dictionary, 30th ed. (2003) at 778. Astroglia are astrocytes. *Id.* at 170. An astrocyte is "a neuroglial cell of ectodermal origin, characterized by fibrous, protoplasmic, or plasmatofibrous processes." *Id.* at 169.

[4] Ventriculomegaly is "gross enlargement of a ventricle of the brain." Dorland's Illustrated Medical Dictionary, 30th ed. (2003) at 2031.

hypoxic-ischemic injury, e.g., periventricular leukomalacia often seen in premature infants. This pattern often resulted in spastic diplegia,[5] which might explain Devon's use of bilateral leg supports. The distribution of injury would be unusual for an embolic or even a hypotensive etiology. He did not see lacunar[6] type infarcts[7] or injury to the cortical or watershed areas. In Dr. Balchunas' opinion, the MRI scans did not explain Devon's acute left leg symptoms. The scans were consistent with a static remote injury pattern.

On January 3, 2007, petitioners filed an undated letter from Cynthia Rimko, Devon's former physical therapist. Ms. Rimko first evaluated Devon when she was 3 and ½ months old in July 2001. Devon had Down syndrome complicated by pulmonary hypertension caused by two holes in her heart. Her parents planned to have Devon undergo heart surgery, but surgery was postponed. Ms. Rimko saw Devon once a week until September 2001 when she saw Devon twice a month. In December, Ms. Rimko saw Devon once a month due to her steady progression of skills. She began to stand and show signs of being able to stand and walk. In February 2002, Ms. Rimko saw Devon once every three weeks. Devon had a decrease in tone. She was developmentally delayed. At one year, Devon could sit independently. Devon would ambulate if a parent or the therapist held both her hands. She had no heel stroke and could not pull her toes up into a dorsiflexed position. Ms. Rimko saw Devon the last time in July 2002 because her

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[5] Diplegia is "paralysis affecting like parts on both sides of the body." Dorland's Illustrated Medical Dictionary, 30th ed. (2003) at 524.

[6] Lacuna is "anatomic nomenclature for a small pit or hollow cavity." Dorland's Illustrated Medical Dictionary, 30th ed. (2003) at 991.

[7] An infarct is "an area of coagulation necrosis in a tissue due to local ischemia resulting from obstruction of circulation to the area, most commonly by a thrombus or embolus." Dorland's Illustrated Medical Dictionary, 30th ed. (2003) at 927.

mother was due to give birth. Devon continued to ambulate with two hands held or with a push behind a cart. Ms. Rimko resumed giving physical therapy to Devon on October 1, 2002. She saw her again on October 17, 2002 and November 7, 2002, which was her final visit. Devon had a toed-in gait and seemed not to be advancing with her left foot. She turned the whole left leg and dragged the left foot. She had higher tone on the left side.

**TESTIMONY**

Kathleen Van Uum, Devon's mother, testified first for petitioners. Tr at 8. Devon was induced at 38 weeks because the baby was not moving much in utero. Tr. at 9-10. When she was born, Devon had pulmonary hypertension, respiratory distress, and thrombocytopenia. There were two large holes in her heart. Tr. at 10. Devon was not walking independently before her vaccinations, but she would walk flatfooted if someone held her hand. Tr. at 12. Devon had an atrial septal defect and a moderately leaking mitral valve in her heart. Tr. at 13.

On September 25, 2002, in the morning, Devon received acellular DPT and inactivated polio vaccines. Tr. at 14-15. She had an appointment to visit a special needs preschool group comprised of an occupational therapist, a physical therapist, and a speech therapist at noon the same day. They were there to assess Devon's capabilities, but Devon would not stand on her left leg (where she had been vaccinated). Tr. at 15. Two to three days later, Devon put pressure on the left leg and dragged the toes across the floor. Tr. at 16-17.

Ms. Van Uum got a referral to a pediatric orthopedist, Dr. Ballock, whom she saw on November 7, 2002. Tr. at 19. The pediatrician did not understand what had happened to Devon's leg. Tr. at 21. The orthopedist wanted to monitor Devon's left leg for two months

before bracing it. *Id.* Someone with Down syndrome has low tone (hypotonia), not hypertonia. *Id.*

By February 7, 2003, Devon was walking on her toes. *Id.* This began at the end of January 2003. Tr. at 22. Devon had open heart surgery on August 26, 2003. Tr. at 25. The only option for Devon's hypertonic heel cord and tendons is surgery which might not work. Tr. at 28.

Sean Van Uum, Devon's father, testified next for petitioners. Tr. at 38. He agreed with his wife's testimony. Tr. at 39.

Dr. James Benjamin Renfroe, a pediatric neurologist, testified next for petitioners. Tr. at 41. He runs a spasticity clinic which occupies 40% of his time. Tr. at 42. He said that no one in his spasticity clinic has Down syndrome. *Id.* We know that until Devon stopped taking physical therapy in July 2002, she did not have spasticity because of the report of Devon's physical therapist. Tr. at 43.

There is a very strong timeline associated with Devon's immunizations and her development of problems. *Id.* He thinks that of the two vaccinations, DPT caused Devon's spasticity. Tr. at 44. He thinks that Devon has transverse myelitis. Tr. at 45. There were no scanning studies done early on when she developed her problem. *Id.* A lumbar MRI was normal. He thinks Devon has an upper motor neuron lesion. *Id.* The lesion should be above the lumbar spinal cord. Tr. at 46. He is speaking about the cervical or thoracic spine. *Id.* This transverse myelitis would be a fairly small lesion because it affects only one leg. *Id.*

Devon had immediate pain after the immunization and some type of inflammatory response preceding her central nervous system problem. Tr. at 46-47, 48-49. Two to three days after vaccination, Devon was dragging her leg. Tr. at 47. Dr. Renfroe suspected that it was

during this time period when she began to develop the lesion. *Id.* It is controversial whether two to three days is long enough to get a transverse myelitis lesion from a DPT vaccination. But this was Devon's fourth DPT and she had preexisting antibodies that could have led to an enhanced response. *Id.* Devon's leg pain and not bearing weight indicate an inflammatory response unrelated to or maybe preceding her development of a central nervous system problem. Tr. at 48-49. Dr. Renfroe hypothesized that Devon had an excessive reaction because of an enhanced immune reaction due to her previous immunizations. Increased inflammatory response at the vaccine site was likely. Tr. at 49. Later, due to some autoimmune phenomenon, she developed a demyelinating disorder, i.e., transverse myelitis. *Id.*

Dr. Renfroe said that in a traumatic injury, one can have a spinal shock syndrome where initially the injury is a flaccid limb, as Devon dragged her toes. *Id.* He hypothesized there was some injury to the lower motor neuron at that point, weakening her leg. *Id.* As she healed, she later developed the acute lesion of spasticity which is characterized by the brain's loss of control of that lower motor neuron so the lower nerve emitted too much information, causing the muscles it controlled to be spastic. Tr. at 49-50.

This was a maturation of the same lesion. Tr. at 50. The lower motor neuron is in the spine. It is the last nerve that sends a signal to the leg or to any muscle. It is controlled by the upper motor neuron. Tr. at 50-51. A lesion in the spine can affect both especially in an initial lesion when there is inflammation and swelling. Tr. at 51. As it heals, the lower motor neuron, if it survives, becomes intact and one has scar tissue interfering with the control of the lower motor neuron by the upper motor neuron. *Id.*

In cases of transverse myelitis, the patient can initially have weakness and floppiness because he has an acute inflammatory process in the spine which may be very focal or devastating. When that acute inflammatory process clears, the patient gets scar tissue, causing a decrease in communication of the spinal pathways, resulting in spasticity. Tr. at 52. The scar tissue causes the upper motor neuron lesion. *Id.*

Dr. Renfroe said it was possible to get an upper motor neuron lesion in the brain, but Devon's brain was very complicated with evidence of periventricular leukomalacia. Tr. at 53. Periventricular leukomalacia can certainly result in spasticity. *Id.* Periventricular leukomalacia in a premature infant is the most common cause of spasticity in the legs. *Id.* Devon had bilateral scarring of the white matter of her brain around the ventricles in the central part of her brain, but amazing sparing of both her legs. *Id.* If Dr. Renfroe had seen Devon's brain MRI on an examination and were asked the outcome, he would have answered spastic diplegia, i.e., spasticity in both legs. *Id.* But Devon had instead monoplegia with bilateral ventricular leukomalacia. Tr. at 54.

Dr. Renfroe did not see any ministrokes or emboli in Devon's brain MRI. *Id.* If someone had an emboli, i.e., a blood clot shooting off into the brain, as it got into smaller and smaller arteries, at some point, it would get stuck, clog everything distal to that artery, and result in a wedge-shaped lesion because the artery will branch out due to the wedge-shaped lesion. Tr. at 55. Dr. Renfroe did not see any evidence of that type of lesion or embolic phenomena in Devon's brain MRI. *Id.* He said Devon had remarkable sparing of the cortex of her brain. *Id.* She has white matter disease, but it would be very difficult to clot one of her arteries in the white matter and not affect any of the cortex. *Id.* The outside of her brain is not involved. One would

expect if someone had an embolus, i.e., a blood clot that caused a stroke in the brain, even a small one would be peppered throughout the brain. Tr. at 56. It would spread outward to involve both the white matter and the cortex. *Id.* In Devon's case, the damage is along the middle part of the brain, which is seen when the child is at 24 to 28 weeks' gestation and the blood vessels in the middle of the brain are very immature. If at that stage, Ms. Van Uum or Devon had a significant problem, those blood vessels could not get enough flow or got too much and burst, resulting in the periventricular leukomalacia we see in Devon. *Id.*

Dr. Renfroe disagreed with respondent's expert Dr. Bodensteiner that, before Devon had the holes in her heart repaired, an embolus broke off and entered her brain. Tr. at 57. He consulted a pediatric cardiologist, Dr. Mehta, who said that it would be unknown to her that a Down syndrome child would have a right to left shunt. The shunt goes left to right, meaning the high-pressure side of the heart pushes blood back, and it keeps going through the heart, resulting in the risk of failure. *Id.* Devon's doctors thought her heart defects were benign enough to delay surgery until she had a chance to grow. Tr. at 58.

Devon's August 26, 2003 surgery reported an atrioseptal defect, the blood flowed from high-pressure areas to low-pressure. *Id.* Dr. Mehta told him that blood that flows from the left side of the heart back to the right does not run the risk of emboli because the left side is high pressure and the right is low pressure. *Id.* Dr. Renfroe said that if Devon had emboli from the heart for whatever reason, the emboli would impact multiple areas as small dots, including the brain, lungs, kidney, liver, gastrointestinal system, and fingernails. Tr. at 58-59. Devon should have had multiorgan system involvement if she had emboli. Tr. at 59.

Devon had isolated periventricular problems in her brain: scarring in the periventricular area. The ventricles are in the center part of the brain containing fluid. The cortex is the nerve cell on the outside of the brain, sending wiring down and through. *Id.* The periventricular area consists of wiring within the brain. *Id.* The bilateral scarring of the blood vessels in periventricular leukomalacia due to injury of those immature, fragile blood vessels is not an embolic phenomenon. Tr. at 60.

Devon is globally developmentally delayed as a consequence of her periventricular leukomalacia. *Id.* Her Down syndrome is also associated with her global developmental delay. Tr. at 60-61. Dr. Renfroe could not attribute Devon's monoplegia in her left leg, at first dragging her toes and then walking on her toes, to her Down syndrome or her periventricular leukomalacia. Tr. at 62. Devon's upper motor lesion will always be there which is why Botox is just a temporary solution to her spasticity, lasting three months for each application. Tr. at 64. Dr. Renfroe recommended tendon lengthening, rather than tendon severing, when Devon is five or six years old. Tr. at 64-65.

Dr. Renfroe admitted on cross-examination that Dr. Balchunas, a neuroradiologist, concluded after reviewing Devon's MRI scans that they did not represent a postvaccinal injury pattern. Tr. at 67. Dr. Balchunas did not agree with either Dr. Renfroe or Dr. Bodensteiner. Tr. at 68. Dr. Balchunas felt that Devon's problem was most likely periventricular leukomalacia. *Id.* Devon appeared to have something that preceded whatever caused the monoplegia. Dr. Balchunas did not say he saw evidence of emboli in Devon's brain. Tr. at 69.

It took until the end of January 2003 for Devon's left foot dragging to become spastic, a time of four months. Tr. at 76. To Dr. Renfroe, that fits the normal transition of an injury. Tr. at

77. But the transition time of four months seemed to Dr. Renfroe to be extended. *Id.* It should more likely have taken a month or two months. *Id.* Devon's case deals with both a peripheral nerve (lower motor neuron) and central nervous system nerve (upper motor neuron) injury. Tr. at 78.

Dr. Renfroe testified that he is not sure where Devon's lesion is. It could be in the cervical or thoracic spine or in the brain, anywhere above the lumbar spinal cord. Tr. at 80. The lesion could be small enough in the brain not to be detected or hidden among all the other problems in Devon's brain. *Id.* The average age for noticing asymmetry in a child according to the literature is 18 months. Tr. at 82.

Dr. Renfroe said that Devon had loss of muscle strength and weakness of her left ankle acutely that caused her toe drop within two to three days of her vaccination. Tr. at 84-85. Tone does not address strength. Tr. at 85. A Down syndrome child is diffusely floppy or low tone. Tr. at 86. Devon was walking flat footed with assistance before the vaccination *Id.* Something occurred to Devon's peripheral nerve or that anterior horn cell that affected her muscles and she started dropping her foot, meaning that the muscles in her anterior leg did not pick that foot up adequately. *Id.* A lesion that affects the wiring that controls the lower motor neuron may be in the cervical, thoracic, or lumbar spinal cords. The weakness in the foot is due to an injury to the nerves that go to those muscles. Tr. at 87. Because this is a demyelinating process, the electrical signals to the muscles are not properly conducted. *Id.*

Devon followed a progression that suggests injury to an area of her central nervous system. The initial clinical presentation was weakness of her leg and dragging her foot. Tr. at 88. Over time, as her nervous system healed, scarring resulted in a blockage of control of that

lower motor neuron which healed, resulting in spasticity. *Id.* When Dr. Goldstein tested Devon's deep tendon reflexes in her left leg, she had 2 plus reflexes. *Id.* Dr. Renfroe has seen in his practice a case of spinal inflammatory process, which he assumed was a transverse myelitis, resulting in right arm monoplegia. Tr. at 90.

Dr. John B. Bodensteiner, a pediatric neurologist, testified for respondent. Tr. at 92. His specialties are cerebral palsy and neuromuscular disease. Tr. at 95. Devon has cerebral palsy. Tr. at 93. Dr. Bodensteiner testified that Devon has a lesion in her cerebrum, in the white matter of her brain, which anatomically accounts for her monoparesis of the left leg. Tr. at 94. We do not know when Devon's cerebral lesions that are visible on the MRI occurred, but it is a great deal more likely that they are due to embolization of material from a right to left shunt in Devon's heart than from an undocumented autoimmune response involving the spinal cord following the immunizations. Tr. at 96.

Dr. Bodensteiner reviewed Devon's two cerebral MRI scans and two lumbar spinal MRI scans both alone and with three different pediatric neuroradiologists. Tr. at 97. He identified a particular lesion in her brain scan that is responsible for her monoplegia. *Id.* The lesion is in Devon's cerebral white matter, just underneath the right motor strip which is atypical for periventricular leukomalacia (PVL). *Id.* Although it is in the white matter adjacent to the ventricles, it is not a PVL lesion. *Id.* It is a focal lesion which has the characteristic of a vascular lesion and is directly anatomically underneath the motor strip. It is fairly high in the white matter, which is why it does not involve the arm because the fibers from the cerebral cortex that move the arm come out from a more lateral position than the fibers that move the legs. *Id.* This lesion is exactly in the right location to cause monoparesis. Tr. at 97-98. We do not know when

this lesion occurred, but since Devon had a right to left shunt in the heart, the most common cause would be embolization from the heart or coming through the heart and bypassing the lungs. Tr. at 98. This lesion is not demyelinating. Tr. at 99.

Rather than being a demyelinating process, it is a vascular lesion. Tr. at 100. On the scan, Devon had four to five vascular lesions which are wedge-shaped. Tr. at 101. One was directly under the motor strip which caused her monoplegia. *Id.* Dr. Bodensteiner disagreed with the pediatric cardiologist Dr. Mehta with whom Dr. Renfroe consulted who said the flow was left to right. Dr. Bodensteiner said the blood flow was right to left in Devon's heart. Tr. at 102. He knows this to be so because Devon was cyanotic which would not happen if the blood were flowing from left to right but only if the blood was going from right to left and bypassing her lung. Tr. at 102-03. Cyanotic means blue. Tr. at 103. In addition, Devon had pulmonary hypertension which means that the pressure on the right side was too high and the pressure was not as great on the left. *Id.*

Dr. Bodensteiner does not know when the lesion in Devon's brain occurred, but it came more likely from the heart than from the vaccinations for several reasons. Tr. at 106. It seems outrageously unlikely to say Devon had these lesions with this much residual damage without an encephalopathy. *Id.* He has, in 35 years of practice, never seen a transverse myelitis affecting just one limb without some residual evidence on the MRI of the spinal cord. *Id.* Cardiogenic emboli are the most common causes of stroke in children. *Id.*

Devon's PVL lesions were too mild to have clinical manifestations. Tr. at 107. It is possible that the dragging of the toes manifested increased rather than decreased tone, but we will never know the cause of the dragging of Devon's toes. Tr. at 109. Devon's monoplegia

probably occurred prior to the repair of her right to left shunt since afterwards there was a greatly diminished possibility of embolization. Tr. at 110. We will never know precisely when the lesion occurred. *Id.* There was a shower of emboli because we see multiple lesions. The one causing the monoparesis is directly under the motor strip. Tr. at 110-11. We do not see emboli in Devon's other organs because no one looked for them, and these other organs have collateral circulation unlike the brain. Emboli would not cause an infarct with collateral circulation, whereas the brain has end arteries and no collateral circulation. Tr. at 111.

We do have a shower of emboli in her brain because Devon has multiple lesions there, at least four, maybe five, lesions compatible with embolization in her brain. *Id.* The brain takes 40 percent of the cardiac output of the heart in Devon's age group. If you have emboli, they are more likely to go to the brain than anywhere else, and these are all in the middle cerebral artery distribution. Tr. at 112. The middle cerebral artery is the largest flow of the cerebral arteries. If someone has an embolus, it is more likely to go into the middle cerebral artery than anywhere else. *Id.* Down syndrome children frequently have heart defects and the heart defect causes them to have strokes. *Id.* A relatively small percentage of Down syndrome children have heart defects. And of those with heart defects, a relatively small number have emboli. Tr. at 113.

Dr. Renfroe testified that Devon's brain MRI shows evidence of injury to the white matter, which is the wiring of the brain. Tr. at 117. At this point, both Dr. Bodensteiner and Dr. Renfroe had the December 24, 2005 brain MRI on their computer screens, looking at image 17 of 24 for the T-2 axial image and image 14 of 27 on the coronal. Tr. at 123. Dr. Renfroe saw a triangular shape in the lesion on image 14 of 27 and asked Dr. Bodensteiner what vascular distribution that would be and why it stopped at the cortex. In other words, why was this solely a

white matter process. Tr. at 124. Dr. Bodensteiner responded that the white matter received a lot of vascular supply, too, not just the gray matter. In the immature brain, the white matter received relatively more vasculature than the gray matter compared to what occurred in older people. Both the lesions the doctors were looking at on their computer screens were in the white matter as were the two others on the left side of Devon's brain. They were all in the distribution of the middle cerebral artery. *Id.*

Dr. Renfroe asked a second question, looking at image 17 of 24, which showed a triangular-shaped lesion extending with its narrow point to the ventricle, becoming wider, but stopping outside the gray matter of the brain. Tr. at 125. He was unaware of any vascular distribution that would isolate itself to the white matter instead of traveling to the gray from the white. *Id.* Dr. Bodensteiner said the lesion did not reach the gray matter because it was not that big. He could tell from looking at this lesion that it was embolic and not typical of PVL. Tr. at 126.

Dr. Renfroe said that if we were going to say the lesions were embolic, they needed to fit an embolic anatomic pattern and he was unaware of any embolic anatomic pattern that this would fit. Tr. at 127. The lesion went directly to the gray-white junction or border, and abruptly stopped. He was unaware of a vascular pattern that would cause that. *Id.*

Dr. Bodensteiner said we know that the vasculature in the white and gray matters changes from infancy until later in childhood. We see maturation and development of the blood vessels in the germinal matrix region, for example. *Id.* The pattern of Devon's lesions suggests that they occurred fairly early in her life. Tr. at 128. Dr. Renfroe stated that all the lesions Dr. Bodensteiner discussed were isolated white matter. He wanted to know why they spared the gray

matter. Tr. at 131. Dr. Bodensteiner replied that they do not spare the gray matter. There was a lesion on the other side that involved gray matter. But the vasculature that supplied the white matter was relatively separate from the vasculature that supplied the gray matter. One could embolize those vessels just as easily as the gray matter. *Id.*

Dr. Bodensteiner stated that Devon's lesions are vessels that are smaller than namable vascular patterns. They concern end arteries and the white matter has plenty of vasculature, more in the infant than older children have, and there is gray matter involvement. Tr. at 132. If one looked at the coronal image, and the opposite side in the superior temporal gyrus, one would see gray matter involved, and probably another lesion which involved gray matter. Tr. at 132-33. Thus, there is a vascular pattern that satisfies the concept that an embolus could cause this lesion. Tr. at 133.

Dr. Bodensteiner considered Devon's spasticity as relatively mild. You would not notice the discrepancy between the use of her two legs until Devon got to the point where you would expect her to walk fairly well. Tr. at 136. Devon would have been expected to walk well at 36-37 months, and that would be when one would notice the discrepancy between one leg and the other. *Id.* That puts us right in the time frame of what occurred here. Tr. at 137.

For Devon to have a fresh lesion at the end of September and given Botox by the first of March or April is unheard of in Dr. Bodensteiner's experience. *Id.* Even though she had a lesion which might develop into spasticity, for it to develop far enough for her to have actual shortening of the heel cords and be up on her toes would take far longer than six months. It would take a couple of years. *Id.* Dr. Bodensteiner meant that the lesion near the motor strip must have preceded the vaccinations by a year in order for Devon to have enough spasticity to result in

shortening of the heel cord of the gastrocnemius tendon to necessitate Botox injections. Tr. at 137-38. Spasticity would have been there within about eight weeks after the injury, but not enough to necessitate Botox. Tr. at 138.

If the lesion or injury had occurred at the end of September, Devon was very unlikely to have developed a tight heel cord by January such that she required Botox in the Spring. *Id.* Ms. Van Uum corrected the record by stating that Devon did not receive Botox until July 2003. Tr. at 139. Dr. Renfroe stated that, in cases of transverse myelitis or traumatic spinal cord injury, he frequently used Botox within days of the insult to the spine or brain and did not wait for shortening of the heel cord which was a more permanent issue. Tr. at 139-40.

Dr. Bodensteiner said there was no way to tell when the lesion in the motor strip occurred, but by the time the brain MRIs were done, it was an old injury. Tr. at 140. He cannot say the lesion in the motor strip was present in September or October 2002. *Id.* He believes the lesion developed before the spasticity at the end of January 2003 developed. Tr. at 141. The spasticity probably occurred considerably before January 2003. *Id.* There is no evidence that Devon had spasticity before the vaccinations, but no exclusion of it either. Tr. at 143. Devon's onset of spasticity was at the age of 22 months, before the age of three years that Dr. Bodensteiner had described earlier, but he said if the injury had occurred at birth, one would not expect to see its manifestation until some time after one year. Tr. at 144.

The medical records include a cardiac ultrasound examination on May 31, 2001 that indicate Devon had an abnormal heart with a right to left shunt and cyanosis (page 153 attached to the petition). Tr. at 146. Page 155 states that Devon had a large atrioventricular septal defect with bi-directional shunts and a large patent ductus arteriosus with right to left shunt. Tr. at 148.

Either one or both could be the source of the emboli. Tr. at 149. The bi-directional shunt in the atria probably reflects pulmonary hypertension so that when the pulmonary blood flow was decreased or low, the blood went in the correct direction. *Id.* When the pulmonary blood flow was high, the blood went in the opposite direction. But there was the possibility of right to left shunt in both heart areas. *Id.*

Dr. Renfroe asked if the patent ductus arteriosis were not a fetal remnant of the arteries connected to bypass the lungs not uncommonly seen in this group which might close on its own or need surgical ligation but was not a cardiac abnormality. *Id.* Dr. Bodensteiner agreed that the PDA was not a cardiac abnormality. He had been thinking of a patent foramen ovale. Tr. at 149-50. But Devon still had unoxygenated blood going into the arterial side. Tr. at 150. "Unoxygenated" means it had not been through the lungs and, therefore, particulate matter had not been filtered out, and became the source of the emboli. *Id.* Forty percent of it would go to the brain. *Id.* Devon was not blue enough to necessitate surgery right away and could wait two years for the surgery. *Id.*

Dr. Bodensteiner said this heart condition is seen much more frequently in Down syndrome children than in other children. Tr. at 151. Any textbook on Down syndrome describes midline heart defects and a variety of types. *Id.* Devon had emboli on both sides of her body. *Id.* Dr. Bodensteiner has had many patients with similar symptoms. *Id.* The usual occurrence was that the stroke was larger than Devon's and the child had a hemiparesis and not just a monoparesis; specifically, the arm or the leg was more severely involved. Tr. at 152.

Dr. Renfroe stated that Devon had a preoperative report saying she had normal pulmonary artery pressure. Tr. at 154. He thinks Devon did have some shunting of blood. *Id.* Dr. Renfroe

was concerned about the time frame for the development of spasticity. Both he and Dr. Bodensteiner see children who suffered in utero injuries and developed cerebral palsy, which means an abnormality in the brain causing motor dysfunction. Tr. at 155. A person skilled at evaluating such a child would see spasticity much earlier than the 18 to 22 months in Devon's case. *Id.* He frequently sees spasticity in the neonatal intensive care unit. A good physical therapist would not document flat-footed walking in a child who has spastic monoplegia. *Id.* Devon has lesions that were probably caused in utero. Cerebral palsy can also be hypotonic. Tr. at 156. Dr. Renfroe said he thought he could call Devon's motor problem cerebral palsy. *Id.*

Dr. Renfroe thought that the wedge-shaped lesion on images 14 of the axial and 17 of the coronal areas anatomically fit as the cause of Devon's monoplegia. Tr. at 157. But he was not certain that that lesion caused her monoplegia. *Id.* The lesion is in an area that underlies the motor cortex. However, it tends to lateralize more and, as Dr. Bodensteiner pointed out, the lateral aspects there are not the leg, but are the body and the arm. It certainly underlies other areas that would not in isolation affect the leg. So he was at a loss to explain why Devon has such an isolated leg monoparesis. Tr. at 157-58.

Dr. Renfroe tried to explain his difficulty here by saying that the lesion was in an area that could affect the left motor function but he could not get an isolated left monoparesis out of it. Tr. at 158. Devon's periventricular leukomalacia is patchy, and there are several PVL lesions. *Id.* While Devon definitely has a white matter lesion, it abruptly stops at the motor cortex. Tr. at 159. He thinks an embolus was not the cause. *Id.* The other organ systems may not clinically manifest emboli as readily as the brain. Tr. at 160. Dr. Renfroe's problem with the emboli theory is he does not know when Devon had this embolic event. The hemodynamics in utero did

not seem to set her up for in utero strokes when she had PVL occurring. Tr. at 160-61. So Dr. Renfroe assumed, if Devon had strokes, they occurred after her birth, but he does not know when. Tr. at 161. PVL is due to immature blood vessels near the ventricles being challenged by either a drop in blood pressure or by systemic blood pressure causing them to rupture and scar due to their immaturity. *Id.* Dr. Renfroe would expect that if Devon developed lesions not due to PVL but to emboli, she would have manifested some clinical signs of them before 18 or 21 months. Tr. at 162. Dr. Renfroe's opinion was still that the lesion causing Devon's monoplegia could be in the brain and could be the lesion Dr. Bodensteiner identified as due to an embolus. Tr. at 162-63. All he could say was that the lesion was above the level of the deficit. Tr. at 162.

Devon does not have acute disseminated encephalomyelitis (ADEM). Dr. Balcunas specifically stated that. Tr. at 163. Dr. Balchunas also stated Devon did not have embolic phenomena. *Id.* Devon may have a form fruste or mild myelitis. Tr. at 164.

Dr. Bodensteiner responded that Devon's holes in her heart were a major risk factor, and emboli do not require pulmonary hypertension or even a right to left shunt all the time because of changes in various pressures from bearing down, crying, holding her breath, sneezing, or coughing. Tr. at 165. The lesion of interest does not have to go into the motor cortex. It involves the fibers that come from the motor cortex and proceed down the spinal cord. *Id.* You can interrupt those fibers just as if you had a spinal cord lesion. *Id.* You do not interrupt the cortex or damage it. The fact that the lesion does not involve the gray matter in that area means absolutely nothing respecting its causing the monoparesis. Tr. at 166. Dr. Bodensteiner did not know when this cardiogenic injury occurred. There were no careful examinations of Devon before her vaccinations. *Id.* The lesions do not look like acute disseminated encephalomyelitis

(ADEM) even though an ADEM-type of lesion could occur in this location resulting in this monoparesis. Tr. at 167. Dr. Bodensteiner thought the emboli occurred much earlier than the vaccinations. *Id.* It was Devon's lack of or delay in maturation which made it hard to identify earlier. *Id.*

**POST-HEARING BRIEFS**

Both parties submitted post-hearing briefs. Petitioners summarized the testimony and the record in their Closing Argument. They argue that "to give greater weight to the respondent's theory is to minimize or discount entirely the timing of Devon's injury, the logical reasoning of Dr. Renfroe for the cause of the injury, and overlook the opinions of Devon's treating physicians . . . ." P. Closing Arg. at 8.

Respondent argues that petitioners have failed to establish by a preponderance of the evidence that Devon's left leg injury is vaccine-related. Respondent states that prior to the vaccination, Devon was never able to walk on her own and even when aided, she never had a heel strike. R. Post-Hearing Brief at 3. Further, respondent states that when the physical therapist, Ms. Rimko, saw Devon on July 11, 2007, two months before the vaccinations, she noted that Devon actively bent and straightened both legs with a preference to the right. *Id*. at 3. When Ms. Rimko saw Devon six days following the vaccinations, she did not mention in her notes anything about Devon's left leg deficit. *Id.* Again, when Ms. Rimko saw Devon on October 17, 2002, she did not note any foot dragging, but did note that the goals for that day were to facilitate Devon's movement in a correct pattern of the left foot with advancement in gait. *Id*. at 3-4. Although IR (internal rotation) of the left foot was observed, Ms. Rimko stated that Devon was able to correct this with tactile cues at the hip and foot. *Id*. at 4. Respondent argues

that Dr. Renfroe's testimony and opinion rest largely on the lack of prior left leg deficit and the temporal proximity between the shot and the dragging of the left leg. Respondent concludes that petitioner's case is based on timing and lack of alternative cause.

**DISCUSSION**

To satisfy their burden of proving causation in fact, petitioners must offer "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." Althen v. Secretary of HHS, 418 F. 3d 1274, 1278 (Fed. Cir. 2005). In Althen, the Federal Circuit quoted its opinion in Grant v. Secretary of HHS, 956 F.2d 1144, 1148 (Fed. Cir. 1992):

> A persuasive medical theory is demonstrated by "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury[,]" the logical sequence being supported by "reputable medical or scientific explanation[,]" *i.e.*, "evidence in the form of scientific studies or expert medical testimony[.]"

In Capizzano v. Secretary of HHS, 440 F.3d 1317, 1325 (Fed. Cir. 2006), the Federal Circuit said "we conclude that requiring either epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect is contrary to what we said in Althen...."

Close calls are to be resolved in favor of petitioners. Capizzano, 440 F.3d at 1327; Althen, 418 F.3d at 1280. *See generally*, Knudsen v. Secretary of HHS, 35 F.3d 543, 551 (Fed. Cir. 1994).

Without more, "evidence showing an absence of other causes does not meet petitioners' affirmative duty to show actual or legal causation." Grant, 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 show not only that but for the vaccines, Devon would not have had monoplegia, but also that the vaccines were substantial factors in bringing about her injury. Shyface v. Secretary of HHS, 165 F.3d 1344, 1352 (Fed. Cir. 1999).

In essence, the special master is looking for a medical explanation of a logical sequence of cause and effect (Althen, 418 F.3d at 1278; Grant, 956 F.2d at 1148), and medical probability rather than certainty (Knudsen, 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.

The Federal Circuit stated in Althen, 418 F.3d at 1280, that "the purpose of the Vaccine Act's preponderance standard is to allow the finding of causation in a field bereft of complete and direct proof of how vaccines affect the human body."

The Federal Circuit in Capizzano emphasized the opinions of petitioner's four treating doctors in that case. 440 F.3d at 1326.

As the Federal Circuit stated in Knudsen, 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." The undersigned's task is to determine medical probability based on the evidence before the undersigned in this particular case. Althen, 418 F.3d at1281 ("judging the merits of individual claims on a case-by-case basis").

First, respondent appears to question Devon's left foot dragging two to three days after the vaccination. R. Post-Hearing Brief at 3-5. However, Mrs. Van Uum impressed the undersigned as a credible witness and extremely attentive parent. During Devon's November 7, 2007 visit to Dr. Ballock, Mrs. Van Uum gave a history that Devon had been dragging her left foot two months previously. Med recs. at 248. The undersigned, therefore, accepts that Devon's foot dragging began shortly after the vaccinations.

The expert testimony in this case from two highly competent pediatric neurologists differed dramatically. Dr. Renfroe, who works in a spasticity clinic, stated that acellular pertussis vaccine and/or inactivated polio vaccine caused Devon's monoplegia by forming a small lesion starting in the lower motor neuron area and then advancing to the upper motor neuron area because of scarring during the healing process of the lesion. Although he initially placed the lesion in the cervical or thoracic spinal cord areas, after hearing Dr. Bodensteiner's testimony, he agreed the lesion could also be in Devon's brain near the motor strip. Although Dr. Renfroe thought that the four-month process from Devon's walking on her left toes to Devon's left leg

spasticity was longer than he would have thought appropriate, he still opined that Devon's permanent left leg spasticity was a residuum of her vaccine injury.

Devon's treating pediatric neurologist, Dr. Edward M. Goldstein, on December 31, 2003, diagnosed Devon with a post-dysimmune reaction to vaccinations with residual left lower extremity monoparesis. Respondent argues that "there is no evidence that [Dr. Goldstein's] impressions are based on anything other than petitioners' history and belief." R. Post-Hearing Brief at 12. However, Dr. Goldstein's notes also state that his conclusion that Devon had a post-dysimmune reaction to the vaccination was based on Devon's history as well as her general physical and neurological examinations. Med. recs. at p. 312. The undesigned is also impressed with Dr. Renfroe's description of Dr. Goldstein as having given Devon her "first good neurologic examination" and a "wonderful evaluation." Tr. at 60. That makes Dr. Goldstein's diagnosis of a post-dysimmune reaction to vaccinations more significant to the undersigned because he has an impressive ability and thoroughness.

Respondent's expert, Dr. Bodensteiner, thought Devon's monoplegia was not due to a vaccine reaction but rather to an embolus that the wedge-shaped lesion in her brain represented. It was in the appropriate place in the brain to cause motor difficulties. Devon, having holes in her heart, was at major risk for emboli due to blood shunting through the heart without proper oxygenation and removal of particulates from it because the blood never reached her lungs. He could not say when the embolus at issue broke off, but it would have been much earlier than her 18-month vaccinations. He stated, "[Y]ou won't notice the discrepancy between the use of the two legs until the child gets to the point where you would expect them [sic] to walk fairly well." Tr. at 136. Because of Devon's Down syndrome, Dr. Bodensteiner would not expect Devon to

walk well until 36-37 months, which is "when you would start to see the discrepancy between one leg and the other in a child with a relatively mild spasticity." *Id*. In essence, he considered that Devon's dragging her left toes two to three days after her vaccinations was coincidental.

Dr. Bodensteiner opined that the emboli near the motor cortex caused her spasticity. Dr. Renfroe believed that had Devon's spasticity been present prior to the vaccination, it would have been observed because she was under close and regular scrutiny not only by her parents, but by her physical therapist. P. Closing Arg. at 6.

The undersigned has the disadvantage of picking between two excellent experts with divergent opinions. Both are eminently qualified to testify about the case. There is a third opinion, however, from Dr. William Balchunas, a neuroradiologist, that Devon's brain MRIs do not show either an acute vaccine injury or the presence of emboli. Dr. Balchunas's conclusions may support Dr. Renfroe's testimony that the small lesion affecting Devon's left leg was in the cervical or thoracic spinal cord, both areas which were never scanned with an MRI. The only spinal MRI performed on Devon was of her lumbar spinal region. Dr. Balchunas's opinion notably does not support Dr. Bodensteiner's testimony that the brain MRIs show emboli.

The Federal Circuit has emphasized that, in close cases, petitioners should prevail, and this is a close case. The Federal Circuit has also emphasized that the special masters should take into consideration the opinions of treating doctors, and here Devon's treating pediatric neurologist Dr. Goldstein opined that Devon's monoplegia was due to an adverse reaction (the word he used was "post-dysimmune") to her vaccinations. Further, due to Dr. Renfroe's praise of Dr. Goldstein's abilities, the undersigned knows that Dr. Goldstein was the first doctor to give Devon a thorough and appropriate neurological examination. Based on the Federal Circuit's two

principles above enunciated ((1)in a close case, petitioners prevail, and (2) the undersigned must take into consideration the opinion of treating doctors), the undersigned holds that petitioners have made a prima facie case of causation in fact and that Devon's vaccinations administered when she was 18 months old caused her monoplegia even if the evidence does not show precisely how or why this occurred or the specific biologic mechanism. This holding is in accordance with the Federal Circuit's principle stated in Knudsen, 35 F.3d at 549, that this court was "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" and that petitioners do not have to identify and prove specific biologic mechanisms in order to prevail.

Acellular DPT vaccine and/or inactivated polio vaccine caused a lesion that partially obstructed the signals to Devon's lower motor area, causing her left leg spasticity or monoplegia, and without her having received the vaccine or vaccines, she would not have had this injury.

**CONCLUSION**

Petitioners are 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 how to proceed to resolve the issue of damages.

**IT IS SO ORDERED.**

_____ _____

DATE Laura D. Millman
Special Master