In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 08-0266V
Filed: June 6, 2013

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MARIA CARRINO, Spouse	*	
and Executrix of the Estate of	*	Influenza Vaccine; Alleged
SAMUEL CARRINO,	*	Guillian-Barré Syndrome;
	*	Documented Diagnosis of
Petitioner,	*	Coronary Artery Occlusion With
v.	*	Cerebral Infarction; Weight of
	*	Record Evidence Contravenes
	*	Claimed Injury
SECRETARY OF HEALTH	*	
AND HUMAN SERVICES,	*	
	*	
Respondent.	*	
* * * * * * * * * * * * * *		

Thomas Gallagher, Somers Point, NJ, for petitioner

Darryl Wishard, Washington, DC, for respondent

DECISION[1]

On April 14, 2008, Samuel Carrino filed a petition for compensation under the

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[1] Because this decision contains a reasoned explanation for the undersigned's action in this case, the undersigned intends to post this decision on the United States Court of Federal Claims' website, in accordance with the E-Government Act of 2002, Pub. L. No. 107-347, § 205, 116 Stat. 2899, 2913 (codified as amended at 44 U.S.C. § 3501 note (2006)). As provided by Vaccine Rule 18(b), each party has 14 days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, "the entire" decision will be available to the public. Id.

National Childhood Vaccine Injury Compensation Program (the Program).[2] He first alleged that he developed left-sided paralysis and required hospitalization five days after he received the influenza vaccine on October 6, 2006. Petition; Petitioner's Exhibit (Pet'r's Ex.) 8 at 14. Petitioner amended his claim on February 27, 2009 to allege that the flu vaccine he had received in October 2006 caused him to develop Guillain-Barré syndrome (GBS). See Amended Petition. Mr. Carrino died on August 1, 2009.

Three months later, Mr. Carrino's widow, Maria Carrino (Mrs. Carrino), filed a second amended petition as his spouse and the executrix of his estate. She alleged that the flu vaccine caused her husband's GBS event as well as his subsequent death. Second Amended Petition at 1.

Respondent moved for dismissal of petitioner's claim, in part, because there was insufficient proof that Mr. Carrino had suffered a GBS event. See Mot. to Dismiss and Rule 4(c). The undersigned stayed the motion pending further briefing on the relevant issues. A telephonic status conference was conducted on November 8, 2010, to discuss the lack of record support for Mr. Carrino's claimed injury. Order at 1, Nov. 10, 2010. Petitioner's counsel argued that the nature of Mr. Carrino's injury was a genuine issue of material fact that precluded dismissal and requested an expert hearing. Id.

During the pendency of petitioner's claim, she filed a letter in support of vaccine-related causation from Joseph DeMayo, M.D., Mr. Carrino's primary care provider, Pet'r's Ex. 12, and three expert reports from Victor Hogen, M.D., a neurologist. Pet'r's Exs. 15, 19, 21. Respondent, in turn, filed three expert reports from Martin Bielawski, M.D., a neurologist. Resp't's Ex. A, N, Q.

An entitlement hearing was held in Washington, D.C., on May 17, 2011. Mr. Carrino's treating physician, Dr. DeMayo, and both parties' experts testified. The parties disagreed on the nature of Mr. Carrino's injury and whether the injury was vaccine-related.

Following the hearing, the parties were afforded an opportunity to explore the possibility of informally resolving the claim. Order, June 15, 2011. Unsuccessful in their efforts, the parties subsequently filed post-hearing briefs. Id. This matter is now ripe for a ruling.

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[2] The Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 et seq. (hereinafter "Vaccine Act" or "the Act"). Hereafter, individual section references will be to 42 U.S.C. § 300aa of the Act.

At the core of the parties' dispute regarding entitlement is a disagreement regarding whether Mr. Carrino developed GBS or suffered a cerebrovascular accident. Mrs. Carrino maintains that Mr. Carrino endured a debilitating episode of GBS that was caused by the flu vaccine he received. Respondent contends that Mr. Carrino did not develop GBS, but suffered a stroke. The preponderant evidence does not support a finding that Mr. Carrino suffered from a GBS event. There is no diagnosis of GBS in the records from Mr. Carrino's hospitalization in the days after his flu vaccination. The mention of a possible flu-related GBS episode does occur, however, in subsequent hospitalization records as part of Mr. Carrino's past medical history. On this record, petitioner has failed to satisfy her burden of proving that Mr. Carrino suffered a vaccine-related injury and thus, her claim must be dismissed.

I. Factual Background

A. Mr. Carrino's Pertinent Medical History

1. His pre-vaccination health

Mr. Carrino was born in March of 1928. Pet'r's Ex. 1 at 1. His medical history was significant for non-insulin-dependent diabetes, hypertension, coronary artery disease,[3] a lung lobectomy, myocardial infarction,[4] multiple cranial neuropathies,[5] a stroke with right-sided weakness, diabetic polyneuropathy,[6] and former tobacco use. Pet'r's Ex. 8 at 1, 27, 32-33, 55; Pet'r's Ex. 5 at 9.

In May of 1999, Mr. Carrino was evaluated for a five-day history of double vision. Pet'r's Ex. 5 at 12. He was 71 years old. Id. The examining neurologist noted Mr.

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[3] Coronary artery disease is caused by hardening of the coronary arteries; the condition can lead to angina pectoris, myocardial infarction, or sudden death. Coronary artery disease, http://www.dorlands.com/def.jsp?id=100030680 (last visited June 5, 2013).

[4] Myocardial infarction occurs when the blood supply to the heart is interrupted, usually as a result of arterial hardening, and heart muscle dies. Myocardial infarcation, http://www.dorlands.com/def.jsp?id=100053424 (last visited June 5, 2013).

[5] A cranial neuropathy occurs when any of the twelve cranial nerves that exit from the brain are damaged. Cranial neuropathy, http://www.emedicinehealth.com/neuropathy/article_em.htm (last visited June 5, 2013).

[6] A diabetic polyneuropathy is diabetes-induced nerve damage that affects either the sensory, motor, or autonomic nerves, or a combination thereof. Diabetic neuropathy, http://www.dorlands.com/def.jsp?id=100071988 (last visited June 5, 2013).

Carrino's history of hypertension and diagnosed him with "a probable diabetic right sixth nerve palsy"[7] and "a mild diabetic peripheral neuropathy."[8] Id. at 13.

Magnetic resonance imaging (MRI) of Mr. Carrino's brain performed two days later revealed ischemic changes (which are changes in the blood vessels). Pet'r's Ex. 5 at 21. Additional testing showed an occlusion of his left vertebral artery. Id. at 20.

Four months later, a neurologic examination showed that Mr. Carrino was exhibiting symptoms of diabetic neuropathy. Among the symptoms observed were missing right ankle reflexes[9] and an inability to feel vibrations in his toes. Pet'r's Ex. 5 at 9.

Two years later, in January of 2002, Mr. Carrino again developed double vision. Pet'r's Ex. 5 at 7. Imaging of his brain showed age-related white matter changes and a small, old, left-sided cerebellar infarction (an area of tissue death). Pet'r's Ex. 5 at 15-16.

Mr. Carrino was evaluated the following year, in April of 2003, by another neurologist, for an episode of syncope that had occurred a month before his examination. The neurologist detected right arm weakness and diagnosed a stroke in the left hemisphere of Mr. Carrino's brain. An MRI of Mr. Carrino's brain showed a recent hemorrhage in the left basal ganglia region and small areas of recent infarction in the left posterior parieto-occipital region. Pet'r's Ex. 8 at 58. Also evident from the MRI were old lacunar

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[7] Damage to the sixth cranial nerve is described as a sixth nerve palsy; such damage can cause abnormal movement of the eye and double vision. Sixth nerve palsy, http://www.hopkinsmedicine.org/healthlibrary/conditions/nervous_system_disorders/multiple_cranial_neuropathies_134,48/ (last visited June 5, 2013).

[8] Damage to the peripheral nervous system (known as a peripheral neuropathy) disrupts the vast communications network that transmits information from the brain and spinal cord to other parts of the body. See http://www.ninds.nih.gov/disorders/peripheralneuropathy/detail_peripheralneuropathy.htm (last visited June 5, 2013). The presenting symptoms are determined by the affected nerves, and such symptoms develop over a period of days, weeks, or years. Id. Muscle weakness is the most common symptom of motor nerve damage. Id.

[9] The record indicates that Mr. Carrino's loss of his ankle reflexes in 1999, seven years before he received the flu vaccine at issue, was attributed to his pre-existing diabetic polyneuropathy. Pet'r's Ex. 5 at 9; Pet'r's Ex. 8 at 57.

infarcts in the basal ganglia and brain softening (encephalomalacia)[10] in the lower parietal lobes, both findings that were consistent with an old ischemic injury. Ischemic changes also were detected in the brainstem. Pet'r's Ex. 8 at 39-40.

Five months later, in November of 2003, Mr. Carrino was admitted to the hospital with complaints of left upper eyelid pain and a chaotic and irregular heart rate (atrial fibrillation). Pet'r's Ex. 8 at 39. Imaging of his brain taken by a magnetic resonance angiogram,[11] Pet'r's Ex. 8 at 42, 44, showed no acute brain infarcts, Pet'r's Ex. 8 at 42, but did show moderate-to-severe stenosis of his left carotid artery and his right mid-vertebral artery. Pet'r's Ex. 8 at 44.

One month later, Mr. Carrino was evaluated by a neuro-ophthalmologist, for complaints of pain near his left eyebrow, a drooping eyelid, and double vision. Pet'r's Ex. 8 at 32-34. The findings were consistent with a diabetic or microvascular third nerve palsy.[12]

2. Mr. Carrino's flu vaccine and his subsequent hospitalization

Nearly three years later, on October 6, 2006, Mr. Carrino received the seasonal trivalent flu vaccine, an inactivated, injectable vaccine containing killed influenza virus. Transcript (Tr.) at 138; Respondent's Exhibit (Resp't's Ex.) W; Pet'r's Ex. 8 at 14. Dr. DeMayo, Mr. Carrino's primary care physician, administered the vaccine in his office. Tr. at 37.

Four days later, Mr. Carrino was overcome with sudden dizziness after rising from bed. Pet'r's Ex. 10 at 93. Although he was able to walk that morning, he lost his muscle coordination that evening. Id.

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[10] Encephalomalacia is the "softening of the brain," that results from an infarct. Encephalomalacia, http://www.dorlands.com/def.jsp?id=100035052 (last visited June 5, 2013).

[11] A magnetic resonance angiogram (MRA) is a type of MRI; it relies on the use of a magnetic field and pulses of radio wave energy to generate pictures of blood vessels inside the body. See Mosby's Manual of Diagnostic and Laboratory Tests (Mosby's Manual) at 1167 (4th ed. 2010).

[12] Third nerve palsy is a type of cranial neuropathy, associated with sagging or drooping eyelids, double vision, difficulty with eye movement, and larger pupils than normal. Third nerve palsy, http://www.hopkinsmedicine.org/healthlibrary/conditions/nervous_system_disorders/multiple_cranial_neuropathies_134,48/ (last visited June 5, 2013).

On October 11, 2006—five days after his flu vaccination—Mr. Carrino was admitted to Clara Maas Medical Center by his primary care physician, Dr. DeMayo. As noted in the contemporaneous medical records, he could not walk and was experiencing "vertigo, nausea, [and] weakness." Pet'r's Ex. 11 at 1. The documented diagnostic impressions at the time of his hospital admission were cerebrovascular accident (stroke), cerebral infarction,[13] and atrial fibrillation.[14] See id. at 1.

Dr. Brand, one of Mr. Carrino's attending physicians, conducted a neurologic exam the same day as his hospital admission. Pet'r's Ex. 10 at 93. Mr. Carrino reported a spinning sensation, id., and complained of feeling "as if [he were] being pulled to right." Id. But, his speech and vision were unaffected. See id. Dr. Brand noted a slight drooping of Mr. Carrino's right eyelid, paralysis of his right extremities, and uncoordinated movements (ataxia). Pet'r's Ex. 10 at 93. Mr. Carrino's heartbeat was irregular, and he felt nauseous. Id. While awake and alert, he needed support to sit. Id.

A cardiologist also examined Mr. Carrino. The cardiologist diagnosed him with sudden onset of atrial fibrillation. Pet'r's Ex. 10 at 94. The day after Mr. Carrino's admission, he began to complain of double vision. Pet'r's Ex. 11 at 3. His ataxia improved, and by the third day of his hospitalization, he no longer had double vision. Pet'r's Ex. 11 at 7. His left-sided numbness also had diminished. Id. at 8.

Mr. Carrino had three brain MRIs during his hospitalization—two of which were performed within the first 48 hours of Mr. Carrino's admission.[15] The first MRI, performed on the same day as his hospital admission, showed "no acute infarctions." Pet'r's Ex. 10 at 55. The second MRI was performed with and without contrast the next day. As read, this MRI showed no abnormal enhancement. Pet'r's Ex. 10 at 57.

Although Mr. Carrino's first two brain MRIs failed to confirm that he had experienced a stroke, the medical records indicate that his treating physicians—to include

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[13] A cerebral infarction (or cerebral ischemia) is "an ischemic condition of the brain [that] produc[es] local tissue death . . . usually [presenting as] a persistent focal neurological deficit in the [distribution] area of . . . one of the cerebral arteries." Cerebral infarction, http://www.dorlands.com/def.jsp?id=100053413 (last visited June 5, 2013).

[14] Atrial fibrillation is the most common type of irregular heartbeat. Atrial fibrillation, http://www.nhlbi.nih.gov/health/health-topics/topics/af/ (last visited June 5, 2013). It can increase the risk of stroke, particularly if another risk factor—such as high blood pressure—is present.

[15] The third MRI, which was performed more than three weeks later, showed abnormality that was consistent with a lateral medullary stroke.

both Dr. DeMayo and Dr. Brand—continued to view his symptomatology as consistent with a stroke.

On October 15, 2006, four days after Mr. Carrino's hospital admission, Dr. Brand speculated that Mr. Carrino had suffered a stroke event—that had caused his various symptoms—in the area of the brain close to the brainstem and cerebellum. Pet'r's Ex. 11 at 9 (emphasis added) (Dr. Brand remarking: "Patient has neurological signs consistent [with] posterior fossa ischemia."). Among the symptoms noted at that time were: (1) a more pronounced lack of coordination in Mr. Carrino's right arm than in his right leg; (2) diminished pain sensitivity on his left-side; (3) dizziness with movement, and (4) headaches and neck pain. Pet'r's Ex. 11 at 10.

Dr. DeMayo evaluated Mr. Carrino on October 16, 2006. Pet'r's Ex. 11 at 10. Dr. DeMayo noted that although Mr. Carrino had right-sided numbness and weakness, id., he was able to move all of his extremities. Id. He continued to experience dizziness with head movement, but indicated that his "headaches and vertigo" were improving. Id.

A swallow study performed two days later was normal. Pet'r's Ex. 11 at 16.

Many of the physicians who were consulted during Mr. Carrino's hospitalization noted that he was undergoing evaluation for a suspected stroke. See Pet'r's Ex. 11 at 14. (Dr. Edwin Amirata, an examining surgeon, noted that Mr. Carrino had presented with lower body paralysis and hiccups after "a new CVA"); Pet'r's' Ex. 10 at 95 (Dr. Daniel Manzi, a consulting gastroenterologist, indicated that Mr. Carrino was "receiving a neurologic workup for CVA").

3. Mr. Carrino's weakness worsened when he developed a urinary tract infection during his second week of hospitalization

Nine days after his hospital admission, Mr. Carrino developed a low-grade fever. Pet'r's Ex. 11 at 21. He continued to complain of difficulty swallowing, neck pain, and weakness in all of his extremities. Pet'r's Ex. 11 at 25. Dr. DeMayo, his primary care physician, ordered a urine culture. Id. at 21.

Dr. Brand, the attending neurologist, examined Mr. Carrino again on October 21, 2006. He detected greater weakness in both of Mr. Carrino's legs, pain in his right shoulder, and increasing weakness in his right arm. Pet'r's Ex. 11 at 27. Mr. Carrino's facial sensation and strength were intact. Id. But his eyes moved involuntarily when he gazed leftward, and he had double vision when he gazed rightward. Id. Noting that Mr. Carrino had received a flu shot several days before his symptom onset, Dr. Brand considered the possibility that Mr. Carrino had suffered a parainfectious-induced radiculopathy presenting as GBS. Pet'r's Ex. 11 at 32. Dr. Brand initiated a course of steroids and intravenous immunoglobulin (IVIG), a choice treatment for autoimmune

conditions. This documented concern expressed by Dr. Brand is the first mention of GBS in Mr. Carrino's medical records. Pet'r's Ex. 11 at 27.

That same day, Donald Beggs, M.D., an infectious disease specialist, also examined Mr. Carrino. He recorded Mr. Carrino's complaints of "ataxia, dizziness, neck pain [with] stiffness, [and] vision change," as beginning two days before his hospital admission and four days after he had received a flu shot. Pet'r's Ex. 10 at 99. Mr. Carrino developed a bilateral "ascending weakness," id., and lost his left-sided sensation after his hospital admission. The detection of an "[a]scending paralysis" provoked Dr. Beggs to consider the possibility of a GBS event—as had Dr. Brand earlier in the day. Dr. Beggs ordered diagnostic testing for sepsis and prescribed the antibiotic Levaquin.

Mr. Carrino was transferred to the intensive care unit in "progressively worsening condition" on October 22, 2006. Pet'r's Ex. 11 at 28. He had "bilateral leg weakness," and pain in his right eye and his neck. Id. at 29. Mr. Carrino began a course of antibiotics for what was determined later to be a bacterial urinary tract infection. Pet'r's Ex. 11 at 28, 39, 41, 49, 52.

The next day, Mr. Carrino had less pain, was better able to move his neck, and had no trouble swallowing. Pet'r's Ex. 11 at 35.

That same day, Mr. Carrino was evaluated by John Conti, M.D., a specialist in hematology and oncology. Dr. Conti indicated that Mr. Carrino "had been doing well [with his neurologic symptoms]," but had developed a "progressive worsening of his condition," that was marked—over a two day period—by more "weakness and stiffness." Pet'r's Ex. 10 at 102. Mr. Carrino's medical records showed that on hospital admission, he was found to have ischemia in the left parietal lobe of his brain and was deemed to have suffered a stroke. Id. at 102. Dr. Conti prescribed Coumadin, a blood thinner, for Mr. Carrino in an effort to reduce the risk of his irregular heartbeat causing another stroke. See id.

Later that same day, a lumbar puncture was performed to evaluate whether Mr. Carrino had an infection and to rule out GBS. Pet'r's Ex. 11 at 37. As the lumbar puncture study revealed, Mr. Carrino's cerebrospinal protein levels were normal and contained no evidence of infection. Pet'r's Ex. 10 at 31; Pet'r's Ex. 11 at 38.

Dr. Brand, the attending neurologist, ordered needle electromyography[16] (EMG)

[16] Electromyography requires the insertion of thin needle electrodes through the skin into the muscles to evaluate a subject's muscular health and the health of the nerves that control the muscles. Electromyography, www.nlm.nih.gov/medlineplus/ency/article (last visited June 5, 2013).

and a nerve conduction test to evaluate Mr. Carrino's nerve impulses and muscle control. See Pet'r's Ex. 11 at 38. The results of this EMG study were not filed into the record. Dr. Brand's finding that the EMG results were consistent with a peripheral neuropathy was filed; but this finding did not exclude the possibility that Mr. Carrino had suffered a Guillain-Barré type of radiculopathy.

On examination, Dr. Brand found that Mr. Carrino's upper limb reflexes were preserved and his knee reflexes had returned. His ankle reflexes, however, had not returned. Pet'r's Ex. 11 at 44. Mr. Carrino began a prescriptive therapy of Neurontin for pain, and steroids together with IVIG for inflammation and any autoimmune irregularities. Id.

Dr. Beggs, the infectious disease specialist, evaluated Mr. Carrino again on October 24, 2006. Pet'r's Ex. 11 at 41. He noted Mr. Carrino's urinary tract infection, spinal stenosis, and leg weakness. He remarked that a study of Mr. Carrino's cerebrospinal fluid did not point to any infection. Id.

Mr. Carrino's weakness continued to improve. Pet'r's Ex. 11 at 50. By October 26, 2006, fifteen days after Mr. Carrino's hospital admission, Dr. Brand commented that Mr. Carrino had returned to a condition similar to the state in which he had presented on admission, but without the dizziness. Pet'r's Ex. 11 at 64.

Dr. DeMayo's notes indicated that Mr. Carrino was able to move his neck and sit up in bed. Pet'r's Ex. 11 at 50. His notes also indicated that Mr. Carrino had developed "chronic" hiccups—which are notable symptoms that tend to present in subjects who have suffered a lateral medullary stroke. See id., see Resp't's Ex. E at 573; see What is Wallenberg's Syndrome? http://www.ninds.nih.gov/disorders/wallenbergs/wallenbergs.htm (last visited on Apr. 30, 2013). Id.

In the medical assessment plan dated October 27, 2006, Mr. Carrino's weakness was attributed to arthritis, not to a GBS event. Pet'r's Ex. 11 at 62.

Dr. Brand observed that Mr. Carrino had become "more comfortable" over a period of several days; but his neurologic findings after treatment with the intravenous gamma globulin (IVIG) and steroids (Solu-Medrol) were similar to his pre-treatment findings—which suggested that the treatment had not been effective. See Pet'r's Ex. 11 at 69.

The third MRI of Mr. Carrino's brain was performed on November 1, 2006. That imaging showed a "small focus of T2 signal abnormality and enhancement in the right lateral aspect of the brainstem at the cervicomedullary junction, most likely due to ischemia." Pet'r's Ex. 10 at 56. Dr. Brand read the MRI as confirming an "infarct of

lateral medulla . . . on admit." Pet'r's Ex. 11 at 75. It appears that after discussing the case further with the radiologist, Dr. Brand wrote:

> Patient's MRI's reviewed. There is a clear cut [right] lateral medullary infarct seen on several segments. Patient will require on-going [physical therapy]. [Out of bed with] help and rehabilitation. . . . Meyer features[17] remain clinically evident.

Pet'r's Ex. 11 at 77 (emphasis added).

4. Mr. Carrino's medical condition after his hospital discharge in November of 2006

Mr. Carrino was discharged on November 3, 2006 from the hospital to a continuing care facility for rehabilitation. See Pet'r's Ex. 2 at 6; Pet'r's Ex. 10 at 91-92. The discharge summary, authored by Dr. DeMayo, listed as the principal diagnosis: cerebral artery occlusion with cerebral infarction. Id. at 92. The discharge summary contained no mention of GBS. See Pet'r's Ex. 10 at 91-92.

On November 10, 2006, seven days after his admission to the rehabilitation facility, Mr. Carrino was evaluated for "shortness of breath." Pet'r's Ex. 2 at 5. His past medical history of "CVA, hypertension, coronary artery disease, and arrhythmia" was documented during the evaluation. Id.

Mr. Carrino remained in the rehabilitation facility for three months. He was discharged to his home from the facility on February 10, 2007. See Pet'r's Ex. 4 at 4.

The day after his discharge from the rehabilitation facility, Mr. Carrino required an emergent evaluation after he fell off a chair at home. Pet'r's Ex. 4 at 4. The hospital admission record, dated February 11, 2007, indicated that Mr. Carrino had been hospitalized for "bilateral lower extremity weakness possibly due to receiving a flu shot, as per patient's wife." Id. (emphasis added).

The next month, Mr. Carrino was admitted to the hospital again after falling away from his walker while at home and fracturing his right hip. The handwritten medical history for this admission—which was authored by Mr. Carrino's primary care physician,

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[17] This reference pertains to the Fugl-Meyer Assessment (FMA), a stroke-specific, performance-based impairment index. It is designed to assess motor functioning, balance, sensation and joint functioning in patients with post-stroke hemiplegia (the inability to move a group of muscles in one side of the body). Meyer features refer to a patient's Fugl-Meyer Assessment (FMA). Lisa Zeltzer, Fugl-Meyer Assessment of Sensorimotor Recovery After Stroke(FMA), StrokEngine, http://strokengine.ca/assess/module_fma_intro-en.html (last visited June 5, 2013).

Dr. DeMayo—listed "diabetes, [hypertension (HTN)] [coronary artery disease (CAD)], [status post (s/p)] Guillain-Barré, hemiparesis[, and] unsteady gait." Pet'r's Ex. 10 at 422. Notably, the dictated version of this same admission note, which was also prepared by Dr. DeMayo, does not list GBS among Mr. Carrino's prior health conditions. Id. at 421.

On December 23, 2007, Mr. Carrino presented to the emergency room by ambulance transport with altered mental status, facial droop, and an inability to provide any reliable information regarding his condition. Pet'r's Ex. 4 at 24. On admission, Mrs. Carrino insisted that Mr. Carrino had not been diagnosed with a cerebral vascular accident, but she acknowledged that his neurologist had informed her that Mr. Carrino may have had suffered such an injury in October 2006. Id.

Mr. Carrino required additional hospital admissions on a number of occasions between late 2007 and early 2008 after various falling episodes and for diverse complaints, including abdominal pain, chest wall pain, abnormally slow heart rate (bradycardia), pneumonia with sepsis, and respiratory failure. These various hospital admissions periodically listed GBS as a past medical condition. Pet'r's Ex. 10 at 250, 253, 422, 470.

Mr. Carrino died on August 1, 2009, at the age of 81. Pet'r's Ex. 17. GBS is not listed as an immediate cause of death on the death certificate.[18]

B. The Medical Literature

1. Petitioner's literature

Petitioner filed seven articles in this case. Only one article spoke to the pathogenesis of GBS in general; the remaining articles examined the incidence of and relationship between influenza vaccination and the subsequent occurrence of GBS.[19] See Pet'r's Ex. 28. None of the articles submitted by petitioner discussed the diagnostic criteria for or expected clinical presentation of GBS; nor did any of the articles discuss the recommended treatment for the condition. See generally Pet'r's Ex. 22-28. The literature filed by petitioner was of limited assistance to the undersigned in evaluating this claim. More helpful to the undersigned was the unrebutted literature filed by respondent.

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[18] The copy of the death certificate that was filed into this record does not appear to be the official one because the document is marked as VOID. See Pet'r's Ex. 17.

[19] R.A.C. Hughes et al., Pathogenesis of Guillain-Barré Syndrome, 100 J. of Neuroimmunology 74 (1999).

2. Respondent's Literature

Respondent filed literature that specifically addressed the relevant considerations for a diagnosis of GBS.[20] Respondent also filed into the record an article that discussed the symptoms of lateral medullary syndrome,[21] which is characterized by a constellation of neurologic symptoms caused by a stroke in the vertebral or posterior inferior cerebellar artery (PICA) of the brain stem. See Resp't's Ex. A at 11 (citing Resp't's Ex. E[22] at 570). In addition, respondent provided diagrams of the brainstem and transverse views of the inferior medulla near the cervical junction to depict the neuroanatomic structures referenced by Dr. Bielawski when he discussed Mr. Carrino's symptoms and signs. See Resp't's Ex. O.[23]

C. The parties disagree about the condition from which Mr. Carrino suffered during his October 2006 hospitalization.

For perspective before considering the experts' respective positions, a brief summary follows of the injuries about which the parties disagree.

1. GBS

GBS is an "acute, immune[-]mediated polyneuropathy with several variant forms, the most common of which in the United States is acute inflammatory demyelinating polyneuropathy." Resp't's Ex. A at 7. This variant represents 85 to 90 percent of cases. Id.

A diagnosis of GBS is based initially on clinical presentation. The "cardinal clinical features" of GBS are progressive, mostly symmetric muscle weakness and absent

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[20] Francine Vriesendorp, Clinical Features and Diagnosis of Guillain-Barre Syndrome in Adults, http://www.uptodate.com/contents/clinical-features-and-diagnosis-of-guillain-barre-syndrome-in-adults?source=search_result&search=guillain-barre+syndrome&selectedTitle=1%7E150 (last visited June 5, 2013).

[21] Lateral medullary syndrome is also referred to as Wallenberg syndrome or posterior inferior cerebellar artery syndrome.

[22] Respondent's Exhibit E is identified as H.J.M. Barnett et al., Lateral Medullary Syndrome in Stroke: Pathophysiology, Diagnosis, and Management 570 (1986).

[23] R. Trues & M. Carpenter, Human Neuroanatomy 306, 309 (6th ed. 1969).

or depressed deep tendon reflexes. Resp't's Ex. D at 6 (Vriesendorp article).[24] To confirm a GBS diagnosis, spinal taps and neurophysiology studies routinely are performed in patients suspected of having the disorder. Id.; see also Resp't's Ex. C at 2.

A GBS diagnosis is appropriate if cerebrospinal fluid and clinical neurophysiology studies show certain characteristic abnormalities. Id. In GBS patients, the cerebrospinal fluid surrounding the spinal cord and brain contain elevated protein levels. See id.

Nerve conduction studies (NCS) and needle electromyographs (EMG), that measure nerve signaling to muscles, provide important information that assists in diagnosing GBS. See id. at 7. Because the abnormalities detected by nerve conduction tests progress over time, serial clinical and neurophysiologic studies are also helpful. Id.

Among the clinical features most commonly observed in patients with fully developed GBS are: abnormal needle electromyography (99 percent), weakness in legs (95 percent), areflexia[25] (90 percent), weakness in arms (90 percent), protein levels in cerebrospinal fluid exceeding 55 grams/dl (90 percent), paresthesias (85 percent), sensory loss (75 percent), and weakness in face (60 percent). Resp't's Ex. A at 9.

2. Lateral Medullary Syndrome

This syndrome is characterized by a constellation of neurologic symptoms triggered by a stroke in the vertebral or posterior inferior cerebellar artery (PICA) of the brain stem. See Resp't's Ex. A at 11 (citing Resp't's Ex. E at 570).[26]

> Symptoms include difficulties with swallowing, hoarseness, dizziness, nausea and vomiting, rapid involuntary movements of the eyes (nystagmus), and problems with balance and gait coordination. Some individuals will experience a lack of pain and temperature sensation on only one side of the face, or a pattern of symptoms on opposite sides of the body – such as paralysis or numbness in the right side of the face, with weak or numb limbs

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[24] Francine Vriesendorp, Clinical Features and Diagnosis of Guillain-Barré Syndrome in Adults, http://www.uptodate.com/contents/clinical-features-and-diagnosis-of-guillain-barre-syndrome-in-adults?source=search_result&search=guillain-barre+syndrome&selectedTitle=1%7E150 (last visited June 5, 2013).

[25] Areflexia refers to the absence of reflexes. Areflexia, http://www.dorlands.com/def.jsp?id=100007742 (last visited June 5, 2013).

[26] Respondent's Exhibit E is identified as H.J.M. Barnett et al., Lateral Medullary Syndrome in Stroke: Pathophysiology, Diagnosis, and Management 570 (1986).

> on the left side. Uncontrollable hiccups may also occur, and some individuals will lose their sense of taste on one side of the tongue, while preserving taste sensations on the other side.

Wallenberg's Syndrome, http://www.ninds.nih.gov/disorders/wallenbergs/wallenbergs.htm (last visited May 2, 2013); see also Resp't's Ex. E 4-8.

The prognosis for subjects with lateral medullary syndrome depends on the size and location of the brain stem segment damaged by the stroke. See id.

D. The opinions of petitioner's medical witnesses

Mr. Carrino's primary care physician, Dr. DeMayo, states in the letter he drafted on petitioner's behalf in this vaccine action, that his patient received a flu vaccine on October 6, 2006 and shortly thereafter, "developed weakness, loss of balance, unsteady gait and confusion." Pet'r's Ex. 12 at 2. Admitted to the hospital five days after his vaccination, Mr. Carrino's "neurological status deteriorated." Id. Dr. DeMayo indicated that the CAT scans taken of Mr. Carrino's head did not reveal any bleeding or lesions that were suggestive of a cerebrovascular accident (CVA); he did not address, however, the MRI, performed on November 1, 2006, that confirmed the diagnostic impression of stroke held by Mr. Carrino's treating physicians during his hospitalization. Pet'r's Ex. 10 at 56; Id.

Petitioner's expert neurologist, Dr. Hogen, opined that Mr. Carrino became unable to rise from bed or walk or sit independently because he had suffered a GBS event. As evidence that Mr. Carrino suffered a vaccine-related GBS, Dr. Hogen pointed to Mr. Carrino's presentation to the hospital with symptoms of "extensive weakness of his arms and legs," within one week of his receipt of the flu vaccine. Pet'r's Ex. 19 at 2. According to Dr. Hogen, Mr. Carrino "had a thorough work-up to rule out other causes of [his] weakness," id., and "was given a diagnosis of Guillain-Barré Syndrome to account for [his] . . . diffuse[] weakness." Id. Dr. Hogen reasons that Mr. Carrino must have suffered from GBS because his attending physicians in the hospital treated him with Solu-Medrol and IVIG, both of which therapies are routinely administered to GBS patients. Id. As further support for his opinion, Dr. Hogen relied on the notations of Mr. Carrino's past history for GBS, which were contained in subsequent medical records dated more than one year after Mr. Carrino's hospital discharge. Pet'r's Ex. 21 at 1.

Respondent's neurology expert, Dr. Martin Bielawski, challenged petitioner's claim that Mr. Carrino suffered from GBS. Dr. Bielawski asserted that Mr. Carrino's signs and symptoms at hospital admission were "consistent with right lateral medullary syndrome, a vascular ischemic event." Resp't's Ex. A at 9. Dr. Bielawski pointed to the

findings on Mr. Carrino's brain MRI, which was performed on November 1, 2006 (three weeks after Mr. Carrino's hospital admission), as confirmation of the stroke event.

The November 2006 MRI findings did show a right lateral medullary infarct. Resp't's Ex. N at 2. Dr. Bielawski explained that the location of Mr. Carrino's brain injury had determined what his clinical symptoms and signs were on presentation to the hospital. Id. at 1.

Dr. Bielawski observed that notwithstanding certain of his symptoms, Mr. Carrino did not "fulfill the diagnostic criteria for GBS." Resp't's Ex. A at 9. Nor did his discharge summary reflect a diagnosis of GBS, but rather of a cerebral artery occlusion with cerebral infarction. Id. at 6.

Unlike the conclusory reports and circular arguments regarding vaccine-related injury prepared by Dr. DeMayo and Dr. Hogen, Dr. Bielawski's expressed opinion was amply supported by detailed references to Mr. Carrino's hospitalization records from his October 2006 admission—which did not include a diagnosis of GBS—and to the literature he filed. See generally Resp't's Ex. A, N, and Q.

Before turning to evaluate petitioner's claim, the undersigned first sets forth the applicable legal standard.

II. STANDARDS OF ADJUDICATION

A. Elements of petitioner's claim

If petitioner alleges that an injury listed on the Vaccine Injury Table (Table) occurred within the correlative time frame set forth in the Table, petitioner's vaccine claim is deemed a Table claim, and a presumption of vaccine causation attaches. See § 300aa-14; see also 42 C.F.R § 100. If petitioner alleges an injury that is not listed on the Table (such as the GBS injury alleged in this case), the vaccine claim is deemed a non-Table case, and no presumption of causation attaches. In such circumstances, petitioner must satisfy her burden of proof. See § 300aa-13(a)(1)(A).

To prevail on a non-Table vaccine claim, such as petitioner has asserted here, she must allege: (1) that the vaccinee "sustained, or had significantly aggravated any illness, disability, injury, or condition not set forth in the Vaccine Injury Table;" and (2) that the injury "was caused by a vaccine." 42 U.S.C. § 300aa-11(c)(1)(C)(ii)(I). Petitioner must show that the vaccine was "'not only a but-for cause of the injury but also a substantial factor in bringing about the injury.'" Moberly v. Sec'y of Health & Human Servs., 592 F.3d 1315, 1321 (Fed. Cir. 2010) (quoting Shyface v. Sec'y of Health & Human Servs., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)).

As required by the Federal Circuit, petitioner must prove that the flu vaccine given to Mr. Carrino caused his injury and death by preponderant evidence that shows: (1) a medical theory causally connecting the vaccine and Mr. Carrino's injury; (2) a logical sequence of cause and effect showing that the vaccine was the reason for his injury; and (3) a showing of a proximate temporal relationship between the vaccine and his injury. Althen v. Sec'y of Health & Human Servs., 418 F.3d 1274, 1278 (Fed. Cir. 2005); 42 U.S.C. § 300aa–13(a)(1) (requiring proof by a preponderance of the evidence).

Because the causation theory must relate to the injury alleged, a petitioner must provide a reputable medical or scientific explanation that pertains specifically to the vaccinee's case—although the explanation need only be "legally probable, not medically or scientifically certain." Knudsen v. Sec'y of Health & Human Servs., 35 F.3d 543, 548–49 (Fed. Cir. 1994); Moberly, 592 F.3d at 1322. To be clear, "the function of a special master is not to 'diagnose' vaccine-related injuries, but instead to determine 'based on the record evidence as a whole and the totality of the case, whether it has been shown by a preponderance of the evidence that a vaccine caused the [claimed] injury.'" Andreu v. Health & Human Servs., 569 F.3d 1367 at 1382 (Fed. Cir. 2009) (quoting Knudsen v. Sec'y of Health & Human Servs., 35 F.3d 543, 549 (Fed. Cir. 1994)).

The preponderance of the evidence standard has been interpreted to mean that a fact is more likely than not. Moberly, 592 F.3d at 1322 n.2 (Fed. Cir. 2010). A petitioner who satisfies this burden is entitled to compensation unless the government can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors unrelated to the administration of the vaccine." 42 U.S.C. § 300aa-13(a)(1)(B).

B. Establishing the nature of petitioner's injury

When—as in this case—the parties dispute the nature of the injury at issue, the special master must first determine which injury is best supported by the evidence before applying the Althen test to determine whether the vaccine caused the injury. Broekelschen v. Sec'y of Health & Human Servs., 618 F.3d 1346, 1350 (Fed. Cir. 2010); see also Locane v. Sec'y of Health & Human Servs., 685 F.3d 1375 (Fed. Cir. 2012); Lombardi v. Sec'y of Health & Human Servs., 656 F.3d 1343 (Fed. Cir. 2011). A special master's findings regarding the nature of petitioner's injury may be sufficient to resolve the case because the special master determines, from the record evidence, that the injury petitioner suffered was not the injury that was contemplated in petitioner's theory of causation. See Lombardi, 656 F.3d at 1356; Broekelschen, 618 F.3d at 1350. To assist a special master in evaluating a claim, the Federal Circuit has provided guidance that evidence of an injury other than the one alleged can be relevant—not only to the "factors unrelated" defense on which the government bears the burden of proof—but also to the showing petitioner must make that the vaccine was a substantial factor in causing the claimed injury. Stone v. Sec'y of Health & Human Servs., 676 F.3d 1373, 1380 (2012).

C. Evaluating the presented evidence

Petitioner cannot establish entitlement to Program compensation based solely on the claims of petitioner alone. Rather, a vaccine claim must be supported either by the medical records or by the opinion of a competent physician. 42 U.S.C. § 300aa-13(a)(1). In determining whether petitioner is entitled to compensation, a special master shall consider all material contained in the record, 42 U.S.C. § 300aa-13(b)(1), including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation . . . of the petitioner's illness." 42 U.S.C. § 300aa-13(b)(1)(A) (emphasis added).

1. Reliability of medical records

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 created contemporaneously to the medical events. Cucuras v. Sec'y of Health & Human Servs., 993 F.2d 1525, 1528 (Fed.Cir.1993) (citing United States v. U.S. Gypsum Co., 333 U.S. 364, 396 (1947). The Federal Circuit's decision in Cucuras v. Secretary of Health & Human Services clearly supports the view that medical records are favored over oral testimony in circumstances when there is a conflict between the former and the latter and when the prepared medical records are internally consistent and complete. Id.

2. The experts' opinions

The persuasiveness of the experts' testimony must be evaluated, and the testimony of one side's expert may be rejected when there is a reasonable basis for doing so. Burns v. Sec'y of Health & Human Servs., 3 F.3d 415, 417 (Fed. Cir. 1993).[27]

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[27] In the Vaccine Program, an expert's opinion may be evaluated according to the factors identified by the United States Supreme Court in Daubert v. Merrell Dow Pharms., Inc., 509 U.S. 579 (1993). Terran v. Sec'y of Health & Human Servs., 195 F.3d 1302, 1316 (Fed. Cir. 1999). As recognized in Terran, the Daubert factors for analyzing the reliability of testimony are:

> (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and, (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

When evaluating the reliability of an expert's opinion, it is important to ascertain whether the information on which the doctor is relying is accurate because inaccuracies in the expert's factual assumptions compromise the reliability of the view offered. See Perreira v. Sec'y of Health & Human Servs., 33 F.3d 1375, 1377 (Fed. Cir. 1994) (an expert opinion is no better than the soundness of the reasons supporting it).

Applying these standards to determine whether petitioner has established that she is entitled to compensation for Mr. Carrino's alleged vaccine-related injury of GBS, the undersigned finds that she has not. Therefore, she is not entitled to compensation. The undersigned's reasoning is explained, in detail, below.

III. ANALYSIS

The contemporaneous medical records reflect a general and consistent consensus about the nature of Mr. Carrino's injury. His attending physicians recorded the same diagnostic impression, and the testing and clinical evaluations supported the treating neurologist's diagnostic conclusion. The difficulty with petitioner's vaccine claim is that the vaccine-related injury she asserts her husband suffered is not the injury with which he was diagnosed in the medical records. Petitioner's experts have offered opinions in support of petitioner's claim that rely on selectively chosen aspects of the medical records, while disregarding the consistent diagnostic conclusions reached by Mr. Carrino's attending physicians. Remarkably, Mr. Carrino's primary care physician now offers an opinion of causation that contradicts his earlier, contemporaneously recorded impressions of Mr. Carrino's condition.

In vaccine cases, expert testimony is helpful when the medical records are unclear. But, when—as here—the medical records speak plainly about the injury at issue, the value of the experts' testimony is somewhat diminished.

Apparently, petitioner has offered expert testimony to support her vaccine injury claim because the contemporaneous medical records of Mr. Carrino's October 2006 hospitalization contain a diagnosis of a condition other than the GBS event petitioner

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Terran, 195 F.3d at 1316 n.2 (citing Daubert, 509 U.S. at 592-95). After Terran, decisions issued by of the Court of Federal Claims have consistently cited to Daubert. E.g. De Bazan v. Sec'y of Health & Human Servs., 70 Fed. Cl. 687, 699 n.12 (2000) ("A special master assuredly should apply the factors enumerated in Daubert in addressing the reliability of an expert witness's testimony regarding causation."), rev'd on other grounds, 539 F.3d 1347 (Fed. Cir. 2008); Campbell v. Sec'y of Health & Human Servs., 69 Fed. Cl. 775, 781 (2006); Piscopo v. Sec'y of Health & Human Servs., 66 Fed. Cl. 49, 54 (2005).

alleges. Petitioner presented the medical testimony of Mr. Carrino's primary care physician, Dr. DeMayo, and an expert neurologist, Dr. Hogen. Their opinions, which were developed in the context of this litigation conflicted with the contemporaneous medical records and thus, failed to persuade.

In contrast, the testimony of respondent's expert neurologist, Dr. Bielawski, effectively relied on and was consistent with the medical records and the referenced literature.

The undersigned reviews the experts' positions in turn.

A. The opinion of the parties' experts

1. Mr. Carrino's primary care physician, Dr. Joseph DeMayo[28]

In his written report and at hearing, Dr. DeMayo asserted that Mr. Carrino suffered a flu vaccine-related GBS event that precipitated his October 2006 hospitalization. This opinion, however, contradicted his diagnostic assessment (and treatment) of Mr. Carrino during that hospitalization.

Dr. DeMayo administered Mr. Carrino's flu vaccination five days prior to his hospitalization, and served as his admitting physician at the hospital. The contemporaneous medical records do not indicate that Dr. DeMayo contemplated a diagnosis of GBS; instead, Dr. DeMayo indicated that Mr. Carrino's presenting symptoms on hospital admission were consistent with a cerebrovascular accident. Pet'r's Ex. 11 at 1. In fact, prior to the initiation of this Vaccine Program claim, the record is devoid of any evidence that Dr. DeMayo diagnosed Mr. Carrino with GBS.

In the opinion he has offered on petitioner's behalf here, Dr. DeMayo outlined the symptoms of "weakness, loss of balance, unsteady gait, and confusion" that Mr. Carrino developed five days after he received the flu shot. Pet'r's Ex. 12 at 2. He reasoned that Mr. Carrino must have suffered a GBS event because he developed neurologic symptoms within a medically acceptable time period after receiving the flu vaccine, and because the performed "diagnostic testing. . . [had] eliminate[d] other causes." Id.

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[28] Consistent with the guidance from the Federal Circuit in Capizzano, Dr. DeMayo's earlier prepared medical records are accorded more weight than his later-developed medical opinion of vaccine-related causation because while he was acting as a "treating physician" contemporaneous to the events at issue, he was better positioned to determine whether "a logical sequence of cause and effect show[s] that the vaccination was the reason for the injury." Capizzano v. Sec'y of Health & Human Servs., 440 F.3d 1317, 1326 (Fed. Cir. 2006) (internal citations omitted).

Although at hearing, Dr. DeMayo expressed agreement with respondent's expert, Dr. Bielawski, that the combination of clinical symptoms and the results of cerebrospinal fluid testing and needle electromyography is important to consider when making a diagnosis of GBS, he had difficulty identifying the predicate factors that led to the change in his view of Mr. Carrino's medical condition.

Because the medical records were clear and consistent, the undersigned accords more weight to Dr. DeMayo's impressions recorded in his contemporaneous treatment notes than to his later-offered opinion of causation.

2. Petitioner's expert neurologist, Dr. Victor Hogen

The reliability and persuasiveness of Dr. Hogen's expert opinion were significantly diminished by his lack of familiarity with critical details in the medical records from Mr. Carrino's October 2006 hospitalization.

Further diminishing the reliability and persuasiveness of Dr. Hogen's offered opinion was his reliance on Mr. Carrino's later medical records (for treatment after Mr. Carrino's 2006 hospitalization) to support the theory that Mr. Carrino developed GBS in the days following his flu vaccination. Petr'r's Ex. 21 at 2 (citing numerous medical records from various health care providers in 2008—the year this vaccine claim was filed—and noting a past history of GBS for Mr. Carrino).

With only light record support for his opinion, Dr. Hogen asserted that a subject's clinical symptoms, without more, are sufficient to make a diagnosis of GBS. He posited that the sole two features required for a GBS diagnosis are (1) ascending weakness, and (2) missing or diminished reflexes. Pet'r's Ex. 21 at 2-3. He testified that Mr. Carrino had roughly symmetrical, ascending paralysis, and diminished or lost reflexes. Tr. at 87. He wholly ignores the fact that Mr. Carrino did not present to the hospital with these symptoms, but briefly manifested the symptoms after he acquired a urinary tract infection.

Dr. Hogen disregarded the well-established diagnostic criteria for GBS, urging that neither cerebrospinal fluid testing or nerve electromyography are necessary to confirm a GBS diagnosis after a patient's clinical symptoms have raised a treating physician's suspicions. Tr. at 79. His view concerning the proper diagnostic criteria for GBS conflicted with the opinions of Mr. Carrino's treating physician, Dr. DeMayo, and respondent's expert, Dr. Bielawski. The undersigned did not accord significant weight to Dr. Hogen's opinion because it was a singular perspective lacking adequate support in either the medical records or medical literature.

3. Respondent's expert neurologist, Dr. Martin Bielawski

Dr. Bielawski's testimony offered a more clear and cogent explanation of the contemporaneously created medical records than did the testimony of petitioner's experts Drs. DeMayo and Hogen. Dr. Bielawski filed literature regarding the relevant considerations in diagnosing GBS. A GBS diagnosis is established by the presence of certain clinical features, and the results of cerebrospinal fluid testing and an EMG. Tr. at 125; Resp't's Ex. D at 6-7 (the Vriesendorp article).[29] Dr. Bielawski agreed with Dr. Hogen that the clinical symptoms of "progressive weakness in both arms and legs" and "areflexia" or missing reflexes are required for a GBS diagnosis. Resp't's Ex. A at 7.

Dr. Bielawski asserted that Mr. Carrino did not have GBS because his symptoms at the time of his hospital admission were not consistent with that condition. Dr. Bielawski acknowledged, however, that for a brief period during Mr. Carrino's hospitalization, his neurologic symptoms worsened in a manner that triggered concern for a GBS event. His attending physicians proactively ordered treatment for his suspected GBS with steroids and IVIG, but that particular treatment did not prove effective.

At the same time that his neurological symptoms began to worsen, Mr. Carrino was treated for a urinary tract infection. As the infection resolved, the symptoms that provoked concern that he might have had a GBS event also resolved. Dr. Bielawski testified that the rapid resolution of Mr. Carrino's symptoms after the treatment of his urinary tract infection was appropriate for a stroke patient who had developed an infection. Resp't's Ex. N at 4; see also Tr. at 116.

The parties' experts made reference to Mr. Carrino's medical records to support their respective positions. Because a review of the records indicates that Mr. Carrino was not diagnosed with GBS during his October 2006 hospitalization, the undersigned turns now to evaluate Mr. Carrino's reported symptoms in accordance with the diagnostic criteria for GBS upon which Mr. Carrino's treating physician, Dr. DeMayo, and respondent's expert, Dr. Bielawski, agreed.

B. Mr. Carrino's clinical symptoms during his October 2006 hospitalization did not meet the diagnostic criteria for GBS.

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[29] Francine Vriesendorp, Clinical Features and Diagnosis of Guillain-Barre Syndrome in Adults, http://www.uptodate.com/contents/clinical-features-and-diagnosis-of-guillain-barre-syndrome-in-adults?source=search_result&search=guillain-barre+syndrome&selectedTitle=1%7E150 (last visited June 5, 2013). Dr. Bielawski indicated that this diagnostic guidance was available and relevant in October 2006, when Mr. Carrino was hospitalized several days after his receipt of the flu vaccine. Tr. at 127-28.

The parties agreed that there are a number of clinical symptoms that would be diagnostic of GBS.

1. Lost or significantly diminished deep tendon reflexes[30]

The parties agreed that deep tendon reflexes must be lost or significantly diminished to merit a GBS diagnosis. See Tr. at 24 (Dr. DeMayo); Tr. at 73 (Dr. Hogen); Tr. at 166-7 (Dr. Bielawski); Resp't's Ex. A at 7-8.

Dr. Brand conducted several neurologic examinations of Mr. Carrino during his hospitalization and documented in his notes the state of Mr. Carrino's "preserved reflexes in his knees and in his arms." Tr. at 117 (Dr. Bielawski). The "preserved" condition of Mr. Carrino's reflexes during the early days of his hospitalization in October of 2006 "was. . . a sign pointing against GBS as a diagnosis" Tr. at 116-17 (Dr. Bielawski).

During his hospitalization, Mr. Carrino did briefly lose his knee reflexes when he contracted a urinary tract infection. See Pet'r's Ex. 11 at 32; Pet'r's Ex. 11 at 35; Pet'r's Ex. 11 at 44; Tr. at 117. With antibiotic treatment of that urinary tract infection, Mr. Carrino's reflexes returned within two days. Tr. at 117. Dr. Bielawksi explained that the "rapid recovery" of Mr. Carrino's reflexes was not unusual for a stroke patient "who has had a superimposed toxic metabolic or infectious event"—such as Mr. Carrino experienced with his acquired urinary tract infection. Resp't's Ex. N at 4. Had Mr. Carrino suffered a GBS event, his knee reflexes would not have returned so quickly. See Tr. at 116 (Dr. Bielawski).

Although petitioner's expert, Dr. Hogen, argued that most of Mr. Carrino's treating physicians had identified missing or absent reflexes that were consistent with a GBS diagnosis, Tr. at 74, he did not provide any record citations for his assertions. The records do consistently make reference to Mr. Carrino's absent ankle reflexes; but his missing ankle reflexes had been attributed to his diabetic neuropathy years before the flu vaccine at issue.

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[30] Normally, when a muscle tendon is tapped briskly, the muscle contracts immediately due to a two-neuron reflex arc involving the spinal or brainstem segment that innervates the muscle. Any asymmetry of reflexes suggests that an abnormality is present. H. K. Walker. Deep Tendon Reflexes, in Clinical Methods: The History, Physical and Laboratory Examinations 365, 368 (H.K. Walker et al. eds., 3rd ed. 1990), available at http://www.ncbi.nlm.nih.gov/books/NBK396/#A2362 (last visited June 5, 2013).

The undersigned finds that apart from a brief loss of reflexes in connection with his urinary tract infection, Mr. Carrino did not lose or have significantly diminished deep tendon reflexes after his flu vaccination in October 2006.

2. Progressive weakness

The parties agreed that a hallmark symptom of GBS is progressive weakness or ascending paralysis. Resp't's Ex. A at 7-9; Resp't's Ex. N at 4; see also Tr. at 22 (Dr. DeMayo); Pet'r's Ex. 21 at 1 (Dr. Hogen). Such ascending paralysis, which "is a roughly symmetric, progressive paralysis," typically peaks within fourteen days of symptom onset. Tr. at 57 (Hogen).

At the time of Mr. Carrino's hospital admission, he did not have progressive weakness. Nor did he exhibit any exacerbations of his symptoms during the first nine days of his hospitalization. Resp't's Ex. A at 7-9; Resp't's Ex. N at 4; Tr. at 162-63. Instead, the records reflect that within a few days, his symptoms were better. Mr. Carrino's attending neurologist, Dr. Brand, specifically noted an improvement in Mr. Carrino's ataxia three days after his hospital admission. Pet'r's Ex. 11 at 7.

Nonetheless, petitioner's expert, Dr. Hogen, asserted that Mr. Carrino had "progressive weakness in both arms and both legs." Tr. at 80. A review of Mr. Carrino's medical records show two documented instances of Mr. Carrino's "ascending weakness." Tr. at 119 (Bielawski). The significance of these notations, however, is not entirely clear because they were made by two different consulting physicians—but not Mr. Carrino's attending neurologist, Dr. Brand—and neither provided clinical support for the notation. See Tr. at 156, 157, 162 (Dr. Bielawski); Resp't's Ex. A at 10 (Dr. Bielawski asserting that Dr. Beggs's notation in the medical records that Mr. Carrino "ha[d] [experienced] ascending weakness bilaterally" since his hospitalization was "wrong").

These two notations of ascending weakness were not documented in the hospital records until October 21, 2006, when Mr. Carrino complained to Dr. DeMayo of neck pain, weakness in all of his extremities, and difficulty swallowing. Pet'r's Ex. 11 at 25. Dr. Brand, the attending neurologist, evaluated Mr. Carrino that same day and noted there was more weakness in his legs and pain in his right shoulder with weakness in his right arm. Pet'r's Ex. 11 at 27.

Mr. Carrino's worsening condition resulted in his transfer to the intensive care unit the next day. Pet'r's Ex. 11 at 28. On examination of Mr. Carrino, Dr. Brand found an absence of "knee jerks and ankle jerks with preserved upper extremity deep tendon reflexes." Pet'r's Ex. 11 at 32. This decline in Mr. Carrino's condition provoked his treating doctors to consider a diagnosis of GBS. Tr. at 119-20.

Dr. Bielawski acknowledged in his testimony that Dr. Brand had become concerned about the possibility of a GBS event when Mr. Carrino developed significant, new symptoms of generalized weakness more than one week after his hospitalization. Tr. at 126. Dr. Bielawski also acknowledged that Mr. Carrino received a course of Solu-Medrol and IVIG, which was intended to treat what seemed to be an emerging GBS episode. Tr. at 150-51. However, Dr. Bielawski cogently explained:

> [T]he reason [Mr. Carrino] became weak was because he had a systemic illness which was the urinary tract infection. He had a low grade fever. And we see this all the time in patients who have stroke syndromes, that they have unmasking of various neurological signs, and they can become generally weak, they can become confused. And once the urinary tract infection . . . is treated, they get back to or close to their baseline.

Tr. at 151.

Once treated with antibiotics, Mr. Carrino's infection did resolve—as did his symptoms of pronounced weakness. Pet'r's Ex. 11 at 62. A subsequent neurologic exam indicated that he had returned to a state "similar to that on admi[ssion]." Id.

The undersigned finds that Mr. Carrino briefly exhibited, in association with his urinary tract infection, a progressive weakness that resolved within a five-day period.

3. Paresthesia

A very common symptom of GBS is paresthesia. Tr. at 117 (Bielawski). About 80 percent of affected subjects experience pins and needles (paresthesia) in their hands and feet. Resp't's Ex. A at 6-9, Tr. at 117-18, 141, 166 (Dr. Bielawski).

The parties do not dispute that Mr. Carrino made no complaint of tingling in either his hands or his feet (paresthesia), Pet'r's Ex. 11 at 27; Tr. at 85-86 (Dr. Hogen); Tr. at 117-18 (Dr. Bielawski), and the undersigned finds that Mr. Carrino did not suffer with paresthesia.

4. Symmetric symptom presentation

The parties agreed that symmetrical neurologic symptoms are a sign that is strongly suggestive of a GBS injury. Conversely, a marked, persistent asymmetric presentation of symptoms renders a GBS diagnosis more doubtful. Resp't's Ex. A at 7; Tr. at 24 (Dr. DeMayo); Tr. at 57, 86 (Dr. Hogen). Mr. Carrino's motor and sensory signs were persistent and notable for their asymmetry. Resp't's Ex. A at 9; Resp't's Ex. N at 4; Tr. at 118 (Dr. Bielawski). In particular, the "numbness and diminished sensation [Mr. Carrino] had in his left leg up through [his] rib cage" was inconsistent

with the type of symmetric symptoms that are characteristic of GBS. Tr. at 118 (Dr. Bielawski). The undersigned finds that Mr. Carrino's symptom presentation was asymmetric.

5. Facial weakness

Facial weakness is another common indicator of GBS. Dr. Bielawski testified that roughly 60 percent of GBS patients complain of such weakness. Resp't's Ex. A at 9; Tr. at 121 (Dr. Bielawski). Dr. Hogen agreed that facial weakness can be a "supportive feature" of a GBS finding, but he denied that it is "one of the prime diagnostic factors." Tr. at 86.

That Mr. Carrino did not complain of facial weakness during his hospitalization is undisputed. Resp't's Ex. A at 7-9; Tr. at 24-25 (Dr. DeMayo); Tr. at 86 (Dr. Hogen); Tr. at 120-21 (Dr. Bielawski). The undersigned finds that he did not develop facial weakness.

6. Elevated protein levels in the cerebrospinal fluid

Dr. DeMayo and Dr. Bielawski both agreed that the testing of a subject's cerebrospinal fluid is important in circumstances involving a "clinical suspicion" of GBS. Tr. at 21 (Dr. DeMayo), 125 (Dr. Bielawski). Dr. Bielawski testified that 80 to 90 percent of GBS patients will have "an elevated [protein level in their] cerebrospinal fluid one week after the onset of symptoms." Resp't's Ex. A at 9; see also Tr. at 125.

After a week of hospitalization, Mr. Carrino's neurologic symptoms began to deteriorate further and he developed difficulty breathing and swallowing. This change in his condition provoked his treating physicians to order a spinal tap. The spinal tap, performed twelve days after his admission to the hospital, revealed that Mr. Carrino's cerebrospinal protein levels were normal, Tr. at 55 (Dr. Hogen), an unusual finding for a GBS diagnosis. The undersigned finds that the protein levels in Mr. Carrino's cerebrospinal fluid were not elevated.

7. EMG abnormalities

The parties' witnesses, Dr. DeMayo and Dr. Bielawski, agreed that an abnormal EMG is present in 95 to 99 percent of patients with GBS. They also agreed that in consideration with a subject's clinical symptoms and cerebrospinal fluid test results, an EMG is a critical aid to diagnosing GBS. See Tr. at 21 (Dr. DeMayo); Resp. Ex. A at 7-8; Tr. at 125, 158 (Dr. Bielawski).

Dr. DeMayo testified at hearing that he did not recall Dr. Brand ever ordering an EMG. Tr. at 21-22. Dr. Hogen stated that he did not see an EMG report in the record;

Tr. at 79, nor did he see any notes discussing EMG results in any detail. Id.

The record indicates that Dr. Brand did order an EMG/NCTS on October 23, 2006, Pet'r's Ex. 11 at 38, but the results of the EMG were never filed into the record. Dr. Brand commented in his notes, the day after he ordered the testing, that the EMG "findings" were consistent with peripheral neuropathy, but they did not exclude a Guillain-Barré type of radiculopathy.[31] Pet'r's Ex. 11 at 44; see also Resp't's Ex. at 6.

After careful review of the record as a whole, the undersigned is persuaded that an EMG was performed, and the results did not rule out the possibility of GBS.

The aggregate of Mr. Carrino's symptoms, however, failed to satisfy the diagnostic criteria for GBS upon which the parties' witnesses agreed. Mr. Carrino's presenting symptoms at the time of his hospital admission and during the course of his hospitalization led both his primary care physician and then-treating neurologist to reach a different diagnostic conclusion than GBS. Instead, and Mr. Carrino's diagnosis at the time of his hospital discharge was lateral medullary infarct (stroke). Pet'r's Ex. 11 at 78.

C. The symptoms precipitating Mr. Carrino's hospitalization were strongly suggestive of lateral medullary syndrome

Mr. Carrino presented for admission to the hospital, five days after receiving a flu vaccine, with "vertigo, nausea, weakness, [and an inability] to walk." Pet'r's Ex. 11 at 1. Over the next ten days, Mr. Carrino's symptoms of vertigo, nausea, weakness, inability to walk, headache, neck discomfort, involuntary eye movement, double vision, slight right eyelid drooping, hiccups, and right cerebellar signs were well-documented. Resp't's Ex. A at 6, 8, 10; Tr. at 25, 31, 33, 45 (Dr. DeMayo); Tr. at 82 (Dr. Hogen); Tr. at 86-87, 118-19, 121, 130-32, 135-36, 171 (Dr. Bielawski). The symptoms were all indicative of a stroke. Resp't's Ex. A at 6, 8, 10; Resp't's Ex. E at 4 (noting that "[m]oderate or severe headache is common in lateral medullary infarction" and "vertigo or other feelings of disequilibrium are nearly always present"); Resp't's Ex. E at 5 (finding ataxia "the rule rather than the exception," citing diplopia and hiccups as "other frequent complaints," and identifying nausea and vomiting as "common symptoms" of vestibular dysfunction). On admission to the hospital, Mr. Carrino's primary care physician, Dr. DeMayo, assessed him with a cerebrovascular accident or a stroke, and four days after his hospital admission, Dr.

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[31] The report for this EMG study was never located or filed by petitioner. However, respondent's expert, Dr. Biewlaski, characterized Dr. Brand's notations in the medical record as consistent with the type of remarks that would follow such testing. See Resp't's Ex. A at 6.

Brand was able to speculate where the damage to Mr. Carrino's brain had occurred based on neuroanatomy. Pet'r's Ex. 11 at 1, 9 (Dr. Brand positing that Mr. Carrino's presenting symptoms were caused by a stroke in the area of the brain close to the brainstem and cerebellum).[32]

At hearing, Dr. Hogen testified that Mr. Carrino's symptoms of dizziness, light-headedness and ataxia were a manifestation of the autonomic dysfunction that can occur in some cases of GBS. Tr. at 89-90.

Dr. Bielawski responded by observing that dizziness is not a symptom of GBS and Mr. Carrino "sudden[ly]" and "acute[ly]" developed dizziness and ataxia. Tr. at 130-31; see also Resp't's Ex. E at 5. He added that damage produced by a right lateral medullary infarct can "compromise one of the tracts called the vestibular ocular tract" and cause dizziness. Tr. at 130-31. This testimony was unrebutted.

At hearing, Dr. Bielawski testified that Mr. Carrino's inability to walk at the time of his hospital admission, and his subsequent difficulty with his finger-to-nose neurologic test were signs of a disruption of his right cerebellar connections. Tr. at 130-31. "The cerebellum is [the] organ that . . . [regulates] coordination and [a disruption of] the cerebellar connections on the right side of the brain stem would . . . [produce such signs as] finger[-]to[-]nose ataxia." Tr. at 130-31.

Dr. Bielawski added that eyelid droop or ptosis can occur when "the sympathetic fibers [are] compromised [by] . . . ischemic injury." Tr. at 132. Mr. Carrino's initial eyelid droop and later double vision were additional symptoms of a brain stem stroke. See Tr. at 132.

Dr. Bielawski commented that because patients with autonomic syndrome are further compromised by a GBS event, Tr. at 137, other symptoms of autonomic dysfunction—such as "alternating bouts of high blood pressure and low blood pressure" would be expected. Id. Mr. Carrino did not have these symptoms, which militates against a finding that he was suffering from an autonomic syndrome associated with GBS. Id.

Dr. Hogen attempted to undercut evidence of the initial impression of stroke documented in the hospital records by both Dr. DeMayo, Mr. Carrino's primary care physician, and Dr. Brand, Mr. Carrino's attending neurologist. Dr. Hogen urged that "[a] st[r]oke in [Mr. Carrino's] parietal lobe would have affected the right side of his body . . . [m]aybe. . .produced. . .numbness on the right side." Tr. at 69. Although Dr.

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[32] "Patient has neurological signs consistent with posterior fossa ischemia." Pet'r's Ex. 11 at 9 (emphasis added).

Hogen did not "really remember exactly" what Mr. Carrino's complaints were, he "[thought] that most of [Mr. Carrino's] complaints were left-sided." Tr. at 69. A careful review of the medical records from Mr. Carrino's hospital admission indicates that most of his complaints were right-sided.[33] See Pet'r's Ex. 11 at 3-5, 6.

The medical records and the testimony of respondent's expert persuade the undersigned that Mr. Carrino's symptoms during his October 2006 hospitalization were consistent with a stroke.

1. The absence of evidence of stroke on the initial brain imaging is not determinative because a subsequent MRI confirmed the presence of a stroke

On the day of Mr. Carrino's admission to hospital, his brain MRI was read as showing no acute infarctions. Pet'r's Ex. 10 at 55. The next day, a second brain MRI also was read to show no abnormalities. Id. at 57.

The parties' expert neurologists, Drs. Hogen and Bielawski, disagreed about the significance of these initial MRIs.

Dr. Hogen asserted that the absence of acute infarctions on the brain MRIs is evidence that Mr. Carrino—who had previously suffered strokes—had not suffered a "new stroke." Pet'r's Ex. 21 at 3; Tr. at 63-65. Dr. Hogen testified, without offering any supporting references, that "an MRI-scan with diffusion weighted imaging is almost 100 percent sensitive in the first 24 hours of an acute stroke." Pet'r's Ex. 21 at 3. Dr. Hogen further asserted that "it is not likely that the MRI scan of 10/11/2006 missed a stroke[, and] it is more probable that the stroke seen on 11/01/2006 was acquired during the hospitalization at a much later date." Id.

Dr. Bielwaski disagreed. He contended that the first MRI showed either a brainstem stroke too small to detect or findings that the radiologist simply missed. Resp't's Ex. N. at 2; see Tr. 161. Supportive evidence for Dr. Bielawski's claim that the initial imaging results were simply overlooked or misread is found in the later brain imaging performed on November 1, 2006. This imaging revealed "a clear cut [right] lateral medullary infarct seen on several segments," Pet'r's Ex. 11 at 74, and Dr. Brand, Mr. Carrino's treating neurologist, determined that the neurologic injury had occurred before Mr. Carrino's hospital admission. See id. The November 1, 2006 brain MRI confirmed the location of the infarct about which Dr. Brand, Mr. Carrino's attending neurologist, had speculated four days after Mr. Carrino's hospital admission. The parties'

[33] Dr. DeMayo's medical evaluations, in particular, consistently reflect right-sided weakness. See Pet'r's Ex. 11 at 10, 12, 50.

experts, Drs. Hogen and Bielawski, also disagreed regarding the significance of the November 1, 2006 brain MRI results.

Dr. Hogen testified that the findings were too small, too late and too indeterminate to support a finding that Mr. Carrino had suffered a stroke prior to his hospital admission twenty days earlier. Tr. at 67-69. Dr. Hogen added that the strokes that were visible on the MRI were in the wrong location to have caused the functional loss that Mr. Carrino exhibited. See Tr. at 68. In Dr. Hogen's view, a small stroke at the cervical medullary junction would not have "produced the quadraparesis" that Mr. Carrino suffered. Tr. at 60. Nonetheless, Dr. Hogen conceded that Mr. Carrino's symptoms of "nausea, vomiting, and stenosis" were all consistent with the damage caused by a lateral lineal stroke (or a lateral medullary stroke). Tr. 86-87.

While this imaging evidence alone does not determine the nature of Mr. Carrino's evolving condition, it merits consideration with the other record evidence.

2. Mr. Carrino's past medical history of stroke made him vulnerable to further ischemic injury

Before 2006, Mr. Carrino had suffered at least five ischemic infarctions, including: (1) two incidents involving sixth nerve palsy; (2) one incident involving third nerve palsy; (3) a left cerebellar stroke; and (4) a left basal ganglia hemorrhagic stroke. Resp't's Ex. A at 6; Tr. at 128-29; see also Tr. at 76-77 (Dr. Hogen). His prior medical history rendered him "quite susceptible" to brain stem strokes. Tr. at 129.

3. The "progressive" symptoms that provoked concern that Mr. Carrino had developed GBS were unresponsive to a course of IVIG and steroids but did resolve, over the next five days, with antibiotic treatment

The new symptoms of weakness that emerged ten days after Mr. Carrino's hospitalization were treated preventatively, as if they were symptoms of GBS. That treatment was unavailing, but the course of antibiotics that Mr. Carrino began for treatment of a urinary tract infection was successful. Resp't's Ex. A at 9; Resp't's Ex. N at 4; Tr. at 119-20. Contrary to Dr. Hogen's assertions otherwise, this prophylactic course of treatment—and the limited response it provoked—did not point to a GBS episode.

D. Mr. Carrino did not suffer from the injury for which petitioner now seeks a Program award

As noted previously, special masters are required to evaluate the record as a whole. 42 U.S.C. §300aa-13. The record here—including the contemporaneous medical records and Mr. Carrino's brain imaging, the statements of Mr. Carrino's attending physicians during his October 2006 hospitalization, and the testimony of Drs. DeMayo, Hogen, and Bielawski—undercuts petitioner's claim that Mr. Carrino developed GBS either prior to or during his October 2006 hospitalization, and militates against a finding that Mr. Carrino's injuries were caused by a vaccine-related GBS event.

This determination precludes a finding of causation in petitioner's favor. When the evidence does not support a finding that the vaccinee suffered the injury for which petitioner seeks Program compensation, an Althen causation analysis may not be required. See Lombardi, 656 F.3d at 1356 (affirming special master's decision foregoing Althen analysis after concluding petitioner did not suffer from any of his alleged injuries). Out of an abundance of caution, however, the undersigned evaluates petitioner's theory under the Althen standard.

1. Althen Prong One: Petitioner's Medical Theory

Under Althen Prong 1, petitioner must put forth a biologically plausible theory explaining how the received vaccine "can" cause the injury alleged. Pafford v. Sec'y of Health & Human Servs., 451 F.3d 1352, 1355-56 (Fed. Cir. 2006). To satisfy this prong, "a petitioner must provide a reputable medical or scientific explanation that pertains specifically to the petitioner's case, although the explanation need only be 'legally probable, not medically or scientifically certain.'" Broekelschen, 618 F.3d at 1345 (quoting Knudsen, 35 F.3d at 548-49); see also Moberly, 592 F.3d at 1324 ("[T]he special master is entitled to require some indicia of reliability to support the assertion of the expert witness.").

The offered medical theory must be supported by either the vaccinee's medical records or the opinion of a competent physician. Grant v. Sec'y of Health & Human Servs., 956 F.2d 1144, 1148 (Fed. Cir. 1992). Support for the offered medical theory must also include an explanation that "pertains specifically to the [claim made in] petitioner's case." Moberly, 592 F.3d at 1322. See Veryzer v. Sec'y of Health & Human Servs., No. 06-0522V, 2010 WL 2507791, at *24 (Fed. Cl. Spec. Mstr. 2010) (noting that the relevant inquiry is whether, based on facts known to medical science and logical inferences drawn by a qualified expert, the vaccine at issue is more than likely to have caused the alleged injury), aff'd, 100 Fed. Cl. 349 (2011), aff'd, 475 F. App'x 765 (Fed. Cir. 2012).

Petitioner's theory of causation need not be medically or scientifically certain, Knudsen, 35 F.3d at 548-49, but it must be informed by "sound and reliable medical or scientific explanation." Id. at 548; see also Veryzer v. Sec'y of Health & Human Servs., 98 Fed. Cl. 214, 223 (2011) (noting that special masters are bound by both 42 U.S.C. § 300aa-13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both

"relevant" and "reliable"). If petitioner relies upon a medical opinion to support her theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. See Broekelschen, 618 F.3d at 1347 ("The special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories."); Perreira, 33 F.3d at 1377 n.6 (Fed. Cir. 1994) (citing Fehrs v. U.S., 620 F.2d 255, 265 (Ct. Cl. 1980)) ("An expert opinion is no better than the soundness of the reasons supporting it.").

The undersigned does not evaluate whether petitioner put forth a biologically plausible theory explaining how the received flu vaccine could have caused GBS because petitioner failed to establish by preponderant evidence that Mr. Carrino developed GBS. And petitioner put forth no evidence regarding whether the flu vaccine can cause stroke. Thus, petitioner does not prevail on Prong One.

2. Althen Prong Two: Logical Sequence of Cause and Effect

Under Althen Prong Two, petitioner must prove "a logical sequence of cause and effect showing that the vaccination was the reason for the injury." Althen, 418 F.3d at 1278. Under this prong, petitioner must show that the received vaccine "did" cause the alleged injury. Pafford, 451 F.3d at 1354.

Petitioner need not make a specific type of evidentiary showing. That is, petitioner is not required to offer "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 . . . ." Capizzano, 440 F.3d at 1325. Instead, petitioner may satisfy her burden by presenting circumstantial evidence and reliable medical opinions. See id. at 1325-26.

Here, the record evidence strongly militates against a finding that Mr. Carrino suffered from GBS, and petitioner's expert, Dr. Hogen, acknowledged that the flu vaccine was "less likely than not" to have caused a right lateral medullary infarct, the diagnosis with which Mr. Carrino was discharged from the hospital in November of 2006. Tr. at 91-92.

Petitioner does not prevail on Prong Two.

3. Althen Prong Three: Timing

Under Althen Prong Three, petitioner must establish that Mr. Carrino's injury occurred within a time frame that is medically appropriate for the alleged mechanism of harm. See Pafford, 451 F.3d at 1358 ("Evidence demonstrating petitioner's injury occurred within a medically acceptable time frame bolsters a link between the injury

alleged and the vaccination at issue under the 'but-for' prong of the causation analysis."). Petitioner may satisfy this prong by producing "preponderant proof that the onset of symptoms occurred within a time frame for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." De Bazan, 539 F.3d at 1352.

Petitioner may discharge her burden by showing: (1) when the condition for which she seeks compensation first appeared after vaccination and (2) whether the period of symptom onset is "medically acceptable to infer causation." Shapiro v. Sec'y of Health & Human Servs., No. 99-552V, 2011 WL 1897650, at *13 (Fed. Cl. Spec. Mstr. Apr. 27, 2011), aff'd in relevant part, vacated in non-relevant part, 101 Fed. Cl. 532, 536 (2011), aff'd 503 F. App'x 952 (2013) (per curiam). The appropriate temporal association will vary according to the particular medical theory advanced in the case. See Pafford, 451 F.3d at 1358.

Because petitioner failed to establish that Mr. Carrino suffered from the claimed injury, petitioner cannot prove that the onset of the alleged condition occurred within a medically acceptable time frame after receipt of the flu vaccine. Thus, petitioner does not prevail on Prong Three.

IV. Conclusion

For the foregoing reasons, petitioner's claim for Program compensation must fail and the petition **SHALL BE DISMISSED**. The Clerk of Court shall enter judgment consistent with this decision.[34]

IT IS SO ORDERED.

s/Patricia E. Campbell-Smith
Patricia E. Campbell-Smith
Chief Special Master

[34] Pursuant to Vaccine Rule 11(a), entry of judgment is expedited by the parties' joint filing of notice renouncing the right to seek review.