In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 15-1553V
Filed: May 17, 2021
PUBLISHED

JONATHAN PATTON, Petitioner, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	Special Master Horner Decision Dismissing Petition; Influenza Vaccination; Brachial Neuritis; Radiculomyelitis

Michael Andrew London, Douglas & London, P.C., New York, NY, for petitioner.
Claudia Barnes Gangi, U.S. Department of Justice, Washington, D.C., for respondent.

DECISION[1]

On December 21, 2015, petitioner Jonathan Patton[2] filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10-34 (2012),[3] alleging that he suffered brachial neuritis ("BN"), otherwise known as parsonage-turner syndrome ("PTS") or neuralgic amyotrophy ("NA"),[4] as a result of his January 11, 2013 influenza

[1] Because this decision contains a reasoned explanation for the special master's action in this case, it will be posted on the United States Court of Federal Claims' website in accordance with the E-Government Act of 2002. *See* 44 U.S.C. § 3501 note (2012) (Federal Management and Promotion of Electronic Government Services). **This means the decision will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), petitioner has 14 days to identify and move to redact medical or other information the disclosure of which would constitute an unwarranted invasion of privacy. If the special master, upon review, agrees that the identified material fits within this definition, it will be redacted from public access.

[2] In fact, when the petition was filed Mr. Patton was a minor and the action was brought by his mother on his behalf. Petitioner was subsequently substituted as petitioner on February 18, 2016. (ECF No. 10.)

[3] All references to "§ 300aa" below refer to the relevant section of the Vaccine Act at 42 U.S.C. § 300aa-10-34.

[4] Throughout the record of this case, these three terms – i.e. BN, PTS, and NA – are variously used to describe the same basic condition. Notwithstanding any nuances, the parties and experts have treated the terms as interchangeable. For consistency this decision will use the term brachial neuritis except where other of the terms are included in quotation; however, no distinction is intended.

vaccination. (ECF No. 1.) He later amended his claim to allege that he also experienced radiculomyelitis as a result of the same vaccination.[5] (ECF No. 34.)

For the reasons set forth below, I find that petitioner is not entitled to an award of compensation for his injury. Specifically, there is not preponderant evidence that petitioner suffered radiculomyelitis. Although petitioner more likely than not suffered brachial neuritis, petitioner has not established by preponderant evidence that his brachial neuritis was caused by his vaccination.

I. Applicable Statutory Scheme

Under the National Vaccine Injury Compensation Program, compensation awards are made to individuals who have suffered injuries after receiving vaccines. In general, to gain an award, a petitioner must make a number of factual demonstrations, including showing that an individual received a vaccination covered by the statute; received it in the United States; suffered a serious, long-standing injury; and has received no previous award or settlement on account of the injury. Finally – and the key question in most cases under the Program – the petitioner must also establish a causal link between the vaccination and the injury. In some cases, the petitioner may simply demonstrate the occurrence of what has been called a "Table Injury." That is, petitioners may show that they suffered an injury of the type enumerated in the "Vaccine Injury Table," corresponding to the vaccination in question, within an applicable time period following the vaccination also specified in the Table. In such cases, the Table Injury is presumed to have been caused by the vaccine. § 300aa-13(a)(1)(A); § 300 aa-11(c)(1)(C)(i); § 300aa-14(a); § 300aa-13(a)(1)(B).

In many cases, however, the vaccine recipient may have suffered an injury not covered by the Vaccine Injury Table. In these "off-table" cases, an alternative means exists to demonstrate entitlement to a Program award. The petitioner may demonstrate entitlement by showing that the recipient's injury was "caused-in-fact" by the vaccine they received, a showing often referred to as "actual causation." § 300aa-13(a)(1)(B); § 300aa-11(c)(1)(C)(ii). In off-table cases, the presumptions available under the Vaccine Injury Table are inoperative, and the burden is on the petitioner to introduce evidence demonstrating that the vaccination was responsible for the injury in question. *Althen v. Sec'y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005); *Hines v. Sec'y of Health & Human Servs.*, 940 F.2d 1518, 1525 (Fed. Cir. 1991).

To show actual causation, petitioner must satisfy the "preponderance of the evidence" standard, the same standard ordinarily used in tort litigation. § 300aa-

[5] As with petitioner's brachial neuritis, there is some variation in terminology in the record regarding this additional condition. Reference is made most often throughout the record to "radiculomyelitis," but petitioner's first expert referenced the same condition as "myelo-radiculitis." In medical terminology, "myel(o)" is "a combining form denoting relationship to marrow, to the spinal cord, or to myelin" and radiculitis refers to "inflammation of the root of the spinal nerve." (*Dorland's Illustrated Medical Dictionary*, p. 1202, 1547 (33rd ed. 2020).) Conversely, "radicular" generally refers to something that is "of or pertaining to a root" and "myelitis" refers to inflammation of the spinal cord. *Id.* For consistency this decision will reference "radiculomyelitis" except where the other term is used in quotation.

13(a)(1)(A); *see also Althen*, 418 F.3d at 1279; *Hines*, 940 F.2d at 1525. Under that standard, the petitioner must show that it is "more probable than not" that the vaccination caused the alleged injury. *Althen*, 418 F.3d at 1279. The petitioner need not show that the vaccination was the sole cause of the injury or condition, but must demonstrate that the vaccination was a "substantial factor" and a "but for" cause. *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999). This standard has been interpreted to require "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury;" the logical sequence must be supported by "reputable medical or scientific explanation, *i.e.*, evidence in the form of scientific studies or expert medical testimony." *Althen*, 418 F.3d at 1278; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). A petitioner may not receive a Vaccine Program award based solely on his or her assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. § 300aa-13(a)(1).

In what has become the predominant framing of this burden of proof, the *Althen* court described the "causation-in-fact" standard, as follows:

> Concisely stated, Althen's burden is to show by preponderant evidence that the vaccination brought about her injury by providing: (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 proximate temporal relationship between vaccination and injury. If Althen satisfies this burden, she is "entitled to recover unless the [government] shows, also by a preponderance of the evidence, that the injury was in fact caused by factors unrelated to the vaccine."

Althen, 418 F.3d at 1278 (citations omitted). The *Althen* court explained that petitioners are not required to provide medical literature supporting their theory of causation so long as they supply the medical opinion of an expert. *Id*. at 1279-80. The *Althen* court also indicated that Program fact finders may rely upon "circumstantial evidence" to determine causation, a standard it held to be consistent with the "system created by Congress, in which close calls regarding causation are resolved in favor of injured claimants." *Id*. at 1280.

In this case, petitioner alleges that he suffered brachial neuritis and radiculomyelitis as a result of his influenza vaccination. Because neither of these injuries are listed as a Table Injury relative to the flu vaccine, petitioner must satisfy the above-described *Althen* test for establishing causation in fact. *See* 42 C.F.R. § 100.3(a).

II. Procedural History

On December 21, 2015, petitioner's mother filed a petition on his behalf seeking compensation for the injury of brachial neuritis. (ECF No. 1.) Petitioner was later substituted as petitioner once he reached the age of majority. (ECF Nos. 9, 10.) The case was assigned to then Chief Special Master Nora Beth Dorsey on December 22,

2015. (ECF No. 4.) Following an initial status conference on February 18, 2016, petitioner submitted medical records and a Statement of Completion. (Exs. 1-8, ECF Nos. 8, 12, 13.)

On June 20, 2016, respondent filed his Rule 4(c) report identifying a number of missing medical records and recommending against compensation. (ECF No. 15.) Following respondent's Rule 4(c) report, petitioner filed additional medical records and a second Statement of Completion on September 2, 2016. (ECF Nos. 21, 23.)

This case was reassigned to Special Master Laura D. Millman on December 5, 2016. (ECF No. 25.) After the case was reassigned, petitioner filed an expert report from Dr. Thomas F. Morgan, who opined that petitioner had suffered radiculomyelitis. (ECF No. 31, Ex. 12.) Shortly thereafter, on June 19, 2017, petitioner amended his petition to allege that he suffered radiculomyelitis. (ECF No. 34.) Respondent filed a responsive expert report from Dr. Vinay Chaudhry on November 7, 2017. (ECF No. 40, Ex. A.) Dr. Chaudhry disagreed that petitioner had either brachial neuritis or radiculomyelitis. The parties filed further supplemental reports by each expert before this case was reassigned to me on June 6, 2019. (ECF No. 42; Ex. 17 (Morgan supplemental report); ECF No. 44; Ex. G (Chaudhry supplemental report); ECF No. 47 (Notice of Reassignment).)

On July 12, 2019, the parties confirmed that the case was ripe for an entitlement hearing. (ECF No. 50.) On August 20, 2019, I scheduled a two-day entitlement hearing for October 27, 2020. (ECF No. 54.) Petitioner subsequently advised, however, that Dr. Morgan would no longer be participating as an expert in the vaccine program and therefore would not be testifying at the entitlement hearing. Petitioner indicated in a status report on May 11, 2020 that he wished to file a report by a different expert who would testify in place of Dr. Morgan. (ECF No. 55.) Petitioner filed an expert report from Dr. Salvatore Q. Napoli on August 25, 2020. (ECF No. 65, Ex. 21.) Dr. Napoli opined that petitioner suffered both brachial neuritis and radiculomyelitis. Respondent filed a responsive report from Dr. Chaudhry on September 14, 2020. (ECF No. 66-2, Ex. H.)

An entitlement hearing was held on October 27, 2020.[6] (*See* ECF No. 80, Transcript of Proceedings ("Tr"), October 27, 2020). Drs. Napoli and Chaudhry testified. Following the entitlement hearing, petitioner and respondent filed post-hearing briefs on November 17, 2020 and December 10, 2020. (ECF Nos. 81, 82.) This case is now ripe for resolution.

[6] Due to Covid-19, the hearing was held remotely via Webex video conference.

III. Factual History

Apart from a history of nosebleeds, petitioner was a healthy 15-year old boy prior to receiving a flu vaccine on January 11, 2013. (Ex. 2, p. 3.)[7] There are no records of any physical or mental trauma beyond an ankle laceration. (*Id.*) Petitioner received the typical childhood vaccines during his infancy and never experienced any neurological symptoms nor was he ever diagnosed with any neurological or autoimmune condition. (Ex. 1, pp. 1-2; ECF No. 72, p. 1.) Petitioner's pertinent medical history begins on January 11, 2013, when he received the flu vaccine that serves as the basis for this claim.

On January 17, 2013, six days after receiving the flu vaccine, petitioner awoke early in the morning with a nosebleed that was more severe than usual. (Ex. 2, p. 2.) Petitioner was unable to move his arms, describing the feeling as "dead weight." (*Id*.) After alerting his mother of his condition, petitioner was brought and admitted to the All Children's Hospital ("ACH") Emergency Department. (*Id*.) Petitioner's arms were so weak at this point that he was unable to dress himself or fasten his seatbelt on his own. (Ex. 7, p. 59.) He also reported he had experienced an occipital headache, neck pain, and bilateral shoulder pain since waking. (*Id.*)

Petitioner was initially examined around 3:42 AM on January 17, 2013 at ACH ED by Dr. Ricardo Jimenez. (Ex. 7, p. 58.) Dr. Jimenez's neurological exam revealed bilateral upper extremity weakness and left pronator drift. Petitioner was able to lift his legs against resistance without difficulty. (*Id.* at 60.) Petitioner showed downgoing Babinski on the right, and upgoing on the left. (*Id.*) Dr. Jimenez did not observe any ankle clonus. (*Id.*) Petitioner's deep tendon reflexes were intact, sensation was intact to bilateral face in all nerve distributions, upper extremities, and lower extremities. (*Id.*) Petitioner also presented with a severe headache, described as a "6-7/10." (*Id.* at 59.) Dr. Jimenez was concerned that petitioner was experiencing ischemia and ordered a stat CT scan. (*Id*. at 60.)

After petitioner's CT scan came back negative, Dr. Jimenez reported that petitioner's upper extremity weakness was more distal than proximal. (Ex. 7, p. 60.) After his CT, petitioner's Babinski was upgoing on the right, downgoing on the left, and equivocal at times. (*Id.*) Petitioner's pronator drift had resolved and his sensation remained intact. (*Id.*) Petitioner was able to walk on his tiptoes and heels, but had difficulty walking heel-toe. (*Id.*) He was able to stand on his right leg, but had difficulty taking his left heel and going up and down on his right shin which was also apparent when he was lying down. (*Id.*) Petitioner's bloodwork showed a high concentration of carboxyhemoglobin and methemoglobin.[8] (*Id.* at 62, 66.)

[7] Exhibit 2 was not bates-stamped. This decision cites to the pagination generated by CM/ECF.

[8] Carboxyhemoglobin refers to hemoglobin bound to carbon dioxide instead of oxygen. (*Dorland's*., p. 284.) Carboxyhemoglobinemia, the presence of carboxyhemoglobin in the blood, is associated with carbon monoxide poisoning. (*Id*. at 284, 1459.) Methemoglobin is a pigment formed in blood due to oxidation. It is normally present in small amounts, but excess methemoglobin – methemoglobinemia – can be associated with injury or toxic agents and can cause cyanosis and headache as well as dizziness,

Dr. Jimenez recorded differential diagnoses of central nervous system mass, carbon monoxide poisoning, dehydration, electrolyte abnormality, unspecified headache, migraine, tension headache, intracranial hemorrhage, meningitis, post-concussion syndrome, shunt malfunction, and stroke. (Ex. 7, p. 60.) Dr. Jiminez was particularly concerned that petitioner was suffering from ischemia.[9] (*Id.*) Dr. Jiminez ordered brain and spinal MRIs which showed no abnormalities other than a small central disc protrusion at the C5 and C6 level. (*Id.* at 63.)

Petitioner was later examined by Dr. Leslie Carrol. (Ex. 7, p. 72.) During this exam, petitioner was unable to bring his hands up to his face due to weakness, unable to fully clench his fists or extend his fingers, and unable to shrug his shoulders. (*Id.* at 69.) Petitioner showed normal strength in his lower extremities, and abnormal sensation to light touch on his neck, upper chest above the nipple line, and bilateral upper extremities including all of his fingers. (*Id.*) Petitioner reported that a light touch on his chest or left upper back would create a painful, hot sensation. (*Id.*) He experienced normal sensation in his bilateral lower extremities. (*Id.*) He was initially unable to sit up on his own due to pain in his upper back. (*Id.*) Dr. Carroll did not examine petitioner's gait because she was afraid that petitioner would fall due to his weakness. (*Id*.)

Dr. Carrol listed differential diagnoses of spinal cord compression, spinal cord ischemia, acute demyelinating process, Guillain-Barre Syndrome ("GBS"), and carbon monoxide toxicity. (*Id.* at 72.) It was noted that carbon monoxide toxicity was unlikely due to petitioner's normal mental state, metabolic acidosis, respiratory function, and complexion. (*Id.*) GBS was believed to be unlikely because petitioner's symptoms did not ascend from his lower extremities. (*Id.*) Due to the puzzling presentation of petitioner's condition, a neurological consult was scheduled. (*Id.*)

Petitioner was examined by neurosurgeon, Dr. Luis Rodriguez on January 17, 2013. (Ex. 7, p. 77.) Dr. Rodriguez noted that petitioner's weakness had improved and that it is unlikely his symptoms were related to the central disc bulge found on his MRI. (*Id.* at 75-77.)

Petitioner was evaluated by neurologist Dr. Steven Parrish Winesett on January 18, 2013 at 10:09 AM. (*Id.* at 73.) Dr. Winesett noted an unremarkable medical history except for a flu shot several days prior. (*Id*. at 73-74.) Dr. Winesett explained that petitioner did not report severe pain but did have mild shooting pains across his chest the night before. (*Id. at73.*) Petitioner was able to shrug his shoulders at this time and told Dr. Winesett that his condition had been improving since he was admitted. (*Id.*) Petitioner's motor exam showed significant increase in his strength since he had been admitted the previous morning: up to 4/5 in his right deltoid but inability to raise it above 45 degrees, 4+/5 strength in both biceps, 4+/5 strength in his left deltoid, 4+/5 strength

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fatigue, ataxia, dyspnea, tachycardia, nausea, vomiting, and drowsiness. It can lead to coma and sometimes death. (*Id*. at 1130.)

[9] A condition related to loss of blood supply to the spinal cord. (*Dorland's,* p. 949.)

in his hands, and good triceps strength. (*Id.* at 74.) Petitioner's biceps, brachioradialis, patella, and ankle reflexes were all symmetrical and normal. (*Id*.) Petitioner experienced good, although somewhat slow, finger tapping, with a sensory level at his upper chest. (*Id.*)

Dr. Winesett's impression was that petitioner was suffering from "possible Parsonage Turner Syndrome with an autoimmune brachial plexitis." (*Id.*) He also reported, however, that "[o]ther autoimmune processes are also possible." (*Id*. at 74.) Dr. Winesett recommended an MRI of petitioner's brachial plexus to ensure that there were no mass lesions, an EMG and nerve conduction study in two weeks "when it would be more useful," and a spinal tap if petitioner's condition deteriorated. (*Id.* at 74-75.)

Petitioner was seen by Drs. Ronald A. Ford and Jordan Larden on January 18 and 19, 2013. (Ex. 7, pp. 78, 81.) On January 18, Dr. Ford noted that petitioner had 4/5 distal strength in his bilateral upper extremities, 5/5 proximal strength in his bilateral upper extremities, and 5/5 strength in his bilateral lower extremities. (*Id.* at 83.) Dr. Ford noted that the top of his differential diagnosis was "brachial plexopathy secondary to flu vaccination," but also considered autoimmune etiologies with "ANA, CRP, ESR." (*Id.* at 83-84.) On January 19, Dr. Ford observed poor grip strength, but 4/5 distal strength in petitioner's bilateral upper extremities, 5/5 proximal strength in petitioner's bilateral upper extremities, and 5/5 strength in bilateral lower extremities. (*Id.* at 79.) Dr. Ford believed that petitioner's recent flu vaccine was the "most likely etiology" of his bilateral upper extremity weakness and neuropathic pain. (*Id.* at 80.) Dr. Ford also noted that he would consider IVIG or steroids if petitioner's condition deteriorated. (*Id.*)

Petitioner was examined by Dr. Winesett again on January 19 and 20, 2013. (Ex. 7, pp. 84-85.) On January 19, Dr. Winesett noted that petitioner continued to improve, could bring food to his mouth, and was able to scratch his nose. (*Id.* at 85.) Dr. Winesett also noted that petitioner had good biceps strength and could raise his arms over his head. (*Id.*) Petitioner's grip strength had returned to normal, his deltoids were still weak on the right side at approximately 4+/5 at this time, and stronger, but not back to normal on the left. (*Id.*) Dr. Winesett noted that he intended to order a brachial MRI, but that overall, he believed petitioner's weakness was improving and did not see the need for IVIG or plasmapheresis at the time. (*Id.*) The following day, January 20, Dr. Winesett reported additional progress, noting that petitioner could lift his hands above his head, had very strong grip, had much more rapid finger-tapping, but had "a little bit" of pain in the back of his neck and shoulders. (*Id.* at 84.) Dr. Winesett believed that petitioner's condition was a result of "resolving [PTS]." (*Id*.)

Prior to discharge from ACH, petitioner was evaluated by Occupational Therapist Lisa J. Kezar, OTR/L. (Ex. 7, pp. 158-63.) Ms. Kezar found that petitioner presented with a "significant decline" in bilateral upper extremity active range of motion ("AROM"). (*Id*. at 160.) Ms. Kezar noted a right-shoulder-flexion of 1-10 degrees before compensatory scapular elevation and abduction, decreased AROM of the pinky, and 0-40 degrees left-shoulder flexion. (*Id.*) Ms. Kezar also noted that petitioner's bilateral

UE strength was "significantly compromised" with his shoulder movements being the most significantly impacted. (*Id.* at 161.) Ms. Kezar did not test petitioner's sensation, and believed that he may have "some altered sensation due to brachial plexus involvement" but believed that petitioner's condition was "primarily motor based." (*Id.*)

Petitioner was discharged from ACH on January 20, 2013 with a diagnosis of brachial neuritis and neuropathic pain. (Ex. 2, p. 9.) Petitioner was seen by his primary care physician Dr. Lynne Ellis on January 21, 2013 who noted that petitioner had "a reaction to our flu vaccine." (Ex. 1, p. 2.)

Petitioner attended physical therapy at the Therapy and Sports Center in Saint Petersburg, Florida from January 22, 2013 to September 24, 2013. (Ex. 4, pp. 4-34.) He was initially admitted with an injury described as a "result of the flu shot 1/11/13," with onset being noted as starting on January 17, 2013. (*Id.* at 4.) Records from the Therapy and Sports Center show that petitioner's strength and endurance consistently improved shortly after he began physical therapy and remained at around 90 to 100% for the remainder of his therapy. (*See generally*, Ex. 4.)

After he was discharged from ACH, petitioner received several follow-up exams from Dr. Winesett. (Ex. 3, pp. 1, 3, 5.) The first follow-up occurred on January 30, 2013. (*Id.* at 5.) During that exam, petitioner received a nerve conduction study of his left arm and leg, and an EMG of his left and right upper arms, the results of these studies were normal besides a rare fasciculation on petitioner's EMG. (*Id.* at 5-8.)

Petitioner returned to Dr. Winesett on April 15, 2013. (Ex. 3, p. 1.) Dr. Winesett noted that petitioner's bilateral shoulder weakness had resolved after some months, and that he was "back to normal except for his right leg has continued to be numb." (*Id.*) Petitioner's motor exam showed normal tone, bulk, and strength in all four extremities. (*Id.*) However, Dr. Winesett noted that petitioner's sensory exam was "quite abnormal" in that petitioner experienced a distinct change in sensation by about 50% at about one centimeter from the midline in the front and back, and at the T4 nerve root distribution in the front and back. (*Id.*) Dr. Winesett observed normal and symmetric reflexes in all four extremities, downgoing Babinskis, normal gait, and normal coordination. (*Id.*) Dr. Winesett also noted that a Magnetic Resonance Angiography ("MRA") of petitioner's brain and cervical spine was normal. (*Id.* at 2.) On May 1, 2013, Dr. Winesett performed a cervical and thoracic spinal MRI, the results of which were normal apart from the subtle bulge at C5-C6 observed during petitioner's initial MRI on January 18, 2013. (*Id.* at 3.)

Petitioner later saw podiatrist Dr. Kopelman for an unrelated toe contusion where he reported continued right leg numbness. (Ex. 5, p. 7.)

The final record from Dr. Winesett documents a neurological follow up exam on November 13, 2013. (Ex. 8, p. 17.) Dr. Winesett noted at this exam that petitioner's condition had largely resolved apart from some numbness in his mid-chest. (*Id.*) Petitioner's motor exam was normal, but did reveal a slight postural tremor that Dr.

Winesett did not believe was related to petitioner's brachial neuritis. (*Id*.) Dr. Winesett concluded this exam with a diagnosis of slowly recovering brachial neuritis. (*Id*.)

IV. Experts

a. Petitioner's First Expert, Thomas F. Morgan, M.D – Initial Report

Petitioner initially filed an expert report from neurologist Thomas F. Morgan, M.D., to support his claim.[10] (Ex. 12.) Dr. Morgan believed that petitioner's symptoms of hyperreflexia in the lower extremities, bilateral weakness of the upper extremities, and burning paresthesia across the chest support an anatomic diagnosis of radiculomyelitis. (*Id.* at 3.) Dr. Morgan explained that although petitioner's normal EMG and nerve conduction tests do not support a finding of pure brachial plexopathy, such a diagnosis cannot be ruled out without a spinal fluid analysis, which petitioner never received. (*Id.*) Consequently, Dr. Morgan was unable to diagnose or rule out a pure brachial plexopathy based on petitioner's existing medical records. (*Id.*) Instead, Dr. Morgan believed that petitioner suffered radiculomyelitis triggered by an immune response to his January 11, 2013 flu vaccination. (*Id.* at 3-4.) Dr. Morgan opined that the lack of spinal fluid analysis does not impact this clinical diagnosis because the symptoms with which petitioner presented to the ER are consistent with radiculomyelitis. (*Id.* at 4.)

Dr. Morgan further opined that petitioner's immune antibody response from his flu vaccine triggered a mechanism known as "molecular mimicry", causing a cross reaction "with the myelin of his dorsal and ventral nerve roots and a portion of the adjacent spinal cord," causing funicular spinal cord pain from C5 to T4." (*Id.*) He concluded his report by noting that the time of onset of petitioner's post vaccinal reaction is consistent with the time of onset for a molecular mimicry immune response following a vaccine and therefore, the temporal relationship between the condition and the vaccine should not be at issue. (*Id.*)

b. Respondent's Expert, Vinay Chaudhry, M.D. – Initial Report

Respondent filed a report by neurologist Vinay Chaudhry, M.D. in response to petitioner's expert report by Dr. Morgan.[11] (Ex. A.) Dr. Chaudhry did not agree that

[10] Dr. Morgan received his medical degree from Meharry Medical College in 1970. (Ex. 13, p. 1.) He is board certified by the American Board of Independent Medical Examiners, the American Board of Psychiatry and Neurology, and the Rhode Island Board of Medical Licensure and Discipline. (*Id*. at 3.) As of the date of his curriculum vitae, Dr. Morgan is an assistant clinical professor at Brown University where he also serves on the university healthcare team and as a neurology consultant. (*Id*. at 4.) Dr. Morgan has authored numerous publications relating to neurology and neuropathology and has conducted a variety of neurological studies. (*Id*. at 4-5.)

[11] Dr. Chaudhry received his medical degrees in 1980 from the All India Institute of Medical Sciences in New Delhi, India. (Ex. B, p. 1.) He received his MBA in the Business of Medicine from Johns Hopkins University in 2009. (*Id.*) Dr. Chaudhry is licensed in the state of Maryland and holds over a dozen certifications from organizations in the United States, United Kingdom, and India. (*Id.* at 28-29.) Dr. Chaudhry was chief resident of neurology at the University of Alabama School of Medicine, and a clinical

petitioner's clinical presentation and lab results support a diagnosis of either brachial neuritis or radiculomyelitis.

Dr. Chaudhry noted that brachial neuritis is "typically heralded by the abrupt onset of severe pain (≥ 7/10) located in the shoulder or upper arm region that lasts for 2-3 weeks." (Ex. A, p. 3 (citing Jeroen van Eijk, Jan T. Groothuis & Nens van Alfen, *Neuralgic Amyotrophy: An Update on Diagnosis, Pathophysiology, and Treatment Muscle Nerve*, 53 MUSCLE AND NERVE 337 (2016) (Ex. C)).) Dr. Chaudhry indicated that petitioner awoke with weakness instead of severe pain and that petitioner's treating neurologist, Dr. Winesett, specifically noted that petitioner only experienced some shooting pain but never any severe pain. (Ex. A, p. 3.) Dr. Chaudhry also explained that although petitioner suffered headaches that were a 6 to 7 on the pain scale, headaches are not a symptom of brachial neuritis. (*Id.*) Additionally, Dr. Chaudhry explained that recovery from brachial neuritis takes at least several months and often 2-3 years. (van Eijk, Groothuis, & van Alfen, *supra*, at Ex. C.) In petitioner's case, he was showing fluctuating improvement within hours and had almost fully recovered within three months. (*Id.* at Ex. A, pp. 4-5, (citing Ex. 3, p. 4).)

According to Dr. Chaudhry, in cases of brachial neuritis, prominent weakness and wasting occurs in shoulder girdle muscles in the distribution of individual nerves. (*Id.* at 3.) Most often, the long thoracic, suprascapular and anterior interosseous nerves are involved. (*Id.*) Brachial neuritis patients often experience tingling of the superficial radial and lateral antebrachial cutaneous nerves, and a numb path on the lateral upper arm in the axillary nerve distribution. (Chee Keong Chan et al., *Cervical Cord Compression Presenting with Sciatica-like Leg Pain*, 20 EUR. SPINE J. 217 (2011) (EX. E).) Further, when bilateral involvement occurs with brachial neuritis, such involvement is asymmetrical and follows a specific nerve distribution. (Ex. A, p. 4.) Petitioner however, experienced abrupt, symmetrical and bilateral weakness of the upper extremities and did not experience any sensory loss in any of the nerve distributions associated with brachial neuritis. Petitioner also noted neck, chest, and leg symptoms including varying pain, sensory loss, and weakness in these areas, which Dr. Chaudhry explained are inconsistent with brachial neuritis. (*Id.*)

Dr. Chaudhry also noted that injuries to the brachial plexus result in loss of reflexes, weakness, and sensory loss. (*Id.* at 5.) Petitioner however, had normal reflexes and sensation in his biceps, brachioradialis, patella, and ankle. (*Id.*) Additionally, petitioner's nerve conduction, EMG findings, and MRIs were all normal and showed no change in petitioner's brachial plexus which, according to Dr. Chaudhry, "virtually excludes nerve injury." (*Id.*) Dr. Chaudhry explained that petitioner's clinical manifestation, normal EMG, and normal brachial plexus MRI all rule out any injury to the brachial plexus and therefore rule out a brachial neuritis diagnosis. (*Id.*) Dr. Chaudhry

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and research fellow focusing on neuromuscular diseases at Johns Hopkins University. (*Id.* at 2.) He has taught neurology at Johns Hopkins since 1989 and has authored over 120 scholarly articles and textbook chapters on neurological disorders. (Ex. A, p. 1.) Dr. Chaudhry evaluates over 2000 patients a year mostly related to peripheral nerve disease. (*Id.*)

suggested that Dr. Morgan was mistaken when he noted that petitioner developed painful bilateral upper extremity weakness from C5 to T4, because there is no evidence that his initial presentation of weakness was associated with pain. (*Id.*)

Dr. Chaudhry also disagreed with Dr. Morgan's proposed diagnosis of radiculomyelitis. Dr. Chaudhry explained that the first component of radiculomyelitis is radiculopathy which he describes as a nerve root injury associated with abnormal nerve conduction and EMG. (Ex. A, p. 6.) Radiculopathy is also accompanied by reduced or absent reflexes, but Dr. Chaudhry noted that petitioner exhibited normal or increased reflexes. (*Id.*) Further, Dr. Chaudhry noted EMG findings in brachial neuritis cases are identical to those in radiculopathy cases. (*Id.*) Thus, Dr. Chaudhry believed that petitioner's normal EMG rules out radiculopathy in the same way it rules out brachial neuritis. (*Id.*)

According to Dr. Chaudhry, radiculomyelitis involves acute transverse myelitis which is "invariably associated with paralysis in the legs, urinary urgency, incontinence, pathological brisk reflexes, and spasticity in addition to a sensory level." (Chitra Krishnan & Benjamin Greenberg, *Transverse Myelitis*, UPTODATE (2017) (Ex. F).) Because he found no evidence that petitioner had any incontinence, leg paralysis, or abnormal reflexes, Dr. Chaudhry concluded that petitioner's symptoms were inconsistent with myelitis. (Ex. A, p. 6.)

Dr. Chaudhry opined that Dr. Morgan's diagnosis of radiculomyelitis is unreliable because petitioner's medical records lack any objective diagnostic studies supporting such a diagnosis. (*Id.*) He indicated that not only are the clinical findings inconsistent with radiculomyelitis, but petitioner's normal medical imaging explicitly rules out radiculopathy and myelitis. (*Id.*) Dr. Chaudhry concluded that petitioner suffered from neither radiculopathy nor myelitis and thus did not suffer from radiculomyelitis. (*Id.*)

Dr. Chaudhry conceded that petitioner did suffer some abnormal condition, potentially as a result of his high carboxyhemoglobin and high methemoglobin levels. (*Id.* at 7.) However, Dr. Chaudhry opined that petitioner's symptoms, EMG, and MRI all fail to support either a diagnosis of brachial neuritis or radiculomyelitis. (*Id.*)

c. Dr. Morgan's Supplemental Report

In response to Dr. Chaudhry, Dr. Morgan agreed that petitioner's clinical manifestations, normal EMGs, and normal MRIs do not support a diagnosis of brachial neuritis, which he had initially declined to rule out. (Ex. 17, p. 1.) Dr. Morgan disagreed with Dr. Chaudhry however, regarding whether petitioner originally complained of upper extremity pain. (*Id.*) Dr. Morgan cited records from ACH, which note that petitioner was suffering from neck and shoulder pain in addition to upper extremity weakness. (*Id.* (citing Ex. 7, p. 76.)) He suggested that Dr. Chaudhry confused radiculomyelitis with classic transverse myelitis, and that petitioner's neck and shoulder pain, while inconsistent with transverse myelitis, are nonetheless consistent with radiculomyelitis. (Ex. 17, p. 1.)

Dr. Morgan believed that although there is no evidence in the record clearly supporting his preferred diagnosis, this is only because petitioner did not receive the tests necessary to collect such evidence. (*Id.* at 1-2.) Dr. Morgan explained that the pathology of radiculopathy can be evidenced by an abnormal EMG, but that neither petitioner's ascending sensory tracts in the spinal cord nor his motor nerve roots at C8 through T4 were tested by somatosensory evoked responses or EMG and nerve conduction. (*Id.* at 2.) Because petitioner's pain was referred up to the C5 level in the spinal cord and did not involve the C5 nerve roots itself, Dr. Morgan believed that EMG and nerve conductions for the brachial plexus or peripheral radiculopathy would not have detected any abnormalities even if these tests were ordered. (*Id.*)

Dr. Morgan suggested that, contrary to Dr. Chaudhry's report, reduced or absent reflexes would "not necessarily be reflected in a myeloradiculitis located from C5 through T4." (*Id.*) He did concede Dr. Chaudhry's point however, that petitioner did not have a classic presentation for acute transverse myelitis. (*Id.*) Instead, Dr. Morgan argued that petitioner had a "subacute post vaccinal myelo-radiculitis that was an atypical presentation," sparing the C5 nerve. (*Id*.) Dr. Morgan noted that the medical literature shows that atypical cases often require repeated examinations to localize abnormalities to the spinal cord. (*Id.* (citing Allan H. Ropper, Martin A. Samuels & Joshua P. Klein, *Adams and Victor's Principles of Neurology* (10th ed. 2014) (Ex. 18)).) Dr. Morgan explained that petitioner did not receive repeat cervical or thoracic MRI scans with gadolinium, nor somatosensory evoked response tests for the spinal cord tracts, any of which, in his opinion, would have localized petitioner's injury to the spinal cord. (Ex. 17, p. 2.)

Dr. Morgan believed that petitioner's myelitis in effect simulated brachial neuritis, which led to that diagnosis being made by the treating physicians. Although petitioner's normal EMG, normal MRI studies, and clinical presentation were against brachial neuritis, the tests run on petitioner did not observe the spinal cord from C5 to T4, which Dr. Morgan seemingly presumes would have confirmed petitioner's myelitis. (*Id.*) Dr. Morgan believed that petitioner's condition is anatomically located in the spinal cord consistent with a post vaccinal immune mediated neurologic syndrome, and not his brachial plexus. (Ex. 17, p. 3.)

Dr. Morgan disagreed with Dr. Chaudhry's conclusion that petitioner's symptoms were unclear and of uncertain cause. According to Dr. Morgan, the first step of basic diagnosis is to "localize patient's complaints, symptoms and signs and to determine the neuro-anatomic location of the problem." (*Id.*) Dr. Morgan believed that the anatomic location of petitioner's symptoms was "the spinal cord myelitis that effected the exiting nerve roots to account for his myelo radiculitis." (*Id.*) According to Dr. Morgan, this location would indicate a "subacute, post vaccinal myelo-radiculitis." (Ex. 17, p. 3 (citing Ex. 20).) Dr. Morgan suggested that further diagnostic testing such as a high field MRI, somatosensory response testing, and spinal fluid analysis would have been helpful, but that even without these studies, he believed that petitioner suffered from "a post vaccinal myelo-radiculitis and not a diagnosis of uncertain cause." (Ex. 17, p. 3.)

d. Dr. Chaudhry's First Supplemental Report

Dr. Chaudhry's first supplemental report specifically responded to points raised by Dr. Morgan's supplemental report. First, Dr. Chaudhry noted the inconsistency between Dr. Morgan's first report, wherein he stated that petitioner presented with clinical findings of myelitis and brachial neuritis, and Dr. Morgan's supplemental report, wherein he conceded that petitioner's clinical presentation does not support a diagnosis of brachial neuritis. (Ex. G, p. 1.) Dr. Chaudhry further stressed that none of petitioner's treating physicians entertained a radiculomyelitis diagnosis even though a variety of other neurological conditions were considered. (*Id.*) Additionally, Dr. Chaudhry reiterated that petitioner never had a spinal fluid evaluation, never had an MRI which supported such a diagnosis, and was never treated for spinal cord inflammation which is a common symptom of myelitis. (*Id.* at 1-2.)

Dr. Chaudhry pointed out that Dr. Morgan's conclusion that petitioner's EMG was normal because the C5 nerve was spared by his radiculomyelitis is incorrect. According to Dr. Chaudhry, several symptoms indicate a C5 nerve root involvement including weakness in petitioner's biceps, deltoids, and hands. (*Id.* at 2.) Further, Dr. Morgan suggested twice in his first report that petitioner's C5 nerve root was involved, and again in his supplemental report. (*Id.*) Dr. Chaudhry explained that Dr. Morgan incorrectly noted that reduced or absent reflexes are not reflected in radiculomyelitis from C5 to T4. (*Id.*) However, physicians routinely test reflexes of the biceps, brachioradialis, and triceps when diagnosing radiculomyelitis. (*Id.*) Each of these muscles involve either the C5, C6, or C7 nerve root, and therefore, reduced or absent reflexes from C5 to T4 can indeed be reflected in radiculomyelitis. (*Id.*)

Dr. Chaudhry concluded his report by noting that Dr. Morgan's diagnosis of myelitis is not supported by any of petitioner's medical records, and in fact, can be excluded by petitioner's multiple normal MRIs. (*Id.* at 3.) Further, Dr. Chaudhry pointed out that Dr. Morgan provided no references nor support from the record for his finding of "atypical myeloradiculitis." (*Id.*) First, the degree of myelitis that Dr. Morgan references involves eight spinal cord levels and should have shown some abnormalities on petitioner's MRIs. (*Id.*) Additionally, a Somatosensory Evoked Potential study was ordered on May 8, 2013, and while the results of this study are missing from the record, Dr. Chaudhry noted that there were no notes referencing the test on a follow up by Dr. Winesett. (*Id.*) Based on the above findings, Dr. Chaudhry indicated he is unable to agree with Dr. Morgan's diagnosis of atypical radiculomyelitis. (Ex. G, p. 3.)

e. Petitioner's Second Expert, Salvatore Napoli, M.D.

Following Dr. Morgan's withdrawal as an expert in the case, petitioner filed a report by neurologist Dr. Salvatore Napoli.[12] (ECF No. 65; Ex. 21.) Dr. Napoli

[12] Dr. Napoli received his medical degree from Albany Medical College in 1999. (Ex. 22, p. 2.) He is currently the president and owner of the Neurology Center of New England, specializing in MS care with focuses on other neurological diseases. (*Id.* at 1.) He also holds staff positions at Beth Israel at Milton

reintroduced the brachial neuritis diagnosed by petitioner's treating physicians as a relevant condition, beginning his report by describing brachial neuritis as a condition characterized by severe onset of regional pain, muscle weakness, and wasting. (Ex. 21, p. 4.) He explained that most patients experience these symptoms in a single event, but approximately 12% of patients experience multiple bouts or recurrences involving either the same limb or the contralateral limb. (*Id*. (citing Mark A Ferrante & Francesc Graus, *Neuralgic Amyotrophy*, NEUROLOGY MEDLINK (2020) (Ex. 23) (herein "Medlink").) According to Dr. Napoli, these recurrent cases may also involve the same nerves or have a different distribution of nerve involvement. (Ex. 21, p. 4.) Dr. Napoli noted that in cases of bilateral symptoms like petitioner experienced, the involvement is "sequential in the majority and simultaneous in the minority." (*Id.* (citing Medlink *supra* at Ex. 23.)) Dr. Napoli believed that the medical literature suggests both sides can be involved at the same time and petitioner's physicians seemed aware of this given their diagnosis of brachial neuritis. (Ex. 21, p. 4.) Dr. Napoli further explained that available evidence suggests that an autoimmune pathogenesis is likely related to a genetic susceptibility that most commonly generates an axonal-loss lesion. (*Id.*) (citing Medlink *supra* at Ex. 23.)

According to Dr. Napoli, "it is a known fact" that brachial neuritis has a wide variety of clinical presentations. (Ex. 21, p. 4.) However, the distinctive features include a recognized trigger, regional pain, and regional muscle weakness. (*Id.*) Even when this presentation is inconclusive, the disorder is typically recognized by the important features of pain and muscle weakness which are almost always present. (*Id.*) In a study of 281 brachial neuritis patients, only 8% presented without pain. (*Id.* (citing Mark A. Ferrante & Asa J. Wilbourn, *Lesion Distribution Among 281 Patients with Sporadic Nerualgic Amyotrophy*, 55 MUSCLE & NERVE 858 (2017) (herein "Ferrante and Wilbourn") (Ex. 27).) Although reports found that a majority of patients presented with focal pain, Dr. Napoli noted that a minority of patients can present with focal sensory loss like petitioner. (Ex. 21, p. 4 (citing Ferrante & Wilbourn, *supra*, at Ex. 27, p. 2.)

Dr. Napoli explained that the Ferrante study identified a trigger in 73% of brachial neuritis patients. (Ex. 21, p. 4.) Dr. Napoli indicated that the study reported that 5% of the cases were associated with a vaccination, and 24% of cases were associated with an upper respiratory or nondescript flulike illness. (*Id.*) Dr. Napoli suggested that these vaccine and viral associations support the assertion that brachial neuritis can have an autoimmune etiology. (*Id.*) He also noted that the Medlink study found that the most commonly affected nerves in brachial neuritis cases are the suprascapular nerve and the long thoracic nerve, both of which are pure motor nerves. (*Id.*) According to Dr. Napoli, petitioner's distribution of weakness suggests that at least one, and potentially both of these nerves were involved. (*Id.*)

Hospital and Steward Norwood Hospital. (*Id.*) Dr. Napoli was also an instructor in neurology at the Brigham and Women's Hospital MS Center at Harvard Medical School. (*Id.*) He holds a Massachusetts full medical license and is certified by the American Board of Psychiatry and Neurology. (*Id.* at 2.) He has published three papers on multiple sclerosis and neuromyelitis optica, and participated in 17 different clinical trials and research projects focused a variety of neurological disorders. (*Id.* at 3-5.)

Dr. Napoli also agreed, however, that petitioner also suffered radiculomeylitis, defining the term as "inflammation that affects the spinal cord and nerve roots and can cause pain, sensory disturbance and weakness, among other symptoms." (*Id.* at 5.) He indicated that a diagnosis of radiculomyelitis can sometimes, but not always, be supported by diagnostic tests like MRIs. (Ex. 21, p. 5.) He also explained that "there have been case reports showing MRI negative cases of myelopathy and clinical transverse myelitis." (*Id.*) The key to diagnosis, Dr. Napoli opined, is looking at how the patient presents clinically instead of depending solely on negative diagnostic tests. (*Id.*)

Dr. Napoli opined that petitioner's clinical presentation was consistent with both brachial neuritis and radiculomyelitis. He explained that a radiculomyelitis diagnosis would be supported by petitioner's T4 sensory level with alternating upper motor neuron signs that are seen in transverse myelitis (e.g. petitioner's Babinski signs). (*Id.*) Dr. Napoli admitted that petitioner did not present with classic transverse myelitis symptoms, but that his symptoms, complaints, and physical exam support a finding of radiculomyelitis. (*Id.*) According to Dr. Napoli, petitioner's symptoms in his right arm and right leg, sensory level involving the chest and nipple, hemisensory loss, and signs of Babinski localized his lesion to the C5-T4 level are consistent with radiculomyelitis. (*Id.*)

Dr. Napoli believed that molecular mimicry explains how petitioner's flu vaccination caused his injury. He explained that molecular mimicry occurs where, after vaccination or infection, a patient's immune system will target not only the vaccine antigens, but also host antigens present in the patient's body which share any homology with the vaccine antigens. (Ex. 21, p. 5 (citing Riita Lahesmaa et al., *Molecular Mimickry Between HLA B27 and Yersinia, Salmonella, Shigella and Klebsiella within the Same Region of HLA α1-helix*, 86 CLINICAL & EXPERIMENTAL IMMUNOLOGY 399 (1991) (EX. 24)).) In these cases, the patient's immune system will target itself in addition to the vaccine antigens and create an immune response that may cause various neurological disorders. (Ex. 21, p. 5.)

Dr. Napoli noted that there have been many examples of flu vaccines triggering peripheral and central nervous system disorders similar to brachial neuritis and radiculomyelitis, with Guillain-Barre Syndrome ("GBS") being the most prominent. (*Id.*) A study conducted by Schonberger et al. in the 1970's found 532 GBS patients had received flu vaccines prior to the onset of their symptoms. (Lawrence Schonberger et al., *Guillain-Barre Syndrome Following Vaccination in The National Influenza Immunization Program, United States, 1976-1977*, AM. J. OF EPIDEMIOLOGY 105 (1979) (Ex. 25).) In 1976, a statistically significant association was found between an increased risk for GBS and the novel swine flu vaccine. (Ex. 21, p. 6.) A third study by Haber et al. which found case reports of GBS following various types of vaccine administrations, including influenza, rabies, oral polio, and hepatitis. (Penina Haber et al., *Vaccines and Guillain-Barre Syndrome*, 32 DRUG SAFETY 309 (2009) (Ex. 26).)

In addition to reports of GBS, Dr. Napoli cited several case reports of acute brachial neuritis reported after various vaccinations, including influenza. (Ex. 21, p. 6.) In one case, a 66-year-old man presented with acute brachial neuritis with an onset beginning approximately one week after receiving the flu vaccine. (Jimmy D. Miller et al., *Acute Brachial Plexus Neuritis: An Uncommon Cause of Shoulder Pain*, 62 AM. FAMILY PHYSICIAN 2067 (2000) (Ex. 28).) This same study noted that up to 15% of cases had been reported to occur following vaccinations. (*Id.*) A second case reported a 46-year-old woman whose symptoms began within days of receiving a flu vaccine. (Maliha Farhana Shaikh, Tanya Jane Baqai & Hasan Tahir, *Acute brachial neuritis following influenza vaccination*, BMJ CASE REP. (2012) (Ex. 29).) A third case reported a 65-year-old man who experienced symptoms within six days of vaccination, and a final case described a 61-year-old male who experienced symptoms within six weeks after vaccination. (Otto Hansen, *Akut brakial neuropati efter influenzavaccination*, 167 UGESKRIFT FOR LAEGER 1297 (2005) (Ex. 30); D. H. Marks, *Parsonage-Turner Syndrome Associated With Influenza Vaccination: A Case Report With Discussion Of Vaccination Neurologic Complications And Causation*, 21 THE INTERNET J. OF NEUROLOGY 1 (2019) (EX. 31).) Dr. Napoli cited these cases to conclude that there is a reasonably possible link between vaccines and neurological disorders such as brachial neuritis and radiculomyelitis. (Ex. 21, p. 6.)

Dr. Napoli indicated that GBS cases have developed within a five-week period following vaccination. (Schonberger et al., *supra*, at Ex. 25.) He suggested this GBS timeline is consistent with the previously cited case reports of both brachial neuritis and radiculomyelitis, which showed onsets ranging from several days to weeks following vaccination. (Ex. 21, p. 6.) Because the onset of petitioner's symptoms occurred within six days of his vaccination, Dr. Napoli concluded that petitioner's injury is consistent with the appropriate timeline for post-vaccine neurologic injuries. (*Id.*) Based on the opinion of Dr. Winesett as well as the timeframe for brachial neuritis onset following vaccination, it is his opinion that petitioner's flu vaccination caused his brachial neuritis. (*Id.* at 7.) Dr. Napoli concluded that petitioner's radiculomyelitis and brachial neuritis were both caused by his January 11, 2013 flu vaccination. (*Id.*)

f. Dr. Chaudhry's Second Supplemental Report

In his final report, Dr. Chaudhry addressed Dr. Napoli's conclusion that petitioner suffered from brachial neuritis. Dr. Chaudhry indicated that the onset of petitioner's symptoms are inconsistent with brachial neuritis, reiterating that brachial neuritis is associated with the abrupt onset of severe pain in the shoulder or upper arm region lasting for two to three weeks, followed by weakness in the distribution of the nerves of the plexus. (Ex. H, p. 1.) Petitioner experienced weakness instead of severe pain and began to recover within 24 hours. (*Id.*) Dr. Chaudhry further explained that brachial neuritis is a dysfunction of one or two nerves of the brachial plexus which results in weakness and atrophy of those muscles on a single side. (*Id.*) In petitioner's case, the entire brachial plexus was involved, and his symptoms occurred bilaterally. (*Id.*) According to Dr. Chaudhry, brachial neuritis also involves axonal degeneration and atrophy which recovers through a drawn-out regeneration process typically lasting six

months to a year. (*Id.* at 2.) Petitioner however, showed significant signs of improvement within 24 hours. (*Id*.) Further, Dr. Chaudhry noted that none of petitioner's EMG or MRI findings suggest any abnormalities which should be present in a brachial neuritis patient. (*Id.*)

Dr. Chaudhry accepted Dr. Napoli's argument that a small percentage of brachial neuritis cases involve bilateral involvement and present without pain, but noted that "none of the clinical features, electrophysiological features, [or] imaging features [were] indicative of brachial neuritis." (*Id.*) He further indicated that Dr. Napoli failed to explain petitioner's rapid improvement, normal imaging, and fluctuating symptoms and thus, has not presented any evidence that this was in fact an atypical case of brachial neuritis. (*Id.*)

Dr. Chaudhry noted that Dr. Napoli "does not provide a single reference" to the term "radiculomyelitis" but defines it as "inflammation that affects the spinal cord and nerve root." (*Id.*) Consequently, Dr. Chaudhry's discussion of Dr. Napoli's radiculomyelitis diagnosis focused on both radiculitis (inflammation of the C5 to T4 nerve roots) and myelitis (inflammation of the C5 to T4 spinal cord).

Dr. Chaudhry does not believe that petitioner suffered radiculitis because there were no EMG changes in the associated muscles and his MRIs were normal. (*Id.*) There was also no sensory loss in petitioner's arms, and petitioner's reflexes were normal. (*Id*.) Further, petitioner's rapid improvement within 24 hours suggests that he was not suffering from nerve root inflammation because, if that were the case, it would certainly not improve so much within one day. (*Id.* at 3.) Finally, the only finding that suggests a nerve root injury is petitioner's numbness at T4 on the right side. (*Id.*) However, Dr. Chaudhry noted that this symptom only appeared on an April 2013 follow up exam and was no longer significant by November 2013. (*Id.*) Consequently, Dr. Chaudhry concluded that nothing in petitioner's medical records support a radiculitis diagnosis. (*Id.*)

Dr. Chaudhry further explained that spinal cord disorders such as myelitis are characterized by motor, sensory, and autonomic abnormalities below the spinal cord injury. (Ex. H, p. 3.) Dr. Chaudhry noted that a spinal cord injury from C5 to T4 should cause bilateral lower extremity weakness, but that petitioner had normal lower extremity strength. (*Id*.) Additionally, a C5 to T4 injury should cause bladder incontinence, sensory loss up to the cervical spinal cord level, pathologically brisk reflexes, and Babinski, but petitioner did not exhibit any of these symptoms at the time that he was thought to have a lesion at the T4 sensory level. (*Id.*)

Most importantly, Dr. Chaudhry opined that spinal cord inflammation demonstrated by either a CSF pleocytosis elevated IgG index, or MRI evidence is a required criterion for transverse myelitis diagnosis. (*Id.* at 4.) Although petitioner did not have a spinal tap, two MRIs failed to show any spinal cord involvement. (*Id.*) Further, petitioner had no sensory, motor, or autonomic dysfunction attributable to the spinal cord either on admission or discharge. (*Id.*) Instead, he developed a transient

sensory level three months after his discharge. (*Id.*) Dr. Chaudhry opined that petitioner's two normal MRIs specifically exclude a diagnosis of myelitis, and therefore, Dr. Napoli's radiculomyelitis diagnosis is incorrect. (*Id.*)

Dr. Chaudhry suggested that while Dr. Napoli discusses molecular mimicry as a hypothesis, he failed to provide any reference showing that brachial neuritis and radiculomyelitis have a molecular mimicry pathogenesis. (*Id.*) Dr. Chaudhry explained that while Dr. Napoli is correct that the flu vaccine has been linked to increased cases of GBS, these findings have no bearing on brachial neuritis or radiculomyelitis. (*Id.*) Dr. Chaudhry stressed that the Institute of Medicine has reviewed all epidemiological and mechanistic evidence of flu related transverse myelitis and brachial neuritis but only found a temporal, not a causal association. (*Id.* at 4-5.)

g. Entitlement Hearing

As noted above, Drs. Napoli and Chaudhry also testified during the October 27, 2020 entitlement hearing. Each provided testimony consistent with their above-discussed opinions. That testimony is not separately summarized, but is referenced and included in the analysis below.

V. Diagnosis

Given the complexity of petitioner's clinical presentation, the parties raise as a threshold issue the question of whether I must first consider petitioner's correct diagnosis before proceeding to a causation-in-fact analysis under *Althen*. (*See* ECF Nos. 81, 82.) Citing prior Circuit decisions in *Lombardi* and *Broekelschen*, respondent argues that "when the existence of the claimed injury is in question, a threshold issue preceding the causation analysis under *Althen v.* [*Sec'y of Health & Human Servs.*], 418 F.3d 1274 (Fed. Cir. 2005), is whether petitioner has established that he suffers from the very injury that he alleges was caused by the vaccine." (ECF No. 82, p. 1) (citing *Lombardi v. Sec'y of Health & Human Servs.*, 656 F.3d 1343 (Fed. Cir. 2011); *Broekelschen v. Sec'y of Health & Human Servs.*, 618 F.3d 1339, 1346 (Fed. Cir. 2010)).) Petitioner, on the other hand, argues that *Lombardi* and *Broekelschen* are inapplicable in this case because such precedent only applies when the respondent has presented evidence to support an alternative diagnosis. (ECF No. 81, pp. 1-2.) Here, respondent has not presented any such evidence and petitioner argues that I am therefore not required to find that any specific diagnoses exist before moving on to the *Althen* analysis.

Petitioner is correct that *Broekelschen* itself involved a dispute between the parties as to the petitioner's correct diagnosis. 618 F.3d at 1345. It was in that context that the Federal Circuit first explained that the analysis of the three *Althen* prongs is conducted relative to the alleged injury and thus, "the question of causation turn[s] on which injury [the petitioner] suffered." *Id.* at 1346. The Court concluded that "identifying the injury is a prerequisite to the [*Althen*] analysis" and that it was therefore "appropriate for the special master to first find which of [petitioner's] diagnoses was best supported

by the evidence presented . . . before applying the *Althen* test . . . ." *Id.* However, in *Lombardi*, the Federal Circuit further considered the reasoning of *Broekelschen* and applied it to a case, similar to the instant case, in which it was the petitioner who came forward with multiple diagnoses. *Lombardi*, 656 F.3d at 1352-53. The Court explained:

> Lombardi argues that by finding that she had failed to prove the existence of any of her injuries, and therefore declining to conduct an *Althen* analysis on any of her alleged injuries, the special master penalized her for alleging that she suffered from more than one injury. Lombardi misstates the special master's reasoning. The special master did not require Lombardi to narrow the number of alleged injuries to one. But the statute places the burden on the petitioner to make a showing of at least one defined and recognized injury. Here, the special master merely found that Lombardi had failed to meet her burden to show by a preponderance of the evidence that she suffered from any medically recognized "injury," not merely a symptom or manifestation of an unknown injury.

Id. at 1353; *see also Lasnetski v. Sec'y of Health & Human Servs.*, 696 Fed. Appx. 497 (Fed. Cir. 2017).

Petitioner stresses the competing consideration that "special masters are not 'diagnosing' vaccine-related injuries," but instead determining whether there is vaccine causation by preponderant evidence based on a review of the record as a whole. (ECF No. 81, pp. 1-2 (quoting *Knudsen v. Sec'y of Health & Human Servs*., 35 F.3d 543, 549 (Fed. Cir. 1994).) In that regard, petitioner intimates that I should conduct an *Althen* analysis, and find in petitioner's favor, because petitioner has suffered autoimmune or immune-related neurological injuries broadly. Critically, however, petitioner has not articulated any basis for considering causation-in-fact based on anything other than his alleged diagnoses of brachial neuritis and/or radiculomyelitis.

Petitioner explained his showing under *Althen* as follows: "(1) Johnathan suffered from Parsonage-Turner Syndrome and radiculomyelitis beginning on January 17, 2013; (2) a medical theory casually connecting the influenza vaccination and *his two neurological injuries* exists; (3) a temporal association exists between the vaccine and *both neurological injuries* . . .; and (4) a logical sequence of cause and effect exists." (ECF No. 77, p. 42; ECF No. 81, p. 4 (emphasis added).) Moreover, during the hearing, petitioner's testifying expert, Dr. Napoli, repeatedly explained that his opinion was that petitioner's condition was explained as concurrent brachial neuritis and radiculomyelitis. (Tr. 12, 48.) (For his part, petitioner's prior expert, Dr. Morgan, relied on the presence of radiculomyelitis but not brachial neuritis.) I specifically asked Dr. Napoli if he would still opine that petitioner's injury was vaccine-caused in the absence of a diagnosis of either brachial neuritis or radiculomyelitis. (Tr. 116-17.) He responded that he would, but when asked to explain the basis for that opinion he indicated that:

> Looking at his clinical scenario, this was a healthy 15-year-old boy who received a vaccine and within one week of the vaccine, mounted an immune response that now created this constellation of symptoms *consistent with*

> *what I believe to be radiculomyelitis and the brachial neuritis*, which he was struggling with for a period of time and, I believe, still struggles to this day. And I believe the trigger was the vaccine.

(Tr. 117 (emphasis added).) Thus, both petitioner's own framing of his case and his experts' causal opinions are explicitly predicated on the presence of brachial neuritis and/or radiculomyelitis. Indeed, by his own description Dr. Napoli confirmed in effect that he cannot separate his ultimate causal opinion from his own conclusion that petitioner's clinical presentation is consistent with brachial neuritis and/or radiculomyelitis. Moreover, brachial neuritis and radiculomyelitis are two very different explanations for petitioner's symptoms, the former being a peripheral axonal nerve injury and the latter representing spinal cord inflammation and possible demyelination. As Dr. Chaudhry expressed during the hearing, an anatomical diagnosis is a threshold question to discussing etiology. (Tr. 184.) He suggested that to jump to etiology without resolving anatomical diagnosis is to say "'Oh, I know what's causing it,' without even telling me what -- I can't even prove what it is, it's just leap of faith." (*Id*.)

In sum, petitioner has not articulated a basis for finding causation in the absence of either a brachial neuritis or radiculomyelitis diagnosis and the question whether petitioner suffered from radiculomyelitis, brachial neuritis, or a combination of both, would result in distinct analyses under the *Althen* test. For these reasons, it is appropriate to first resolve whether there is preponderant evidence that one or both of these two conditions is present in this case before assessing whether petitioner has met his burden under the *Althen* test.

a. Radiculomyelitis

As explained above, petitioner's initial expert, Dr. Morgan, opined that petitioner's condition is entirely explained by radiculomyelitis. His second expert, Dr. Napoli, opined that petitioner's condition is explained in part by radiculomyelitis, but also by brachial neuritis. Dr. Chaudhry opined on behalf of respondent that petitioner's condition is not consistent with either condition. Whereas petitioner was diagnosed with brachial neuritis by his treating physicians, the suspicion of radiculomyelitis was not explored by those physicians and stems only from his experts' opinions. However, those opinions are not well supported by petitioner's medical history.

Significantly, despite multiple MRI studies, there is no objective evidence supporting the proposed radiculomyelitis diagnosis. Petitioner underwent MRI studies on January 17, 2013 (brain and cervical spine) and May 1, 2013 (cervical and thoracic spine), neither of which revealed any abnormalities consistent with radiculomyelitis. (Ex. 7, pp. 71, 246-47; Ex. 3, p. 3.) Dr. Napoli testified that petitioner's initial cervical MRI would not have detected a lesion at the level he describes and a thoracic MRI was not performed until later. (Tr. 76, 82.) However, Dr. Chaudhry explained that the report of symptoms above the nipple line implicates an area higher on the spinal cord than Dr. Napoli indicated. (Tr. 149.) Dr. Chaudhry opined, contrary to both Dr. Napoli and Dr. Morgan, that inflammation of the nerve roots should have been visible on petitioner's

first MRI with contrast enhancement from C5 through T1 if it were present. (Tr. 155.) Dr. Chaudhry explained that MRI is very sensitive to detecting spinal cord inflammation. (Tr. 217.) Moreover, although the thoracic MRI was later, it was also performed at a time when Dr. Winesett was specifically screening for a potential spinal explanation for petitioner's presentation of active sensory symptoms. (Ex. 7, pp. 7-8.)

Dr. Napoli also opined that a normal spinal MRI does not rule out the possibility of a "small" lesion. (Tr. 53.) However, it would still remain the case that the MRI evidence of any lesion is lacking and, especially in light of all of the additional points below, neither Dr. Napoli nor Dr. Morgan is persuasive in suggesting that any spinal lesion should be inferred based solely on clinical presentation. Dr. Chaudry also explained that a normal MRI in the context of myelitis would be "very rare" and, if it was suspected, spinal tap and EMG, would provide further opportunities for confirmation.[13] (Tr. 215-16, 219.) However, no such confirmation exists in this case. Additionally, in response to Dr. Morgan's opinion Dr. Chaudhry questioned the seeming contrast between the extent of symptoms implicated by Dr. Morgan's (and subsequently Dr. Napoli's) theory, which he described as symptoms stemming from eight spinal levels, and the suggested ability of the proposed spinal lesion to nonetheless be small or mild enough to evade MRI detection. (Ex. G, pp. 2-3.)

In addition to the lack of any objective evidence upon imaging, Dr. Napoli was also unable to identify significant evidence of spinal involvement based on petitioner's overall presentation. With regard to petitioner's symptom presentation, Dr. Napoli described the following clinical symptoms of radiculomyelitis: band-like sensation across the chest,[14] hyperreflexia including intermittent bilateral upgoing toes (i.e. Babinski

[13] Specifically, Dr. Chaudhry indicated that nerve root damage at C5 should also have been detected by petitioner's EMG test. (Tr. 176.) Dr. Chaudhry explained that EMG would not reveal etiology, but could detect the nerve root damage constituting the radiculitis. (Tr. 215.) Dr. Chaudhry suggested that EMG may be considered where radiculopathy is still suspected after a normal MRI. (Tr. 216.) Dr. Morgan conceded that radiculopathy can be detected by EMG, but opined that "[petitioner's] ascending sensory tracts in the spinal cord and the C8 through T4 motor nerve roots were not tested by . . . his EMG and nerve conduction. (Ex.17, pp. 1-2.) He opined that petitioner's pain was referred up to the C5 level rather than the C5 root itself being affected. (*Id.*) Dr. Chaudhry indicated, however, that if petitioner had weakness in his biceps, deltoids, and hands that was due to a spinal cord lesion, then the C5 nerve would have been involved. (Ex G, p. 2.) Notably, Dr. Morgan did initially opine that petitioner had myelitis affecting the C5 level. (Ex. 12, p. 4.) It was only after Dr. Chaudhry first raised the fact of the EMG results counseling against a radiculomyelitis diagnosis (Ex. A, p. 6) that Dr. Morgan sought to explain petitioner's symptom presentation as referred pain (Ex. 17, pp. 1-2). Dr. Morgan indicated that reduced or absent reflexes cited by Dr. Chaudhry "would not necessarily" be reflected in radiculomyelitis at C5 through T4. (Ex. 17, pp. 1-2.) Nonetheless, especially given Dr. Morgan's acknowledgement that radiculopathy can be detected on EMG, Dr. Chaudhry is persuasive in discussing what would normally be expected whereas Dr. Morgan appears to strain to harmonize the EMG results with his opinion.

[14] On cross-examination, Dr. Napoli confirmed that his opinion that petitioner experienced a band-like sensation across the chest was based on a notation that petitioner was experiencing "neuropathic pain" at the level of his chest and nipples. (Tr. 75.) However, "neuropathic pain" is not specific to either the central or peripheral nerves. In his earlier report, Dr. Napoli had discussed neuropathic pain in the context of petitioner's brachial neuritis diagnosis from Dr. Winesett (Ex. 21, p. 4) and separately discussed sensory changes at T4 as evidence of radiculomyelitis (*Id.* at 6).

sign), numbness of the distal limbs, and bladder symptoms. (Tr. 30.) To Dr. Napoli, the combination of peripheral and central nervous system findings and the location of the band-like sensation indicates that inflammation existed at the C5 to T4 area of the spinal cord. (Tr. 31.) Dr. Napoli indicated that ankle clonus, which can be seen in conjunction with the Babinski sign, is a physiologic indicator of a lesion in the central as opposed to peripheral nervous system. (Tr. 32.) Bilateral ankle clonus raises a high suspicion for a spinal cord lesion.[15] (Tr. 33.)

However, Dr. Chaudhry explained that there was no leg weakness upon petitioner's initial neurological exam and sensation was intact. (Tr. 142-43, 145.) Dr. Chaudhry also cautioned that the Babinski sign is subjective. Moreover, he further explained that "no individual sign stands on its own." (Tr. 143.) In this case, petitioner's first neurological exam showed intact (as opposed to brisk) deep tendon reflexes and no ankle clonus, findings that are not consistent with a positive Babinski test. (*Id*.) Whereas Dr. Napoli opined that an equivocal Babinski sign is itself an abnormal finding, Dr. Chaudhry reasonably concluded that in the context of this medical record "equivocal" suggests that treating physicians were unsure whether an abnormal finding was actually present. (Tr. 144-45.) This is especially persuasive in light of the fact that petitioner's medical records show inconsistent results from the Babinski test.[16] (Ex. 7, p. 60; Ex. 3, p. 1.)

Upon petitioner's second neurological exam, Dr. Chaudhry stressed that although petitioner continued to decline overall, his lower extremity strength and reflexes were clearly normal and there was no evidence of any abnormal Babinski sign or ankle clonus.[17] (Tr. 149.) Dr. Chaudhry also explained that petitioner was noted to have difficulty initiating urine on only one occasion, which can have many causes and is different from the type of bladder incontinence that is typically associated with spinal issues. (Tr. 180, 234-35.)

Dr. Chaudhry also stressed that it was not until later (in April) that Dr. Winesett specifically expressed any concern regarding a spinal condition and that concern was due solely to sensory findings at the T4 spinal level. At that time, petitioner had normal strength in his legs, normal Babinski sign, and a follow up MRI specifically to screen for myelitis was normal. (Tr. 180-81 (citing Ex. 8, p. 22).) Overall Dr. Chaudhry suggested that *symptoms* (such as imbalance, leg and chest pain or numbness, urinary retention), are not illuminating for spinal cord issues when the *signs* (such as reflex and strength

[15] Spinal cord compression would also be in the differential diagnosis, but Dr. Napoli opined that it was appropriately ruled out. (Tr. 33-34.) Dr. Chaudhry agreed. (Tr. 236.)

[16] Dr. Napoli initially indicated that an equivocal finding on Babinski testing still constitutes an abnormal finding (Tr. 88), but later acknowledged that the medical records reflect that the medical examiner was seeing fluctuating findings (Tr. 89). This is consistent with Dr. Chaudhry's explanation that the Babinski test is a subjective observation and the records reflect uncertainty regarding the presence of the abnormal finding.

[17] On a later exam, petitioner had "three beats" of ankle clonus observed, but again in the context of normal lower extremity strength. Dr. Chaudhry opined that in that context he would only accept "sustained clonus" as an abnormal finding. (Tr. 152.)

tests) are normal. (Tr. 235.) Combined with the lack of radiography, Dr. Chaudhry persuasively indicated that there is "not a shred of evidence" of spinal inflammation. (Tr. 182.)

Also highly significant, none of petitioner's treating physicians agreed with petitioner's proposed theory of radiculomyelitis. Dr. Napoli cited a diagnosis of radiculitis included in petitioner's physical therapy records. (Tr. 51 (citing Ex. 1, p. 2).) On cross examination, however, he acknowledged that radiculomyelitis was never diagnosed by petitioner's treating physicians nor specifically ruled in or out. (Tr. 53-54, 87.) Dr. Napoli also indicated that a spinal tap could be relevant to diagnosis of radiculomyelitis, but acknowledged it was not done in this case. Instead, he noted that he believes it was contemplated. (Tr. 45-46.) However, ascribing any significance to just the fact of that contemplation would be speculative, especially in light of petitioner's complex presentation and broad differential diagnoses.

I have also considered Dr. Morgan's earlier opinion, but this does not change the analysis. Although, unlike Dr. Napoli, Dr. Morgan relies on radiculomyelitis to explain the entirety of petitioner's symptoms, Dr. Morgan likewise acknowledges the lack of any objective clinical testing to support the suspicion of radiculomyelitis, relying, as Dr. Napoli has, on the idea that petitioner's own lesion was situated in a manner that evaded detection. (Ex. 12, pp. 3-4.) He also necessarily relies on the same overall clinical presentation. Moreover, he acknowledges that if petitioner did have radiculomyelitis it would necessarily have been both atypical and subacute. (Ex. 17, p. 2.) In that regard, Dr. Chaudhry's competing opinion remains persuasive for all the reasons discussed above in rebutting the opinion that petitioner's clinical history evidences radiculomyelitis.

Accordingly, based on the opinion of petitioner's treating physicians, the medical literature, and the opinions of the experts in this case, I do not find preponderant evidence that petitioner suffered radiculomyelitis as proposed by petitioner's experts.

b. Brachial Neuritis

Although petitioner likely did not suffer radiculomyelitis, this still leaves the question of whether petitioner's own treating physicians reasonably diagnosed brachial neuritis, a diagnosis that was also endorsed by Dr. Napoli. Based on the opinion of petitioner's treating physicians, the expert opinions, and medical literature, petitioner has established his diagnosis of brachial neuritis by preponderant evidence. With regard to the alleged brachial neuritis diagnosis, the opinion of petitioner's treating physicians provides strong evidentiary support.

In *Capizzano v. Secretary of Health & Human Services*, the Federal Circuit held that the opinions of treating physicians, in particular, the diagnoses they make and the course of treatment they recommend are "quite probative" of whether there was a logical sequence of cause and effect between vaccine and injury. 440 F.3d 1317, 1326 (Fed. Cir. 2006). Thus, treating physician opinions have historically been given

significant weight when determining causation, but are similarly, if not more helpful in the context of diagnosis. *See*, *e.g.*, *D'Angiolini v. Sec'y of Health & Human Servs.*, No. 99-578V, 2014 WL 1678145, at *24 (Fed. Cl. Spec. Mstr. Mar. 27, 2014) (finding a treating physician's opinion regarding diagnosis "worth a great deal" and "almost definitive evidence on that point" while placing less weight on that physician's opinion regarding etiology), *mot. for rev. denied*, 122 Fed. Cl. 86 (2015), *aff'd*, 645 F. Appx. 1002 (Mem.) (Fed. Cir. 2016).

In this case, after much consideration and examination of petitioner's condition, Drs. Ford and Larden, as well as, most notably, neurologist Dr. Winesett, all concluded that petitioner's most likely diagnosis was brachial neuritis. (*See* Ex. 2, p. 9; Ex. 7, pp. 74, 83-84; Ex. 8, p. 17.) Although petitioner's treating physicians did not entertain brachial neuritis as a differential diagnosis upon his initial admission to ACH (Ex. 7, p. 60), respondent's expert, Dr. Chaudhry, acknowledged both that brachial neuritis is a rare condition and also a "diagnosis of exclusion." (Tr. at 207.) Thus, it follows that brachial neuritis appropriately received increased consideration only as the significant list of other differential diagnoses were excluded. Although petitioner had a complicated, and in some instances potentially confounding, clinical presentation that does not seem to fit neatly into any single diagnosis, Dr. Chaudry was not persuasive in suggesting that the treating physicians' diagnosis should be displaced in favor of simply deeming petitioner's condition wholly enigmatic.

Dr. Chaudhry acknowledged that clinically this is a difficult case. Although he felt petitioner's clinical course could be consistent with an ischemic etiology, he was unable to opine in favor of any more compelling diagnoses. (Tr. at 254, 265-66, 278.) Additionally, to the extent Dr. Chaudhry discussed specific findings he felt were inconsistent with brachial neuritis, he also acknowledged that there is no consensus standard for the diagnostic criteria for brachial neuritis. (Tr. at 269-270.) Moreover, the literature filed in this case supports the view that brachial neuritis encompasses a variety of presentations. (*See* Medlink, *supra*, at Ex. 23; Ferrante & Wilbourn, *supra*, at Ex. 27; Van Eijk, Groothuis & Van Alfen, *supra*, at Ex. C.) Consequently, I am concerned that in this context the exacting standard Dr. Chaudhry seems to apply regarding what he considered to be the relevant diagnostic criteria would have the effect of heightening petitioner's burden of proof. Although Dr. Chaudhry explained that certain aspects of petitioner's presentation may be less typical – for example the initial bilateral presentation and lack of muscle wasting – Dr. Napoli demonstrated that these presentations are known in the context of confirmed cases of brachial neuritis. (*See* Medlink, *supra*, at Ex. 23, pp. 1-7.)

Dr. Napoli further supported petitioner's brachial neuritis diagnosis based on petitioner's clinical presentation and the medical literature detailing the diagnosis of brachial neuritis. Dr. Napoli explained that "even if [petitioner's] presentation is inconclusive, the disorder is typically recognized by two important features of pain and muscle weakness" and that "the most commonly affected nerves are the suprascapular nerve and the long thoracic nerve." (Ex. 21, p. 4 (citing Ferrante & Wilbourn, *supra*, at Ex. 27, p. 1; Medlink, *supra*, at Ex. 23).) Petitioner's medical records show that he

presented to ACH urgent care with acute upper extremity weakness and pain so severe that he was treated with morphine. (Ex. 2, p. 10-11.) Dr. Napoli explains that petitioner's distribution of weakness in his upper extremities supports a finding that either one or both of his suprascapular and long thoracic nerves were affected by his condition, lending further support to the diagnosis of brachial neuritis. (Ex. 21, p. 4.) In response to Dr. Chaudhry's concern regarding the lack of any muscle wasting, Dr. Napoli suggested that muscle wasting may not be expected if a patient has a good recovery. (Tr. 17, 113-14.)

Some of Dr. Chaudhry's concern regarding the brachial neuritis diagnosis stems from petitioner's inconsistent symptom presentation in the course of his diagnosis and treatment. Although Dr. Chaudhry posited some other conditions, such as ischemic injury or migraine, that could cause a fluctuating presentation, he indicated that he does not know if any other condition could explain the inconsistencies and would be "only guessing." (Tr. 168.) However, Dr. Chaudhry also suggested based on these conflicting or confounding notations that some of the observations of petitioner's clinical course may be less reliable than others in assessing petitioner's symptoms, raising the question of whether it was petitioner's symptoms, or merely the observations of petitioner's multiple treaters, that were differing. (Tr. at 163-170.) This further underscores the difficulty of reinterpreting the first-hand observations of the treating physicians' in this case with any confidence. In that regard, Dr. Chaudhry suggested that the neurological opinion should be trusted over conflicting observations. (Tr. 164, 169-70.) As noted above, petitioner's neurologist, Dr. Winesett, opined that petitioner suffered brachial neuritis. (Ex. 8, p. 17.)

Additionally, Dr. Chaudhry's own interpretation of the medical record is not persuasive in all instances; most notably, in his reports he disputed that petitioner initially presented to the emergency department with pain. (Ex. H, p. 1.) During the hearing he indicated that petitioner's initial lack of extremity pain accompanying his upper extremity weakness was a significant atypicality that affected his impression. (Tr. 135.) However, the medical records specifically indicate that at the time he awoke with an inability to move his arms, he was also observed by his parents to be "crying in pain." (Ex. 2, p. 2.) This notation is included in a description of petitioner's waking with upper extremity symptoms and includes no mention of headache. (*Id*.) A further notation that does address the headache likewise also confirms that petitioner had bilateral shoulder pain "since waking up." (Ex. 7, p. 59.) Additionally, Dr. Winesett noted that petitioner had had "shooting pains" across his chest during the night of onset and also that upon exam he was tender to touch around the shoulders. (Ex. 2, p. 6.) Moreover, when he was taken to the emergency department he was administered morphine. (*Id*. at 9, 11.) During the hearing, respondent's counsel asked whether petitioner may have instead been administered morphine in the emergency department for his reported headache; however, Dr. Napoli persuasively explained that petitioner had other pain complaints, including his upper extremities, and that morphine would be unusual treatment for a headache. (Tr. 73-74, 118.)

Dr. Chaudhry also relied heavily on petitioner's negative EMG to exclude brachial neuritis. However, notwithstanding Dr. Chaudhry's own testimony that EMG is a near universal means of confirming brachial neuritis (Tr. 207-08), the medical literature he cited suggests that nerve conduction and EMG studies must be approached cautiously in assessing brachial neuritis and that negative EMGs should not be used to exclude a diagnosis. (*See* Van Eijk, Groothuis & Van Alfen, *supra*, at Ex. C, pp. 5, 7 (explaining that "NA is a clinical diagnosis first and foremost; there is no ancillary 'litmus' test that can confirm or refute the diagnosis with a sufficient degree of certainty" and that "[I]n practice, there is a very real chance of obtaining a negative [EMG] result due to sampling error.").) Dr. Chaudhry himself likewise admitted that, while he considers it unlikely, it is possible for brachial neuritis to present with a normal EMG. (Tr. at 207-08.) For his part, Dr. Napoli did not believe the long thoracic nerve, which he felt was implicated by petitioner's presentation, was tested. (Tr. 114.)

Moreover, Dr. Chaudhry's opinion regarding petitioner's EMG is in itself internally inconsistent in its assessment of petitioner's clinical care. During the entitlement hearing I noted that petitioner's EMG report from Dr. Winesett does not detail how the procedure was conducted, nor the specific basis for Dr. Winesett's interpretation of the exam as negative for brachial neuritis. (Tr. at 271-72.) Dr. Chaudhry explained that, despite the risks of sampling error, because Dr. Winesett was noted to have performed the EMG specifically to evaluate petitioner for possible brachial neuritis, Dr. Winesett's clinical judgment would have led him to conduct the test properly to detect brachial neuritis. (Tr. at 273-77.) However, Dr. Chaudhry also explained that interpreting EMG results is subjective. (Tr. 272.) Moreover, Dr. Chaudhry disagrees with Dr. Winesett's overall clinical judgment that petitioner suffered brachial neuritis, a judgment which Dr. Winesett maintained even after interpreting the EMG as negative. (Tr. at 156-57; *see also* Ex. 8, p. 17.) Thus, it is difficult to fully credit Dr. Chaudhry's opinion on this issue because he simultaneously seeks to defer to Dr. Winesett's judgment in the manner of conducting petitioner's EMG (a point the literature suggests may be potentially problematic (Van Eijk, Groothuis & Van Alfen, *supra*, at Ex. C, p. 7[18])), while at the same time dismissing Dr. Winesett's subsequent clinical judgment as to the significance of the negative finding he himself had generated. On this record Dr. Chaudhry has no basis for parsing Dr. Winesett's clinical judgment in administering the EMG test from his clinical judgment in discounting the significance of the results, especially because, as explained above, Dr. Chaudhry's own reliance materials caution against relying on EMG as a diagnostic touchstone.[19]

[18] Dr. Chaudhry's reliance materials specifically caution that "[i]n contrast to NCS, needle EMG is sensitive for detecting signs of denervation or reinnervation, but only when clinically affected muscles are examined. There are about 50 different muscles in the upper extremity, so only a limited number can be examined during a needle study. Many of the muscles involved in NA do not belong to the routine set of muscles that practitioners most commonly explore during their EMG evaluation, thus sample error is very common." (Van Eijk, Groothuis & Van Alfen, *supra*, at Ex. C, p. 7.)

[19] The experts also had competing opinions regarding the best timing for administering an EMG in the hopes of detecting brachial neuritis. (Tr. 43, 52 (Napoli), Tr. 172 (Chaudhry).)

Finally, I note that respondent has stressed that petitioner's initial expert, Dr. Morgan, agreed that brachial neuritis is not an appropriate diagnosis for petitioner. (ECF No. 74, p. 9.) And, indeed, Dr. Morgan did indicate that "I do agree with Dr. Chaudhry that tests for brachial plexopathy including the normal EMG, normal imaging studies and clinical presentation were against brachial plexitis and Parsonage Turner Syndrome." (Ex. 17, p. 2.) Importantly, however, Dr. Morgan's opinion was premised on his alternative explanation that petitioner's clinical presentation could otherwise be explained by radiculomyelitis. (*Id*. at 1-2.) Although Dr. Morgan located petitioner's injury in the spinal cord (Ex. 17, p. 3), he did initially opine that petitioner presented with clinical findings of brachial plexopathy. (Ex. 12, p. 4). Moreover, even in his supplemental report Dr. Morgan continued to explicitly disagree with Dr. Chaudhry's assessment of petitioner's initial complaints of upper extremity pain and weakness. (Ex. 7, p. 1.) He opined that the myelitis he proposed "simulated bilateral brachial plexopathy." (*Id*. at 2.) He further indicated that "I respectfully disagree with Dr. Chaudhry's conclusions of Mr. Patton's bilateral upper extremity and neck weakness, left leg weakness, variable sensory features [as] not clear and of uncertain cause." (Ex. 7, p. 3.) Accordingly, while Dr. Morgan's opinion further demonstrates the difficulty in reaching a definitive diagnosis in this case, it does not serve as significant ratification of Dr. Chaudhry's competing assessment regarding brachial neuritis.

Based on the opinion of petitioner's treating physicians, the medical literature, and the opinions of the experts in this case, I find that petitioner has presented preponderant evidence that he suffered from brachial neuritis. Notably, however, this diagnosis does not capture the entirety of petitioner's clinical presentation as it does not seem to explain his lower extremity symptoms. Nonetheless, petitioner has not provided preponderant evidence otherwise placing those symptoms within the context of any defined and recognized injury.

VI. Analysis of Petitioner's Claim Under *Althen*

As explained above, petitioner's burden is to demonstrate by preponderant evidence, each of the three *Althen* prongs used to determine actual causation (i.e. an acceptable medical theory, a logical sequence of cause and effect, and a proximate temporal relationship). *Althen*, 418 F.3d at 1278. Because I have concluded that there is preponderant evidence that petitioner suffered brachial neuritis but not radiculomyelitis, this analysis will be focused on whether petitioner has demonstrated his brachial neuritis to be vaccine-caused.

a. *Althen* Prong One

Under *Althen* prong one, a petitioner must provide a "sound and reliable" medical theory demonstrating that the vaccine received can cause the type of injury alleged. *Boatmon v. Sec'y of Health & Human Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019). Petitioner's theory need only be "legally probable, not medically or scientifically certain." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 549 (Fed. Cir. 1994). However, the Federal Circuit has clarified that "simply identifying a 'plausible' theory of

causation is insufficient for a petitioner to meet her burden of proof." *LaLonde v. Sec'y of Health & Human Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) (citing *Moberly*, 592 F.3d at 1322). Instead, petitioner "must provide a reputable medical or scientific explanation that pertains specifically to [their] case." *Moberly*, 592 F.3d at 1322. In prior cases, this prong has been described as requiring petitioner to show that the alleged vaccine *can* cause the alleged injury, while prong two requires petitioner to show that the alleged vaccine *did* cause the alleged injury. *See, e.g.*, *Doe 11 v. Sec'y of Health & Human Servs.*, 83 Fed. Cl. 157, 172–73 (2008), *aff'd* 601 F.3d 1349 (Fed. Cir. 2010); *Nussman v. Sec'y of Health & Human Servs.*, 83 Fed. Cl. 111, 117 (2008); *Banks v. Sec'y of Health & Human Servs.*, 2007 WL 2296047, at *24 (Fed. Cl. Spec. Mstr. July 20, 2007); *Zeller v. Sec'y of Health & Human Servs.*, 2008 WL 3845155, at *25 (Fed. Cl. Spec. Mstr. July 30, 2008). Thus, to meet prong one of *Althen*, petitioner must show by preponderant evidence that the flu vaccine is capable of causing brachial neuritis.

Tetanus containing vaccines are presumed to be capable of causing brachial neuritis in this program. *See* 42 C.F.R. § 100. However, as explained above, petitioner's burden in this case is to show that the specific vaccine he received – the influenza vaccine – can also cause brachial neuritis. In that regard, Dr. Napoli has acknowledged that he has provided no evidence -- no study apart from anecdotal case reports (Exs. 28-31)[20] – directly indicating that the flu vaccine can cause brachial neuritis. (Tr. 93, 97.) Instead, Dr. Napoli relies on the notion that brachial neuritis is theorized to be an autoimmune condition associated with multiple triggers, both known and unknown, including infections. (Tr. 16.) Thus, Dr. Napoli opined that the flu vaccine can cause brachial neuritis via molecular mimicry.[21] (ECF No. 77, pp. 35-39; Tr. 38-39.)

However, special masters have repeatedly held in varying contexts that it is insufficient for petitioners to merely invoke molecular mimicry without more. *See*, *e.g.*, *W.C. v. Sec'y Health & Human Servs.*, 704 F.3d 1352, 1361 (Fed. Cir. 2016) (finding that the "special master reasonably considered the lack of evidence connecting the cross-reactivity observed by Wucherpfenning to the facts of petitioner's case to weigh 'against finding that Dr. Tornatore's opinion is perusaisve.'"); *Issac v. Sec'y of Health & Human Servs.*, No. 08-601V, 2012 WL 3609993, at *3-5, *21-22 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for rev. denied,* 108 Fed. Cl. 743 (2013), *aff'd* 540 Fed. Appx. 999 (Mem.) (Fed. Cir. 2013); *Tullio v. Sec'y of Health & Human Servs.*, No. 15-51V, 2019

[20] "[C]ase reports 'do not purport to establish causation definitively, and this deficiency does indeed reduce their evidentiary value'…. [but] 'the fact that case reports can by their nature only present indicia of causation does not deprive them of all evidentiary weight.'" *See Paluck v. Sec'y of Health & Human Servs.,* 104 Fed. Cl. 457, 475 (2012) (quoting *Campbell v. Sec'y of Health & Human Servs.*, 97 Fed. Cl. 650, 668 (2011), *aff'd* 786 F.3d 1373 (Fed. Cir. 2015)).

[21] Although Dr. Napoli explained that certain vaccines are believed to contain a protein that can mimic nerve myelin, he stressed that it is very difficult to prove the presence of cross-reactivity and further that molecular mimicry is not the only possible explanation for autoimmunity. (Tr. 58-59.) Critically, however, Dr. Napoli did not actually describe any of the other causal theories to which he alluded.

WL 7580149, at *12-14 (Fed. Cl. Spec. Mstr. Dec. 19, 2019), *mot. for rev. denied,* 149 Fed. Cl. 448 (2020). Moreover, petitioners have previously tried and failed to specifically connect brachial neuritis to the flu vaccine. *E.g.*, *Grow v. Sec'y of Health & Human Servs.*, No. 16-13V, 2020 WL 7366332, at *24 (Fed. Cl. Spec. Mstr. Nov. 24, 2020) (finding that prong one was not met because "Dr. Gershwin only generally describes how inflammation may factor into autoimmune responses [and] does not explain how an inflammatory process is initiated by the flu vaccine specifically or how such a response leads to [brachial neuritis].").

In this case, Dr. Napoli failed to provide any adequate explanation indicating how the flu vaccine can induce brachial neuritis via molecular mimicry. Instead, Dr. Napoli opined that evidence linking the flu vaccine to GBS is also broadly applicable in the context of brachial neuritis. (Ex. 21, pp. 5-6.) To substantiate his theory, Dr. Napoli sought to rely on evidence relating the flu vaccine to the Acute Inflammatory Demyelinating Polyneuropathy ("AIDP") form of GBS, which involves an autoimmune process believed to involve a cross-reaction affecting myelin tissue, resulting in demyelinating nerve damage. (Haber et al., *supra*, at Ex. 26, p. 3.) Given the relationship between flu vaccines and GBS, Dr. Napoli characterized it as "not out of the realm of possibility to think that the flu vaccine could cause other peripheral nerve disorders." (Tr. 94.) And, to be sure, the literature filed in this case does suggest that the fact that brachial neuritis shares some triggers in common with inflammatory demyelinating polyradiculopathies contributes to the general theory that brachial neuritis has an immune basis. (*E.g.* Medlink, *supra*, at Ex. 23, p. 16.)

Dr. Chaudhry persuasively explained, however, that cases of brachial neuritis overwhelmingly result in axonal damage and not demyelination of the type seen in the AIDP form of GBS. (Tr. at 131-33, 279-80; *see also* Van Eijk, Groothuis & Van Alfen, *supra*, at Ex. C, p. 7.) Dr. Chaudhry analogized nerves to electrical wires, with myelin forming an insulating sheath that surrounds the axons which make up the body of the nerve. (Tr. 131-32.) Although Dr. Napoli cited literature identifying rare occurrences (less than 1% of cases) of a brachial neuritis-like presentation consisting of demyelinating injury[22], that same literature emphasizes that brachial neuritis is generally considered to result in axonal degeneration. (Medlink, *supra*, at Ex. 23, p. 2. ("available evidence suggests that an autoimmune pathogenesis, likely related to a genetic susceptibility most commonly generates an axon loss lesion (focal or multifocal) that involves predominantly motor axons").)

Significantly then, Van Eijk et al., filed by respondent, discusses the autoimmune basis for brachial neuritis as being evidenced specifically by studies of nerve biopsies finding that the nerve axons represented the target of the autoimmune attack in brachial neuritis. (Van Eijk, Groothuis & Van Alfen, *supra*, at Ex C, p. 5.) Both experts agreed that axonal damage represents a more severe form of nerve injury as compared to

[22] To the extent that both the testifying experts and cited literature demonstrate that brachial neuritis lacks definitive consensus criteria, it is difficult to conclude whether this rare occurrence of demyelinating injuries referenced by Ferrante and Graus actually represent instances of brachial neuritis or simply a different condition that closely resembles brachial neuritis. That issue is not discussed by this article.

demyelination (Tr. 84-85 (Napoli), 132 (Chaudhry)); however, Dr. Chaudhry also explained that the two types of injury represent different pathogeneses, with myelin sheath having susceptibility to different antigens. (Tr. 133.) Accordingly, Dr. Napoli's use of evidence linking AIDP to the flu vaccine, which is largely epidemiological rather than mechanistic, is unpersuasive without more. These conditions result in distinct forms of nerve damage and Dr. Napoli has provided no evidence suggesting that a vaccine causing one type of nerve damage via molecular mimicry should be assumed capable of causing the other.

Dr. Napoli does suggest that Acute Motor Axonal Neuropathy (AMAN), a form of GBS affecting the axons, has been hypothesized to result from molecular mimicry in the case of *C. Jejuni* infections. However, he has not presented any evidence, apart from his *ipse dixit*, showing any connection between AMAN and the flu vaccine.[23] *Accord Isaac v. Sec'y of Health & Human Servs.*, No. 08-601V, 2012 WL 3609993, at *22 (Fed. Cl. Spec. Mstr. July 30, 2012) (noting that "[e]vidence that C. jenjuni causes GBS by molecular mimicry does not constitute preponderant evidence that molecular mimicry is a possible explanation for vaccine causation.") Thus, the flu vaccine has not been identified as a trigger for autoimmune axonal loss in either GBS or brachial neuritis. Conversely, Dr. Chaudhry also stressed that *C. Jejuni* has not been shown to result in brachial neuritis, further limiting the relevance of petitioner's reference to AMAN and its potential parallel to brachial neuritis. (Tr.186.)

Respondent has also stressed that the Institute of Medicine ("IOM")[24] examined the relevant medical literature and found no epidemiology evaluating the risk of brachial

[23] All forms of GBS, including AMAN, enjoy a causal presumption under the vaccine injury table when following influenza vaccination. Importantly, however, petitioner does not allege any form of GBS. Therefore, the causal presumption is inoperable in this case. Moreover, GBS was added to the vaccine injury table on the basis of studies showing a causal association between the 1976 swine flu vaccine and demyelinating GBS. National Vaccine Injury Compensation Program: Revisions to the Vaccine Injury Table, 82 Fed. Reg. 6294-01 (Jan. 19, 2017) (to be codified at 42 C.F.R. 100). The government explained that seasonal flu vaccines "include multiple antigens that change from year-to-year, and enhanced surveillance . . . may not occur with each virus strain change." *Id.* Further, "[i]n light of this information . . . the ACCV recommended that the Secretary add GBS consistent with one of its Guiding Principles: That where there is credible evidence to both support and reject a change to the Table, the change should, whenever possible, be made to the benefit of petitioners." *Id*. Thus, especially in light of such policy considerations, the fact that axonal forms of GBS enjoy a causal presumption does not in itself suggest meaningful evidence that the flu vaccine can cause axonal nerve damage, especially in the context of a different condition. Literature filed by petitioner explains that, although there are hypotheses for the etiology of GBS that could potentially apply to both demyelinating and axonal damage, separate explanations have also been advanced that would be unique to AMAN and the etiology remains unresolved. (Haber et al., *supra*, Ex. 26, p. 5.)

[24] The Institute of Medicine is the medical arm of the National Academy of Sciences. The National Academy of Sciences ("NAS") was created by Congress in 1863 to be an advisor to the federal government on scientific and technical matters (*see* An Act to Incorporate the National Academy of Sciences, ch. 111, 12 Stat. 806 (1863)), and the Institute of Medicine is an offshoot of the NAS established in 1970 to provide advice concerning medical issues. When it enacted the Vaccine Act in 1986, Congress directed that the IOM conduct studies concerning potential causal relationships between vaccines and illnesses. *See* § 300aa–1 note.

neuritis following the flu vaccine. (*Adverse Effects of Vaccine: Evidence & Causation*, INSTITUTE OF MEDICINE (Ex. K, p. 3).) Additionally, the IOM examined two studies constituting mechanistic evidence regarding the etiology of brachial neuritis, but found that neither provided evidence linking the mechanisms to the flu vaccine. (*Id.*) IOM reports are routinely relied upon by special masters as trustworthy evidence. *See, e.g. Crutchfield v. Sec'y Health & Human Servs.*, 125 Fed. Cl. 251 (2014) (noting that "it was appropriate for the special master to consider the medical literature presented, including the IOM report" and that "the court often has relied on the findings of the Institute of Medicine.")[25] Importantly, however, special masters are not bound by the IOM's conclusions and it has been observed that the IOM employs a standard for finding causation that is higher than what is required by petitioner's burden of proof. *E.g. Raymo v. Sec'y of Health & Human Servs.*, No. 11-654V, 2014 WL 1092274, at *21, n.39 (Fed. Cl. Spec. Mstr. Feb. 24, 2014). In this case, the IOM's conclusion is informative but not dispositive, serving on this record to underscore Dr. Napoli's failure to come forward with any significant evidence implicating the flu vaccine in the disease process(es) at work in the development of brachial neuritis specifically or autoimmune axonal damage generally.

In light of the above, I find that petitioner has failed to present preponderant evidence showing that the influenza vaccine can cause brachial neuritis.

b. *Althen* Prong Two

The second *Althen* prong requires proof of a logical sequence of cause and effect usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375-77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). However, medical records and/or statements of a treating physician do not *per se* bind the special

[25] *See also, Isaac v. Sec'y Health & Human Servs.,* 108 Fed. Cl. 743, 755 (2013), *aff'd,* 540 Fed. Appx. 999 (Mem.) (Fed. Cir. 2013) (affirming the special master's reliance on findings of the IOM); *Porter v. Sec'y Health & Human Servs.,* 663 F.3d 1242, 1252 (Fed.Cir.2011) (noting the special master's comment that "IOM reports are favored, although not dispositive, in the Vaccine Act Program," then affirming the special master's decision); *Cedillo v. Sec'y Health & Human Servs.,* No. 98–916V, 2010 WL 331968, at *94 (Fed. Cl. Spec .Mstr. Feb. 12, 2009), *mot. for rev. denied,* 89 Fed. Cl. 158 (2009) (affirming special master's reliance on conclusions of IOM), *aff'd,* 617 F.3d 1328 (Fed.Cir.2010); *Rodriguez v. Sec'y Health & Human Servs.,* 67 Fed. Cl. 409, 410 (2005) (relying on IOM report regarding vaccine causation of an injury); *Althen v. Sec'y Health & Human Servs.,* No. 00–170V, 2003 WL 21439669, at *11, n.28 (Fed. Cl. Spec. Mstr. June 3, 2003) ("Due to the IOM's statutory charge, the scope of its review, and the cross-section of experts making up the committee reviewing the adverse events associated with vaccines, the court considers their determinations authoritative and subject to great deference."), *rev'd on other grounds,* 58 Fed. Cl. 270, 272–74 (2003) (citing IOM reports frequently in support of various scientific propositions), *aff'd,* 418 F.3d 1274 (Fed. Cir. 2005); *Terran v. Sec'y Health & Human Servs.,* 41 Fed. Cl. 330, 337 (1998) (affirming special master's reliance on conclusions of IOM), *aff'd,* 195 F.3d 1302 (Fed. Cir.1999), *cert. denied,* 531 U.S. 812 (2000); *Cucuras v. Sec'y Health & Human Servs.,* 993 F.2d 1525, 1529 (Fed. Cir. 1993) (noting that the special master had placed "a great deal of weight" on an IOM report in reaching a decision, then affirming the special master's decision); *Stroud v. Sec'y Health & Human Servs.,* 113 F.3d 1258 (Fed. Cir. 1997) (unpublished)(special master may rely upon an IOM report that neither party filed as evidence); *Ultimo v. Sec'y Health & Human Servs.,* 28 Fed. Cl. 148, 152 (1993) (proper for a special master to rely on IOM report).

master to adopt their conclusions, even if they must be considered and carefully evaluated. *See* Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment test result, report, or summary shall not be binding on the special master or court."); *Snyder v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted."). As described in the preceding section, this prong is at times distinguished from the first by requiring the petitioner to show that the vaccine petitioner received *did* in-fact cause their injury, instead of merely showing that it *can* cause their injury.

The parties agree that brachial neuritis can occur within days of an antecedent trigger. (Medlink, *supra*, at Ex. 23, pp. 2-7; Van Eijk, Groothuis & Van Alfen, *supra*, Ex. C, pp. 1-2.) Here, petitioner presented with symptoms of brachial neuritis within days of his flu vaccination. This timing of onset fits squarely within the accepted timeline for brachial neuritis to develop following an antecedent event. Standing alone, however, a temporal association is not sufficient to satisfy *Althen* prong two. *Veryzer v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 344, 356 (2011) (explaining that a "temporal relationship alone will not demonstrate the requisite causal link and that petitioner must posit a medical theory causally connecting the vaccine and injury.").

Petitioner's initial presentation seemed to confuse his treating physicians at ACH who initially listed 13 differential diagnoses including central nervous system mass, carbon monoxide poisoning, dehydration, electrolyte abnormality, unspecified headache, migraine, tension headache, intracranial hemorrhage, meningitis, post-concussion syndrome, shunt malfunction, and stroke. (Ex. 7, p. 60.) However, as petitioner was further examined, his treating physicians and physical therapists settled on a diagnosis of possible or probable brachial neuritis (referenced as Parsonage-Turner Syndrome) that they related to his flu vaccination. (Ex. 7, pp. 17, 47, 53, 85.) Importantly, however, petitioner's medical records show no evidence of any diagnostic labs such as lumbar puncture which may have helped support a causal, and not merely temporal, link between the vaccine and his injury.

Thus, although petitioner's treating physicians did ultimately settle on the diagnosis of brachial neuritis, their further opinion that the condition was caused by petitioner's flu vaccination relies exclusively on the temporal relationship and the lack of any known alternative triggers. This conclusion carries little weight in light of my finding that petitioner has not satisfied *Althen* prong one relative to brachial neuritis. *See e.g. Grow*, 2020 WL 7366332 at * 27 ("Therefore, the treating physicians' notation temporally associating the flu vaccine with PTS are entitled to little weight in Petitioner's case, because the association is based solely on a temporal relationship reported by Petitioner."); *D'Angiolini v. Sec'y of Health & Human Servs.,* No. 99-578V, 2014 WL 1678145 (Fed. Cl. Spec. Mstr. Mar. 27, 2014) (noting that "there is a difference between a doctor's opinion regarding diagnosis and a doctor's opinion regarding etiology and quoting *Tamraz v. Lincoln Electric Co*, 620 F. 3d 664, 674 (6th Cir. 2010) for the proposition that physicians "may testify to both [diagnosis and etiology] but the reliability

of one does not guarantee the reliability of the other"), *mot. for rev. denied,* 122 Fed. Cl. 86 (2015), *aff'd*, 645 F. Appx 1002 (Mem.) (Fed. Cir. 2016).

Although Dr. Napoli suspected petitioner's brachial neuritis symptoms were caused by demyelination based on the speed of his recovery, he acknowledged that there is no evidence on this record distinguishing whether petitioner's symptoms were caused by demyelination or axonal damage. (Tr. 107.) Dr. Napoli's opinion that radiculomyelitis and brachial neuritis may together provide an explanation for petitioner's symptoms is also based on the assumption that the two conditions petitioner purportedly suffered both represented demyelinating conditions. (Tr. 107.) However, this point is refuted by Dr. Chaudhry and has not otherwise been substantiated by any of petitioner's submissions. Dr. Chaudry persuasively opined that cases of brachial neuritis overwhelmingly result in axonal damage and not demyelination. (Tr. at 131-33, 279-80; *see also* Van Eijk, Groothuis & Van Alfen, *supra*, at Ex. C, p. 7.) And, in any event, Dr. Napoli's and Dr. Morgan's suspicion of radiculomyelitis was not considered by any of petitioner's treating physicians and, for the reasons discussed in Section V.a., above, is not supported by preponderant evidence. Petitioner has failed to show by preponderant evidence a logical sequence of cause and effect whereby the flu vaccine would have induced an autoimmune reaction that caused his axonal-loss lesion resulting in brachial neuritis.

Additionally, petitioner's own presentation is inconsistent with the presentation of AIDP, which formed the basis for petitioner's presentation linking the flu vaccine to peripheral demyelinating injury. AIDP patients present with symptoms of ascending weakness and sensory loss from the lower extremities to the upper extremities in the early stages (Krishnan & Greenberg, *supra*, at Ex. F, p. 21), while brachial neuritis patients present with new-onset pain in the shoulder or upper arm developing into paresis typically involving the long thoracic, suprascapular, and anterior interosseous nerves. (Van Eijk, Groothuis & Van Alfen, *supra*, at Ex. C, p. 1.) Here, petitioner presented with symptoms diagnosed as brachial neuritis and not symptoms of AIDP. Although petitioner had some lower extremity symptoms, it was his upper extremity symptoms that presented most prominently upon onset. None of the experts, or the treating physicians ultimately, opined that petitioner suffered AIDP.

Although petitioner's condition does remain somewhat enigmatic, he has not articulated any basis for concluding that his flu vaccine could be responsible for a logical sequence of cause and effect leading to his constellation of symptoms. Accordingly, I find that petitioner has not met his burden under *Althen* prong two.

c. *Althen* Prong Three

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. This standard has been defined as requiring a "medically-acceptable temporal relationship." *Id.* Here the parties agree that onset of petitioner's symptoms occurred within six days of his vaccination and therefore falls within the "medically appropriate timeframe for the

onset of an immunological injury." (*See* ECF No. 74, p. 18; *see also* ECF No. 77 pp. 39-41.) Consequently, I find that petitioner has carried his burden under *Althen* prong three. However, petitioner's failure to meet prongs one and two means that petitioner cannot be compensated. *Hibbard v. Sec'y of Health & Human Servs.*, 698 F.3d 1355, 1364-65 (Fed. Cir. 2012) (holding the special master did not err in resolving the case pursuant to prong two when respondent conceded that petitioner met prong three).

VII. Conclusion

For all the reasons discussed above, I find that petitioner has failed to carry his burden of showing by preponderant evidence that his flu vaccination was the actual cause of his injury. Specifically, I find that petitioner has failed to demonstrate that he suffered any identifiable injury or condition other than the brachial neuritis diagnosed by his treating physicians. I further find that he has failed both to present a medical theory causally connecting the flu vaccine to brachial neuritis and to present preponderant evidence establishing a logical sequence of cause and effect showing that the flu vaccine did cause his condition. Therefore, this case is dismissed.[26]

IT IS SO ORDERED.

s/Daniel T. Horner
Daniel T. Horner
Special Master

[26] In the absence of a timely-filed motion for review of this Decision, the Clerk of the Court shall enter judgment accordingly.