In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 14-1128V
Filed: November 19, 2021
PUBLISHED

BARBARA GOFORTH, Petitioner, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	Special Master Horner Influenza (flu) Vaccination; Sensory Variant Guillain Barre Syndrome (GBS)

Randall G. Knutson, Knutson & Casey Law Firm, Mankato, MN, for petitioner.
Ronalda Elnetta Kosh, U.S. Department of Justice, Washington, DC, for respondent.

DECISION[1]

On November 19, 2014, petitioner initially filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10-34 (2012),[2] alleging that she suffered adverse effects, including a demyelinating disease of the central nervous system, following her receipt of a flu vaccination on September 29, 2011. (ECF No. 1.) Subsequently, on June 27, 2016, while petitioner was pursuing her claim *pro se*, she amended her petition, alleging that her receipt of a flu vaccination on November 2, 2014, significantly aggravated what she now alleged to be Guillain-Barre Syndrome ("GBS"). (ECF No. 38.) For the reasons set forth below, I conclude that petitioner is not entitled to compensation.

[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] Within this decision, all citations to §300aa will be the relevant sections of the Vaccine Act at 42 U.S.C. §300aa-10-34.

I. Procedural History

As noted above, petitioner filed her claim on November 19, 2014, alleging that the flu vaccine she received on September 29, 2011, caused her to suffer "a plethora of unprecedented symptoms and illnesses, including Demyelinating Disease of the Central Nervous System, Paresthesia, Mononeuritis, Dysphagia, and Speech disturbance." (ECF No. 1.) This case was originally assigned to Special Master Millman. (ECF No. 4.) Onset was originally pled as occurring on November 21, 2011, suggesting the claim was filed about two days prior to the expiration of the statute of limitations. (ECF No. 1, p. 1.) Petitioner filed medical records and an affidavit over the following two months. (ECF Nos. 9-10; Exs. 1-6.)

On January 23, 2015, an initial status conference was held. (ECF No. 11.) The parties discussed the fact that, due to the impending statute of limitation, petitioner's counsel (prior counsel – Andrew Downing, Esq.) filed this claim without the benefit of complete investigation of the medical records. Based on certain records placing onset of symptoms prior to the 2011 vaccination at issue, petitioner's counsel suggested petitioner may pursue a significant aggravation claim. (*Id.*) Thereafter, petitioner filed additional medical records and a Statement of Completion. (ECF Nos. 13, 18, 20, 22, 26; Exs. 7-27.) However, on April 7, 2016, Mr. Downing filed a motion to withdraw as attorney, which was granted on the same day. (ECF Nos. 30-31.) Thereafter, petitioner pursued her claim *pro se* for about one year.

While acting *pro se*, petitioner amended her petition on June 27, 2016. (ECF No. 38.) Petitioner now alleged that her receipt of a second flu vaccination on November 2, 2014, significantly aggravated what was at that time a pre-existing condition, which she continued to allege was initially caused by her prior 2011 vaccination. However, she now indicated onset of that condition was in October 2011 rather than late November 2011 as previously alleged. (*Id.*) Petitioner indicated that she understood that her amended allegation regarding onset rendered her claim relative to her 2011 vaccination untimely. (*Id.*) Petitioner also indicated that subsequent to filing her initial petition the condition at issue had been diagnosed as sensory variant GBS by her treating neurologist, Mohammed Khoshnoodi, M.D. (*Id.*) Shortly after petitioner filed her amended petition, she filed a letter by Dr. Khoshnoodi confirming his causal opinion.[3] (ECF No. 41.) Special Master Millman held a follow up status conference and issued an order instructing petitioner to file a letter from Dr. Khooshnoodi answering various questions she raised. (ECF No. 42.) Petitioner filed a letter responsive to that order on December 19, 2016. (ECF No. 49.) Her current counsel, Randall Knutson, substituted as counsel on March 7, 2017. (ECF No. 53.)

Respondent filed an expert report from neurologist Vinay Chaudhry, M.D. on September 29, 2017, opining that petitioner's symptom presentation was fluctuating,

[3] Accompanying that letter were over one hundred pages of medical records from various providers. None of the documents in this filing were marked with any exhibit designation. However, these documents were later designated as Exhibit 37 by petitioner and were referenced during the hearing as Exhibit 37.

could not be linked to either of her vaccinations, and was not properly diagnosed as GBS. (ECF No. 61; Ex. A.) Additionally, respondent filed his Rule 4(c) report, recommending against compensation and moving to dismiss this case. (ECF No. 63.) Petitioner cross-moved for permission to amend the petition to add a claim for a Table Injury of GBS in light of the addition of GBS to the Vaccine Injury Table on March 21, 2017. *See* National Vaccine Injury Compensation Program: Revisions to the Vaccine Injury Table, Final Rule, 82 Fed. Reg. 6294, Jan. 19, 2017; National Vaccine Injury Compensation Program: Revisions to the Vaccine Injury Table, Delay of Effective Date, 82 Fed. Reg. 11321, Feb. 22, 2017. Special Master Millman denied respondent's motion to dismiss the case and ordered petitioner to file an expert report to support her claim, explaining that while petitioner's claim vis-à-vis her 2011 vaccination was untimely, she had appropriately pled a significant aggravation claim based in causation-in-fact relative to her later 2014 vaccination. (ECF No. 65.) She also indicated that petitioner could not amend her current petition to include a claim for a later-created Table Injury, but could dismiss her petition and file a new petition asserting such a claim. (*Id.*) This was never done.

Petitioner subsequently filed an expert report from Dr. Beatrice Engstrand on August 9, 2018. (ECF No. 70; Ex. 29.) In response, respondent filed a supplemental report from Dr. Chaudhry. (ECF No. 74; Ex. C.)

This case was reassigned to my docket on June 7, 2019. (ECF No. 78.) A two-day entitlement hearing was held remotely on February 17 and 18, 2020, via Webex. (*See* ECF Nos.120-21, Transcript of Proceedings ("Tr"), filed 3/22/2021.) Simultaneous post-hearing briefs were filed on April 30, 2021. Accordingly, this case is ripe for resolution.

II. Factual History

a. As Reflected by the Medical Records

Prior to the vaccinations in this case, petitioner had a history of asthma and bronchitis, previously treated with prednisone, singulair, and albuterol, in addition to later-reported symptoms discussed below. (Ex. 2, pp. 8-9; Ex. 9, p. 28; Ex. 14, pp. 4-5; Ex. 16, p. 27.) Petitioner also had a history of chest pain, syncope, dyspnea, migraines, and severe reflux issues. (Ex. 13, p. 9; Ex. 15, pp. 6, 9, 15; Ex. 17, pp. 65, 70.) Petitioner had previously declined to receive the Hepatitis-B vaccine. (Ex. 9, p. 20.) In 2001, petitioner suffered a fall and sustained an injury to her right wrist. (Ex. 9, p. 53.) Petitioner received the flu vaccination on September 29, 2011 in her right deltoid. (Ex. 10.)

The first medical record post-dating petitioner's September 2011 flu vaccination is a rheumatology referral by Dr. Kwabena A. Donker dated November 3, 2011. (Ex. 2, p. 3.) The referral indicated petitioner's complaint was "pain at multiple sites – unknown cause." (*Id.*) Petitioner was not able to see the rheumatologist (Dr. Wilson) until March 15, 2012. (Ex. 27, p. 9.) In the interim, petitioner presented for care for acute sinusitis

on December 3, 2011, and at that time noted onset to have been three days prior. She denied any fever or cough. (Ex. 18, p. 12.) However, on February 21, 2012, petitioner saw Dr. Donkor for an ongoing cough, laryngitis, and swollen glands. (Ex. 2, p. 6.) Upon review of systems, petitioner indicated having a low-grade fever, mild dyspnea, joint pains, thick coughs, and fatigue. (*Id.*) Dr. Donkor ordered a chest x-ray and assessed petitioner with asthmatic bronchitis with possible upper airway cough syndrome. (*Id*. at 7.) Petitioner saw Dr. Donkor again two days later on February 23, 2012 with a chief complaint "feels really bad, fever." (Ex. 2, p. 4.) Petitioner reported intermittent muscle and joint pains, intermittent skin lesions, and aching in her lower abdomen. (*Id.*) Physical examination revealed tenderness at her shoulder joints and rash in her upper chest and arms. (*Id*. at 5.) Petitioner's studies were negative, and she was assessed with asthmatic bronchitis, arthralgias, and unexplained fever and skin rash. (*Id.*) Dr. Donkor indicated that petitioner may need a rheumatology evaluation.[4] (*Id.*)

On March 14, 2012, petitioner first visited Dr. Jeffrey Wilson after a referral for "pain at multiple sites-unknown cause." (Ex. 2, p. 3.) Dr. Wilson indicated that petitioner had symptoms of joint pain, redness, and swelling, and associated symptoms including fever, rash, fatigue, and extremity weakness. (Ex. 27, p. 9.) Petitioner's neurologic evaluation revealed that her cranial nerves were intact, her reflexes were symmetrical, her gait was normal, and her sensation was normal. Additionally, petitioner had normal muscle strength. (*Id*. at 11.) However, there was decreased grip strength in her right hand when in fist formation. (*Id.*) Dr. Wilson diagnosed petitioner with polyarthralgia and carpal tunnel syndrome with differential diagnosis in varying types of disorders including an inflammatory disorder like rheumatoid arthritis, a metabolic disorder like vitamin D deficiency, a systemic disorder like myeloma, an infectious condition like Lyme disease, or fibromyalgia. (*Id*. at 12.) Petitioner saw Dr. Wilson again on March 29, 2012. (Ex. 27, p. 6.) At this visit, petitioner reported that she had passed out twice about six months ago that this had been deemed to be associated with her heart murmur condition. (*Id.*) Dr. Wilson kept petitioner's diagnosis as polyarthralgia and noted that he was concerned that petitioner had an evolving connective tissue disease. (*Id*. at 8.)

On April 13, 2012, petitioner returned to Centra Health Cardiovascular Group[5] to manage of her chronic cardiovascular conditions including dyspnea, syncope, and heart murmur. (Ex. 13, pp. 3-4.) Petitioner reported that she was diagnosed with lupus and started on prednisone. (*Id*. at 4.) Physical examination was normal. (*Id*. at 5.) Additionally, petitioner's transthoracic echocardiography was overall normal. (*Id*. at 7-8.)

[4] Petitioner testified that the February 23, 2012 follow up was actually just to receive sputum test results. (Tr. 350-55.)

[5] Petitioner had an initial consult at Centra Health Cardiovascular Group on August 23, 2011. (Ex. 13, p. 9.)

Petitioner was admitted to Centra Health Southside Community Hospital on May 15, 2012 for acute cholecystitis. (Ex. 7, p. 39.) She had four days of right upper abdominal pain and at the emergency room, petitioner received a cholecystectomy. She was discharged home on May 16, 2012. (*Id*.)

Petitioner visited Dr. Wilson on May 24, 2012 for a follow up. (Ex. 3, p. 4.) Dr. Wilson indicated, as part of petitioner's history of present illness, that petitioner had arthritis and joint pain and had been treated with prednisone. (*Id*.) Petitioner also reported other symptoms including fever, rash, fatigue, and extreme weakness in hands. (*Id*.) At this visit, petitioner reported improvement following taking prednisone; however following gallbladder removal surgery, she continued having pain in her right side across her abdomen. (*Id*.) Petitioner indicated feeling numbness, tingling, headaches, and paresthesias. (*Id*. at 5.) Upon physical examination, Dr. Wilson indicated normal muscle strength, normal gait, and normal sensation. (*Id*.) Dr. Wilson assessed petitioner with inflammatory polyarthropathy and was concerned about an evolving connective tissue disorder.

On June 6, 2012, petitioner visited the Orthopaedic Center of Central Virginia and was seen by Nurse Practitioner Andrea Yesalis with a chief complaint of not feeling well since 2006 and numerous colds and a diagnosis of multiple joint pain. (Ex. 6, p. 5; Ex. 11, p. 14.) Petitioner also indicated that she had pain on the bottom of her right foot. (Ex. 6, p. 5.) On physical examination, it was noted that petitioner had full range of motion in her upper extremities, but there was exquisite point tenderness over both bursa area and bottom right foot. (*Id*. at 7.) Petitioner was assessed with bilateral trochanteric bursitis and possible Morton's neuroma in her right foot. Chronic use of steroids and NSAIDs was noted. (*Id*. at 6.)

Petitioner returned to the Orthopaedic Center on June 20, 2012, for burning pain and follow up regarding her right foot pain and was seen again by N.P. Yesalis. (Ex. 6, p. 13, 18; Ex. 11, p. 20.) Petitioner's bone density test was normal. (Ex. 6, p. 11.) It was noted that petitioner did not have an autoimmune inflammatory joint disease nor lumbar spine radiculopathy, but petitioner was experiencing continuous burning pain in her right foot. Petitioner needed a referral regarding the burning sensation in her right foot. (*Id*. at 14-15.) She was ordered to taper off prednisone. (*Id*. at 15.)

Petitioner first saw neurologist Dr. W. Miles Wallace on July 3, 2012, for an evaluation of pain in her lower extremity. (Ex. 4, p. 5.) Petitioner reported her main symptoms to include burning, throbbing stabbing pins and needles pain and cold feeling in her legs. (*Id*. at 9.) Based on petitioner's prior records, Dr. Wallace indicated that petitioner since last fall "developed [fluctuating] pain multiple joints and tingling, aching, burning, feeling in her right leg below the knee," and "intermittent tingling in her left foot but not as prominently as the right." (*Id*. at 5.) Dr. Wallace noted that petitioner reported no neurologic symptoms in the past. On examination, petitioner's strength, tone, and deep tendon reflexes were normal and symmetric. (*Id*.) Petitioner's nerve conduction and EMG studies were overall normal, except Dr. Wallace was not able to get a right sural or medial plantar sensory response. (*Id*.) Dr. Wallace diagnosed

petitioner with a predominantly sensory peripheral neuropathy and posited that petitioner has some underlying inflammatory illness. (*Id*.)

Following evaluation from Dr. Wallace, petitioner returned to the Orthopaedic Center regarding her condition now diagnosed as peripheral neuropathy on July 23, 2012. (Ex. 6, p. 23; Ex. 11, p. 26.) Petitioner reported continuing symptoms of episodes of stabbing pain in her feet and arms. (Ex. 6, p. 23.) N.P. Yesalis noted that petitioner responded to Neurontin. (*Id*. at 24.)

Petitioner's brain MRI taken on August 7, 2012, revealed vague areas of increased signal that are nonspecific but compatible with multiple sclerosis. (Ex. 16, pp. 31-32; Ex. 7, p. 29.) On August 14, 2012, petitioner saw Dr. Wallace again for a neurology evaluation. (Ex. 4, p. 3.) Dr. Wallace indicated that he first assessed petitioner with small fiber neuropathy based on her sensory findings when he saw her on July 3, 2012. However, at the August 14, 2012 visit, petitioner reported feeling pins and needles and burning in her lower legs since 2006 and the symptoms continued throughout into 2010. In 2010, petitioner's symptoms were more severe, and she began developing rapid eye movements and trouble swallowing. (*Id*.) Dr. Wallace reported that, upon examination, petitioner's strength, tone, and deep tendon reflexes were normal and symmetric in her upper and lower extremities. (*Id*. at 4.) Additionally, petitioner's MRI scan revealed two small areas of hyperintensity in the cerebral white matter and one in the left thalamus, where it was concluded that the findings were "nonspecific but compatible with MS." (*Id*.) Dr. Wallace reported that "other than finding evidence for a probable sensory neuropathy, [he was] not able to make a diagnosis," and that there were insufficient findings to support a diagnosis of multiple sclerosis.

On September 11, 2012, petitioner saw Dr. David E. Jones at the James Q. Miller MS clinic for a consultation. (Ex. 5, p. 4.) Again, petitioner reported that she experienced symptoms of burning sensation, muscle spasm, cold paresthesia, and urinary frequency since 2006. (*Id*.) In 2009, petitioner started feeling shocks in her arms. Petitioner indicated that in March 2012, she began having ambulatory dysfunction and in July 2012, she was experiencing severe pain in her lower extremities. Dr. Jones noted that he reviewed some neurology records that appeared to be consistent with petitioner's report and therefore, he "suspects that she was reading the list of symptoms from the same notebook that she brings with her today." (*Id*.) Dr. Jones also noted that petitioner was concerned about MS, although her MRI was not indicative of MS. (*Id*.) Upon examination, Dr. Jones indicated that vibration was reportedly less in the right medial malleolus and reflexes were normoactive and symmetric in upper and lower extremities. (*Id*. at 5.) His impression was that petitioner had numerous non-specific symptoms of unclear etiology. Dr. Jones stated that "[a]lthough her neurological examination is clouded by functional overlay, I can not say that there is not underlying organic abnormalities; however, the MRI above is not overly [s]uggestive of multiple sclerosis." (*Id*. at 6.)

Petitioner saw Dr. Jones again on December 11, 2012. (Ex. 5, p. 7.) At this visit, she reported various symptoms including joint pain, pain across her thorax, left eye

jumpiness, electric static noise in her right ear, persistent cough, cold feeling in her back, severe dizziness with nausea, difficulty organizing thought, and more. (*Id*. at 8.) Dr. Jones reviewed her tests and indicated that her exam was normal and that petitioner did not meet the criteria for MS. (*Id*. at 10.) However, Dr. Jones wanted to proceed with a spinal tap. (*Id*.)

Petitioner's January 18, 2013, brain MRI noted that there were multifocal white matter abnormalities, which supports a diagnosis of a demyelinating disease. (Ex. 7, p. 23.) On January 22, 2013, petitioner returned to see Dr. Jones. (Ex. 5, p. 11.) At this visit, petitioner complained of pins and needs in both of her lower and upper extremities, pain in her right jaw, and aching, throbbing pain in both hands. (*Id*. at 12.) Here, Dr. Jones indicated that there was evidence of inflammation from her spinal tap results, but that this is insufficient to diagnose MS. (*Id*. at 14.)

On April 12, 2013, petitioner sought treatment with Dr. Vara P. Bonagiri for a follow up regarding multiple sclerosis, which was noted to be a new diagnosis. (Ex. 16, p. 21.) Petitioner reported still experiencing intermittent numbness and requested a neurology referral. (*Id*.)

Petitioner visited Dr. Jones again on September 16, 2013. (Ex. 5, p. 15.) Petitioner reported some improvements in her symptoms. (*Id*. at 16.) Dr. Jones noted that he "struggle[d] to explain [petitioner's] symptom complex from a neuroanatomical point of view, and one must consider whether her disorder lies in the borderlands between neurology and psychiatry." (*Id*. at 17.) Additionally, petitioner saw Dr. Jones again in January 2014, where the exam was essentially similar without much progress in diagnosing petitioner with MS. (*Id*. at 20-21.) However, petitioner's brain MRI taken on September 23, 2013, found "small areas of elevated T2 signal in white matter of cerebral hemispheres consistent with given diagnosis of multiple sclerosis." (Ex. 7, p. 17.)

Petitioner went to Bon Secours Neurology Clinic at Memorial Regional to see Dr. Meghan Rodden as a new patient for possible demyelinating disease on January 23, 2014. (Ex. 12, p. 5.) Petitioner provided her medical history, stating that in 2005, she had burning pain in her right foot and knee, which continued until 2007, progressing to back pain and pins and needles in her wrists. Further, in 2009, she caught the swine flu and in 2011, she got chicken pox. (*Id*. at 6.) Additionally, petitioner stated that in August 2012, her MRI showed some areas of increased signal changes that are consistent with MS and her spinal tap was positive for six oligoclonal bands. (*Id*.) Upon examination, Dr. Rodden noted 4/5 regarding strength on petitioner's right side and decreased sensory pinprick perception "everywhere" except left lower extremity. (*Id*. at 9.) Dr. Rodden indicated that petitioner's imaging showed white matter lesions and therefore, based on her prior workup and treatment, petitioner "needs to be on disease modifying therapy for MS." (*Id*. at 9.)

Thereafter, on March 24, 2014, petitioner returned to see Dr. Rodden after being treated with Rebif (an anti-inflammatory). (Ex. 12, p. 21.) Petitioner felt better during

this visit, but indicated that she had numbness, fatigue, and electrical shocks sensations for about three days. (*Id*.) Petitioner was ordered to continue Rebif and try Provigil for MS fatigue. (*Id*. at 23.) Three months later in June 2014, petitioner visited Dr. Rodden again and reported that she had a flare (blurred vision, weakness on her right side, dysphagia) since her last visit. (*Id*. at 45.) Dr. Rodden then ordered repeat imaging and repeat evaluation of treatment due to petitioner's "significant decline." (*Id*. at 47.) When petitioner returned for a follow up on September 15, 2014, petitioner was "doing well" and taking Rebif for MS management. (*Id*. at 60.) Additionally, aside from MS, fatigue, and vision abnormalities, petitioner was additionally assessed with nocturnal leg cramps. (*Id*. at 61.)

Petitioner visited Farmville Internal Medicine on July 28, 2014 with complaints of cold symptoms. (Ex. 36, p. 4.) Petitioner reported that she was diagnosed with MS and had been receiving treatment. (*Id*.) Petitioner did not report any weakness, muscle pain, paresthesia, nor numbness. (*Id*.)

On October 13, 2014, petitioner experienced elevated blood pressure, sinus infection, cough, and shortness of breath. (Ex. 16, p. 12.) Petitioner saw Dr. Bonagiri concerning these symptoms. Dr. Bonagiri recorded MS as part of petitioner's medical history. (*Id*.) On November 13, 2014, petitioner saw Dr. Bonagiri again for a follow up concerning her hypertension. (Ex. 16, p. 10.) Petitioner reported no complaints of foot numbness or paresthesias at this visit and denied any pain in muscles or joints.[6] (*Id*.) Petitioner's condition was under control and petitioner was ordered to continue with medication. (*Id*. at 10-11.)

Also on October 13, 2014, petitioner had a neurology consult with Dr. John Hennessey regarding her visual complaints in both eyes. (Ex. 12, p. 68.) On exam, petitioner had 5/5 motor strength in upper and lower proximal and distal muscles and a score of 2+/4 regarding deep tendon reflexes. (*Id*. at 70.) Dr. Hennessey indicated that petitioner's complaints could be optic neuropathy from MS, but also could be an intrinsic eye disease like monocular diplopia. (*Id*. at 70-71.)

Petitioner received another flu vaccination on November 2, 2014. (Ex. 26.) On November 13, 2014, petitioner went to Farmville Internal Medicine for a follow up regarding her hypertension. Petitioner did not have any new complaints and denied any headache, chest pain, foot numbness or paresthesia. (Ex. 36, p. 8.)

Petitioner returned to Bon Secours Health System on November 20, 2014, but was seen by Diane L. Burton, N.P. instead. (Ex. 12, p. 79.) During this visit, petitioner complained of numbness and tingling in all of her extremities and increased weakness in her legs. (*Id*.) Petitioner indicated that the numbness, tingling, and vibration in her legs and hands started in October 2011, following her flu shot. However, the numbness

[6] Similarly, during other visits on January 15, 2014 and July 28, 2014 with Dr. Bonagiri, petitioner also denied pain in muscles or joints and did not have any limitation regarding range of motion, paresthesia, or numbness. (Ex. 16, pp. 14, 16.)

and gait impairment showed improvements after nine months, but after receiving the flu shot "6 weeks ago," her neuropathic symptoms started again. (*Id.*) It was noted that due to the unchanged MRI and generalized presentation, petitioner did not have a MS flare or exacerbation. (*Id.*) N.P. Burton noted "unsure of the relationship to the Flu shot at this time." (*Id.*) Physical examination revealed 4/5 strength in bilateral lower extremity and 1+ in reflexes of bilateral lower extremities. (*Id.* at 83.) Bilateral leg weakness, polyneuropathy, bilateral arm weakness, and back pain were added to petitioner's problem list. (Ex. 12, p. 44.)

On January 13, 2015, petitioner had a follow up with Dr. Rodden regarding her EMG/NCS. (Ex. 20, p. 19.) Here, petitioner was recorded as being sick since her flu shot in 2012, [7] but her symptoms had worsened. Petitioner still experienced numbness in her hands and feet and had electrical sensations and sharp pains. (*Id.*) Dr. Rodden noted that previous medical providers, after reviewing repeat MRIs, ruled out MS as a diagnosis. (*Id.*) Petitioner's electrodiagnostic study was abnormal and revealed severe bilateral carpal tunnel, mild bilateral ulnar sensory neuropathy, moderate polyneuropathy in her legs, and probable L5 radiculopathy. (*Id.* at 6.) Carpal tunnel syndrome and lumbar radiculopathy was added to petitioner's problem list on this visit. (Ex. 12, p. 44.)

On January 22, 2015, petitioner transitioned into care with Dr. Matthew T. Mayr regarding her bilateral leg numbness and weakness. (Ex. 48, p. 1.) Dr. Mayr recorded, as part of petitioner's history of present illness, that she began having flu-like illness after receiving a flu shot in November 2011, and then began having trouble walking, electric shocks down her arms and legs, rashes, and joint pain. (*Id.*) Additionally, petitioner reported that she was diagnosed with peripheral neuropathy in 2012, but after being unable to walk due to the weakness, she was diagnosed with MS in 2014. (*Id.*) At this visit, petitioner added that she continued having electrical shocks in her arms and legs, trouble walking, nausea, vomiting, blurriness, diplopia, face numbness, and falls. (*Id.*) Petitioner's physical examination was generally normal and Dr. Mayr indicated that petitioner's imaging studies were normal except her MRI showed a grade 2 spondylolisthesis and neural foraminal narrowing. (*Id.* at 3.) Petitioner was assessed with spondylolisthesis of lumbar region, but Dr. Mayr indicated that this condition does not explain the electric shock feelings. (*Id.* at 2-3.) Dr. Mayr noted that petitioner's findings "sounds more like cord involvement, probably from her multiple sclerosis. It is unusual to not have any cord signal change with such profound symptomology." (*Id.* at 2.)

On March 16, 2015, petitioner visited Farmville Internal Medicine regarding her hypertension and a burn injury on her left thigh. (Ex. 36, p. 10.) Petitioner reported that she fell down when dumping hot grease and moreover that she has a history of falls due to her MS/CIDP. (*Id.*) Petitioner did not report any muscle or joint pain, numbness, or weakness at this visit. (*Id.*)

[7] Petitioner received her flu shot in 2011, not 2012.

Petitioner had a sensory nerve conduction study conducted on March 24, 2015, at Johns Hopkins. The results indicated that the bilateral sural, median, and radial nerves showed no response and the right ulnar nerve also showed no response. (Ex. 21, p. 6.) This study concluded that there is electrophysiologic evidence of a severe generalized sensory neuropathy or neuronopathy as well as superimposed median neuropathy in both wrists. (*Id.*) Additionally, on March 24, 2015, petitioner had a consultation and evaluation with Dr. Mohammad A. Khoshnoodi regarding her neuropathy in both arms and legs. (Ex. 21, p. 1.) From the new patient questionnaire, Dr. Khoshnoodi summarized petitioner's history of present illness as:

> [M]ultiple complaints, including numbness, falls, inability to perform fine motor tasks with her hands, paresthesia and many others. [Petitioner] reports that she was at her usual state of health until about 2011, working as a surgical nurse assistance/ [sic]. At that time, she received a flu shot and developed flu symptoms few days later. Along with flu symptoms, she developed numbness and paresthesia all over her body, in her toes, fingers, trunk and even the face.

(Ex. 21, p. 1.)

Additionally, petitioner reported that she was feeling "slightly better until she received another flu shot last winter that made everything worse." (*Id.*) On examination, Dr. Khoshnoodi noted that pinprick sensation and temperature sensation were reduced in a patchy distribution, vibration sensation was reduced at the great toe bilaterally, and gait was slightly wide-based but not unsteady. (*Id.* at 4.) Dr. Khoshnoodi noted that petitioner's complaints were mostly sensory, and examination revealed that she had normal reflexes and no sensory ataxia in her upper extremities. (*Id.*) From his findings, he posited various possible diagnosis including idiopathic sensory neuronopathy and an acute form of sensory GBS. Dr. Khoshnoodi was not convinced petitioner had MS. He also noted that her condition could have happened in 2011 after the flu shot and resulted in residual neuronopathy or sensory neuropathy because "it has been reported that reflexes can be preserved in this condition which makes it more likely compared to other causes of neuronopathies." (*Id.*)

Petitioner returned to see Dr. Rodden on April 16, 2015. (Ex. 20, p. 47.) Here, petitioner reported that following her second flu shot in November 2014, her symptoms including leg weakness, were exacerbated. Petitioner reported sensory changes in both arms and legs and tongue numbness. (*Id.*) Dr. Rodden noted that petitioner was diagnosed with sensory GBS at Johns Hopkins and further, she assessed petitioner with sensory variant GBS likely related to her flu shot in 2011 at this visit. (*Id.* at 49.)

Through referral from Dr. Khoshnoodi, petitioner began physical therapy at Progressive Therapy. (Ex. 49.) Her physical therapy was for a diagnosis of nerve root/plexus disorder, where petitioner "started with pins and needles in both of her legs," and stumbling at the onset of her symptoms in October 2011. (*Id.* at 1.) Petitioner's prior level of function was noted to be fine with the exception of asthma before October

2011. (*Id*.) It was noted that petitioner had lower extremity and core strength deficits, balance deficits, loss of sensation and pain in the lower extremities, and GBS. (*Id*. at 5.) Based on the evaluation of her right upper extremity, petitioner showed signs and symptoms consistent with her diagnosis and petitioner's rehabilitation potential was good. (*Id*. at 3.) Petitioner's therapy plan was twice a week for six weeks. (*Id*.) During her third session, "a flat affect with hoarse vocal quality," asymmetry of the face and eyes, and reduced muscle movement in areas when talking, were noted. (*Id*. at 12.) Thereafter, an evaluation for speech sound production and language comprehension and expression was conducted. (*Id*. at 13.) By October 8, 2015, petitioner was making good progress in speech therapy. (*Id*. at 29.)

On June 30, 2015, petitioner had another follow up regarding her hypertension with Dr. Bonagiri. (Ex. 36, p. 12.) Petitioner reported that her MS diagnosis was questionable, but now she was diagnosed with polyneuropathy. Petitioner reported that she was no longer taking the flu vaccine. (*Id*.) Petitioner continued seeing Dr. Bonagiri at Farmville Internal Medicine regarding her hypertension thereafter and into 2020. (*See generally* Ex. 36, pp. 15-47.)

Petitioner began seeking treatment from Dr. Shi Yun Lim at Centra Southside Neurology Clinic on February 2, 2016 regarding fainting symptoms. (Ex. 33, p. 125.) Dr. Lim recorded that petitioner has a history of GBS, paraneoplastic syndrome, sensory polyneuropathy, and autonomic disorder. Additionally, petitioner has a history of fainting spells, blurry vision, weakness in her arms, vertigo, intermittent dizziness, shortness of breath, and numbness and tingling in her lower extremities. Petitioner exhibited normal tone, strength, and reflexes, however there was a loss of pinprick sensation in her lower extremities. (*Id*. at 127.) Dr. Lim referred petitioner to the neuromuscular clinic for further evaluation. (*Id*.) Petitioner's EMG results showed no response of the right median sensory, right radial sensory, and right ulnar sensory nerves in her wrist. Additionally, there were no response in her right superficial peroneal sensory and right sural sensory nerves in her calf. (Ex. 37, p. 21.) Petitioner continued seeing Dr. Lim for follow up appointments about every six months thereafter. (Ex. 33, p. 98-124.)

On March 31, 2016, petitioner went to Centra Southside Community Hospital for a headache.[8] (Ex. 35, p. 47.) At this visit, it was noted that petitioner was receiving infusion treatments for GBS related to her flu vaccine. (*Id*. at 50.) Petitioner reported that "she got influenza vaccine in 2011 and says she was misdiagnosed then received it again in 2015. She ended up being hospitalized at Johns Hopkins and diagnosed with GB." (*Id*.) She was discharged home on the same day. (*Id*. at 56.)

On April 15, 2016, petitioner had an evaluation of paraneoplastic antibodies and neuropathy with Dr. Nicholas Paphitis. (Ex. 37, p. 26.) Dr. Paphitis stated that

[8] Petitioner was admitted to Centra Southside Community various times throughout 2015 to 2017 for different reasons including first degree burn, heart fluttering, abdominal pain, fever, and chest pain. (*See* Ex. 35, pp. 2-18.)

petitioner has a long history of neurologic symptoms and abnormalities and possible dysautonomia. Additionally, petitioner tested positive for paraneoplastic antibodies. Petitioner was assessed with neuropathy. (*Id*. at 30.)

Petitioner saw Dr. Aaron Alan Cochran on April 21, 2016 at the outpatient Neuromuscular Clinic at the University of Virginia for an initial consultation. (Ex. 37, p. 31.) Petitioner stated that she started having symptoms of pins and needles sensation in feet and hands, fevers, headaches, urinary frequency, muscle and joint pain, and difficulty staying awake during the day in 2011 about 2-3 weeks after receiving the flu vaccine. (*Id*.) She was treated with antibiotics without relief and thereafter was diagnosed with lupus, so she started prednisone. (*Id*.) It was recorded that petitioner's most recent EMG/NCS showed severe sensorimotor peripheral neuropathy and additionally, her voltage gated potassium channel antibody was abnormal. (*Id*. at 35.) IV-Solu-Medrol was prescribed for petitioner. Dr. Ted M. Burns, also from University of Virginia added that, "very complicated case. It's challenging to know, among others, what symptoms – if any- are related to the VGKC antibody titer. It's likely some are and I hope that immunotherapy will help [petitioner]." (*Id*. at 36.)

In a follow up at the Neurology Clinic at University of Virginia on July 7, 2016, Dr. Lawrence Phillips indicated that petitioner did not respond to treatment. (Ex. 37, p. 53.) Dr. Phillips recorded that petitioner's symptoms were relatively stable from 2011 until November 2014, when her symptoms worsened. (*Id*. at 50.) Dr. Phillips further stated that "[t]he reported clinical course with onset after influenza vaccination and relative stability save for worsening after repeat vaccination is suspicious for potential association, but we cannot say for certain that her vaccinations were the definite triggers for her clinical syndrome." (*Id*. at 53.)

On June 1, 2017, petitioner was admitted to Centra Southside Community Hospital again for increasing muscle cramps and increasing numbness and tingling in both arms and legs. (Ex. 35, p. 86-87.) Petitioner was diagnosed with numbness and headache and discharged home on the same day. (*Id*. at 90.)

On March 8, 2018, petitioner had a follow up appointment at Centra Southside Neurology Clinic for her migraine headaches. (Ex. 33, p. 90.) Additionally, petitioner still had sensory symptoms and burning sensations all over her arms and legs as well as significant muscle spasms and weakness.[9] (*Id*.) Petitioner also reported suffering from fainting spells. (*Id*.) Her physical examination revealed mild weakness in right lower extremity and loss of pinprick sensation in upper and lower extremities. (*Id*. at 92.)

Petitioner had a neurology exam on September 10, 2018 with Dr. Patricia J. Shipley. (Ex. 33, p. 4.) Petitioner complained of cough, difficulty swallowing, shocking sensation, and leg cramps. (*Id*. at 5.) Petitioner's history of present illness noted that petitioner had numbness and tingling in her upper and lower extremities in 2011, two or

[9] This note was similarly recorded during petitioner's visit on July 13, 2017. (Ex. 33, p. 93.)

three weeks after receiving a flu shot. (*Id*.) Petitioner reported receiving IVIG in April 2016 with modest response to her symptoms. (*Id*.) On physical examination, petitioner had sensory ataxia and her muscles reflexes were 1+ absent in the ankles. Additionally, petitioner showed decreased modalities to sensory level of T6. (*Id*. at 7.) Dr. Shipley noted that petitioner had questionable GBS with residual pain paresthesia, sensory ataxic gait, and autonomic dysfunction. (*Id*. at 4.) Dr. Shipley increased petitioner's prescription of gabapentin. However, at a follow up appointment on December 7, 2018, petitioner did not find relief from her nerve pain when increasing gabapentin dosage. (*Id*. at 73.)

On April 30, 2019, petitioner had a procedure regarding a left ankle fracture. (Ex. 34, p. 159.) Petitioner slipped and fell while walking around her pool and broke her ankle. Petitioner was at CMG Southside Neurology Center again on November 11, 2019 and saw Dr. Sanam Anwer[10] for a follow up appointment regarding her neuropathy. (Ex. 33, p. 23.) Petitioner was noted to have started suffering from pins and needles in her lower and upper extremities in 2014/2015 after receiving her flu shot and her EMG/NCS indicated nerve loss. (*Id*. at 26.) At this visit, petitioner complained of pins and needs, burning sensation, and fasciculation. (*Id*.) Regarding petitioner's history of GBS, Dr. Anwer noted that because petitioner did not have frequent relapses, chronic steroid therapy was not needed. (*Id*. at 25.) Dr. Anwer indicated that petitioner was "extremely anxious" and discussed pain management with her. Additionally, Dr. Anwer stated that petitioner was diagnosed with GBS in 2014 but "there have been no change in fasciculations [and] there is no visible atrophy today on exam, the patient herself has not seen any muscle loss. In short, whatever her symptoms are have been present since 2014, they only have exacerbations when she is sick." (*Id*. at 25.)

b. As Reflected by Testimony/Affidavits

i. Petitioner

Petitioner presented two affidavits in this case. (Exs. 1, 23.) The first affidavit was signed on November 13, 2014, shortly before the initial petition was filed. (Ex. 1, p. 2.) The second affidavit was signed on July 6, 2015. (Ex. 23, p. 5.) She also testified during the hearing. (Tr. 226-388.)

In her first affidavit, petitioner averred that she had no major medical issues before her receipt of the flu vaccination on September 29, 2011. (Ex. 1.) She stated that around November 21, 2011, before Thanksgiving of that year, she started experiencing muscle weakness and numbness in her legs and hands. She also began experiencing feeling pins and needles, multiple joint pains, and low-grade fevers. (*Id*.) Due to her trembling hands, it was difficult to carry out her job as a surgical assistant. She indicated that she was prescribed antibiotics as treatment; however, her symptoms continued, and she began having gait disturbance in late June 2012. (*Id*.) Additionally, she had numbness in her tongue, lips, and chin. Petitioner averred that she was

[10] Petitioner first saw Dr. Answer on May 13, 2019. (Ex. 33, p. 48.)

diagnosed with peripheral neuropathy and was still experiencing symptoms that resulted in limitations of movement. (*Id*. at 2.)

In her supplemental affidavit, petitioner addressed her symptoms prior to her September 29, 2011 vaccination. (Ex. 23.) Petitioner addressed the records from Neurology Associates of Lynchburg and Virginia Health System Hospital, claiming that such records were inaccurate. (*Id*. at 1.) Petitioner explained that she started experiencing a tired and aching sensation in her lower legs in 2006; however, this sensation was different than the pins-and-needles feelings she experienced subsequent to her September 29, 2011 vaccination. Petitioner attributed the pain in her legs to working long hours standing on her feet. (*Id*. at 2-3.) Additionally, petitioner averred that she used to get cramps, aches, and shock-like feelings in her arms due to working for extended periods of time and she spoke to a doctor about this in 2009. (*Id*. at 3.) Further, petitioner stated that prior to her second flu shot on November 2, 2014, she was able to function despite her symptoms. However, two weeks after the second flu shot, she felt like she had been "run over by a truck. Specifically, [she] was having severe joint pain and had difficulty walking with an abnormal gait. The vibration in [her] extremities had returned with a vengeance. [She] had severe pins-and-needles sensations and was also experiencing shortness of breath once again." (*Id*. at 4.)

During the hearing, petitioner indicated that she was previously diagnosed with plantar fasciitis and that this was her only significant health problem prior to her 2011 flu vaccine. (Tr. 229-30.) She indicated that complaints in her later medical records identifying leg pain dating back to about 2006 or 2007 are attributable to that condition. (Tr. 230-32.) She denied that her prior leg symptoms included any "pins and needles" or burning sensations. (Tr. 232-39.) As will be discussed further below, petitioner explained that she prepared the list of symptoms for Dr. Wallace which appears in the record at Exhibit 4, page 10 in 2012. She disagrees with his interpretation of her list, but found that Dr. Wallace's notes were nonetheless carried forward by other treating doctors. (Tr. 232-39, 333-39, 341.)

Petitioner indicated that the change in her recollection of the initial onset of her post-vaccination symptoms from November to October of 2011 is attributable to a photograph that she found. She explained that the photo shows her driving her grandchild around for trick-or-treating on Halloween. (Tr. 240-41, 328-29.) She recalled that she had to drive that year because of her symptoms, prompting her to realize onset was prior to Halloween. (*Id.*) Petitioner testified that her initial bout of symptoms got better after about eight to nine months, but then returned "with a vengeance" after her second flu vaccine in November of 2014. (Tr. 253-54.)

Petitioner described three moments as very significant to her understanding of her condition. These moments caused her to reassess her prior medical history in hindsight. She explained that when N.P. Yesalis first placed her on gabapentin, the relief she experienced caused her to realize that the pain she had been experiencing was not joint pain, but rather nerve pain. (Tr. 304-06, 309.) She also indicated that when she experienced what she perceived to be an exacerbation of her condition

following the November 2014 vaccination, she was prompted to realize her condition initially manifested after her 2011 vaccination and was therefore likely caused by her vaccinations. (Tr. 368-69.) Finally, she indicated that her first appointment with Dr. Khoshnoodi was the first time anyone discussed the idea that her condition might be diagnosed as sensory variant GBS and that she had not previously contemplated that possibility. (Tr. 295, 343-44.)

ii. Diane Aslanis, D.O.

Petitioner filed a letter dated May 19, 2015 from Diane Aslanis, D.O., a former colleague of petitioner. (Ex. 24.) Dr. Aslanis also testified during the hearing. (Tr. 148-58.) Dr. Aslanis is OBGYN surgeon and worked with petitioner at Centra Southside Community Hospital during 2010 to 2011. (Ex. 24, p. 1; Tr. 149.) They saw each other about 2-3 times per week. (*Id.*) Dr. Aslanis stated that petitioner's work demanded her to stand for long periods of time and pay close attention to detail. She stated that petitioner "was an excellent surgical assistant and performed her duties more than adequately" and that she "never heard Barbara complain of not feeling well" apart from plantar fasciitis. (Ex. 24, p. 1; Tr. 150-51.)

Dr. Aslanis recounted that she recommended petitioner start wearing Danskos shoes instead of Crocs, noting that petitioner has high arches. She indicated that petitioner later indicated the new shoes had alleviated plantar fasciitis she had been experiencing since 2009. (Ex. 24, p. 1; Tr. 150-51.) She further indicated that "[a]round the middle of November, 2011, and certainly by Thanksgiving of 2011, Barbara did start to complain about pins and needles in her upper and lower extremities." (Ex. 24, p. 1.) Dr. Aslanis recalled additional symptoms of hand weakness, vibration sensation (fingertips and lower extremities), and numbness in her lower extremities and tongue. She noted that petitioner attributed her pins and needles to weight gain from a prior hysterectomy, but also observed onset of symptoms to be occurring in the context of coughing attacks and flu-like symptoms. (Ex. 24, pp. 1-2; Tr. 152.)

Dr. Aslanis left Centra Southside Community Hospital in September of 2012. (Ex. 24, p. 2.) After that, she says of petitioner that "I didn't see her at all. I had no relationship with her after that." (Tr. 157.) However, she did subsequently find out about petitioner's GBS diagnosis, but could not recall the context in which petitioner told her about it. (Ex. 24, p. 2; Tr. 152-53, 157-58.) In contrast, petitioner testified that she sought spinal adjustments from Dr. Aslanis on multiple occasions afterhours that helped alleviate a limp and that on one occasion she mentioned the GBS diagnosis after being invited to look at Dr. Aslanis's blown glass collection. (Tr. 374-75.) That was the last time they saw each other prior to the hearing. (*Id.*)

iii. Terry A. Brooks, CRNA

Petitioner also filed a letter dated May 18, 2015 by Terry A. Brooks, CRNA, who worked at Centra Southside Hospital as a nurse anesthetist from 2009 to 2011. (Ex. 25.) Ms. Brooks also testified during the hearing. (Tr. 272-89.) Ms. Brooks stated that

petitioner was previously "extremely healthy." (*Id*. at 1.) Ms. Brooks recalled that petitioner mentioned having pins and needles sensations in her legs and increasing weakness in her hands that started in November 2011. (*Id*. at 1.) Ms. Brooks also wrote that she has a record of dates of petitioner's symptoms through Facebook messenger. (*Id*.)

Ms. Brooks explained that she left Centra Southside Hospital in early 2011 and cannot speak to petitiner's condition after that time except as relayed in their later conversations. (Tr. 277-78.) She indicated that she stayed in touch with petitioner by phone and on Facebook messenger and that petitioner discussed her medical condition with her over the course of years. (Tr. 277-78, 281-82, 285.) Ms. Brooks suggested that petitioner relied on her for medical input and asked her questions about neurological diseases. (Tr. 281-83.) She advised petitioner to seek a neurological evaluation and to see a neuromuscular specialist; however, she could not recall the specifics of what they discussed. (Tr. 276, 278, 283-84.) Initially petitioner suspected that she had a cervical or back injury. (Tr. 282-83, 288-89.)

iv. Mohammed Khoshnoodi, M.D.

Dr. Khoshnoodi is one of petitioner's treating neurologists. He is a board-certified neurologist and an assistant professor of neurology at Johns Hopkins University, Department of Neurology, Division of Neuromuscular Disorders. (Tr. 100.) He submitted two opinion letters to the court, responded to written questions by the previously presiding special master, and testified during the hearing. (Exs. 22, 37, 38; Tr. 100-146.)

His initial letter to the court addressed post-2011 diagnosis only. (Ex. 22.) Based on her EMG/NCS study, complaints of chronic pain, paresthesia, and sensory loss, clinical findings of preserved reflexes, and acute onset of symptoms following her flu vaccination, he opined that petitioner very likely has a sensory variant of GBS. (*Id*.) In a subsequent letter he additionally asserted that petitioner's 2014 vaccination "result[ed] in worsening of her symptoms." (Ex. 37.) Upon further questioning from the previously assigned special master, he identified only petitioner's subjective complaints as evidence of worsened symptoms. (Ex. 38, p. 2.) During the hearing he answered additional questions relating to the basis for his diagnosis. (Tr. 100-146.) That testimony is discussed further below.

III. Expert Opinions

a. Petitioner's Expert, Beatrice C. Engstrand, M.D., F.A.A.N.

Petitioner filed a report from Dr. Engstrand to support her claim. (Ex. 29.) Dr. Engstrand is board certified in neurology and is currently in private practice. She is also a clinical associate professor of neurology at New York Medical College. (Ex. 29B, p. 1.) She was accepted without objection as an expert in neurology. (Tr. 11.)

Dr. Engstrand indicates that petitioner developed weakness in her legs and hands, numbness, and tingling seven weeks after her receipt of the flu vaccination on September 29, 2011. (Ex. 29, p. 2.) Moreover, Dr. Engstrand opines that after receiving her second flu vaccination on November 2, 2014, petitioner's symptoms were exacerbated. She indicates that petitioner developed a sensory variant of GBS from her 2011 flu vaccine. (*Id*. at 3.) Dr. Engstrand notes that there is a significant association between seasonal flu vaccines and GBS, stating that the "risk in adults ranges from two to five GBS cases per 100,000." (*Id*. at 4.)

Dr. Engstrand explains that GBS is a disorder where the immune system attacks the gangliosides of the peripheral nervous system and the onset of symptoms include weakness or tingling sensations in the legs. (Ex. 29, p. 4.) She further explains that neurological complications that occur following vaccinations are caused by T-cell mediated immune reactions. (*Id*.) Additionally, through molecular mimicry, infectious agents, like a vaccine, trigger an immune response against autoantigens, and GBS follows such inflammation. (*Id*.) Despite petitioner's increased reflexes, looking at petitioner's objective testing, Dr. Engstrand indicates that petitioner showed slow nerve conduction signals and elevated IgG, supportive of a GBS diagnosis.

Dr. Engstrand also testified in accordance with her report. Her opinion and testimony are discussed in greater detail below.

b. Respondent's Expert, Vinay Chaudhry, M.D.

Respondent filed two reports from Dr. Chaudhry. (Exs. A, C.) Dr. Chaudhry is a Professor of Neurology at the Johns Hopkins University School of Medicine. He is additionally the director of the EMG laboratory at Johns Hopkins Hospital. Dr. Chaudhry is board certified in neurology, neuromuscular disease, electrodiagnostic medicine, and clinical neurophysiology. (Ex. A.) He was accepted without objection as an expert in neurology, neuromuscular disease, electrodiagnostic medicine, and clinical neurophysiology. (Tr. 399.)

Upon evaluating petitioner's medical records, Dr. Chaudhry found that petitioner has had multiple symptoms over the years and has been diagnosed with various conditions including lupus, GBS, sensory neuropathy, and multiple sclerosis, but in his professional opinion, none of petitioner's medical issues are related to the flu vaccine. (Ex. A, p. 9.) Dr. Chaudry indicated that petitioner suffered several symptoms (pins and needles, burning sensation, muscle spasm, cold paresthesia, etc.) prior to the September 29, 2011 flu vaccination and all symptoms prior to her receipt of the flu vaccination on November 2, 2014.

Dr. Chaudhry concluded that petitioner does not suffer from GBS. He explained that petitioner did not experience the symptoms that are indicative of GBS, including progressive weakness in legs and arms, areflexia, and elevated spinal fluid protein. Moreover, "GBS is a monophasic illness that peaks at less than 4 weeks and improves after that," while petitioner's condition appears progressive over many years and

remains chronic. (Ex. A, pp. 10-11.) Dr. Chaudhry pointed out that petitioner's objective testing did not show demyelination, as seen in GBS patients. Because petitioner's symptoms "were varied, fluctuating, and ranged from joint pains to numbness to bladder symptoms to visual disturbance," petitioner suffers a sensory neuropathy, but not GBS. (*Id*. at 11.)

In his supplemental report, Dr. Chaudry continued to question petitioner's diagnosis of sensory variant of GBS, emphasizing that weakness, fevers, joint pains, joint stiffness, muscle pains, fatigue, headaches, urinary frequency, shortness of breath, and other symptoms petitioner experienced are not associated with this condition. (Ex. C, p. 1.) On the other hand, areflexia and albumincytological dissociation, symptoms petitioner did not experience, are associated with sensory variant GBS. Additionally, petitioner's treatment was not typical of GBS, especially prednisone, which can worsen the condition. (*Id*. at 2.) Applying the Brighton criteria that Dr. Engstrand mentioned, petitioner failed to meet several criteria. (*Id*.) Again, Dr. Chaudry stressed that petitioner experienced symptoms prior to the 2011 flu vaccination and thus, he disagreed with Dr. Engstrand's theory that the 2014 flu vaccination exacerbated petitioner's condition.

Dr. Chaudhry also testified in accordance with his reports. His opinion and testimony are discussed in greater detail below

IV. 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, it may be shown that the vaccine recipient 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. If so, the Table Injury is presumed to have been caused by the vaccination, and the petitioner is automatically entitled to compensation, unless it is affirmatively shown that the injury was caused by some factor other than the vaccination. § 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* of the type covered in the Vaccine Injury Table. In such instances, an alternative means exists to demonstrate entitlement to a Program award. That is, the petitioner may gain an award by showing that the recipient's injury was "caused-in-fact" by the vaccination in question. § 300aa-13(a)(1)(B); § 300aa-11(c)(1)(C)(ii). In such a situation, of course,

the presumptions available under the Vaccine Injury Table are inoperative. The burden is on the petitioner to introduce evidence demonstrating that the vaccination actually caused the injury in question. *Althen v. Sec'y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005); *Hines v. Sec' of Health & Human Servs.*, 940 F.2d 1518, 1525 (Fed. Cir. 1991).

The condition at issue in this case, GBS, is currently a Table Injury for the influenza vaccination if it occurs between three and 42 days after vaccination. 42 C.F.R. § 100.3(a)(XIV). However, the petition in this case, dating back to November of 2014, was filed prior to the March 21, 2017 addition of GBS to the Vaccine Injury Table. *See* National Vaccine Injury Compensation Program: Revisions to the Vaccine Injury Table, Final Rule, 82 Fed. Reg. 6294, Jan. 19, 2017; National Vaccine Injury Compensation Program: Revisions to the Vaccine Injury Table, Delay of Effective Date, 82 Fed. Reg. 11321, Feb. 22, 2017 (delaying the effective date of the final rule until March 21, 2017). Accordingly, petitioner must bear the burden of demonstrating causation-in-fact without the benefit of a presumption of causation.[11] § 300aa-14(c)(4) (explaining that modifications to the Vaccine Injury Table "apply only with respect to petitions for compensation under the Program which are filed after the effective date of such regulation").

The showing of "causation-in-fact" must satisfy the "preponderance of the evidence" standard, the same standard ordinarily used in tort litigation. § 300aa-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 was the cause of the 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 at least a "substantial factor" in causing the condition, and was a "but for" cause. *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999). Thus, the petitioner must supply "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

[11] As noted above, the previously presiding special master noted that petitioner could dismiss and refile her petition to take advantage of the addition of GBS to the Vaccine Injury Table (ECF No. 65); however, this was never done. Under the terms of the Vaccine Act, the ability to do so expired two years after the change was made to the table. §300aa-16(b).

> 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 noted that a petitioner need not necessarily supply evidence from medical literature supporting petitioner's causation contention, so long as the petitioner supplies the medical opinion of an expert. *Id*. at 1279-80. The court also indicated that, in finding causation, a Program fact-finder may rely upon "circumstantial evidence," which the court found 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.

This case also presents an additional question. Petitioner's initial petition alleging that her GBS was initially caused by her 2011 influenza vaccine was determined to have been untimely filed when petitioner amended her allegation to include an onset date occurring in October of 2011. (ECF No. 65.) This left petitioner's additional claim that her condition was significantly aggravated by her subsequent 2014 influenza vaccination, as presented in her amended petition, as the operative claim. The Vaccine Act allows petitioners to assert claims for any vaccine-related significant aggravation of a pre-existing condition.

A significant aggravation is "any change for the worse in a preexisting condition which results in markedly greater disability, pain, or illness accompanied by substantial deterioration of health." 42 U.S.C. § 300aa-33(4). Where a petitioner in an off-Table case is seeking to prove that a vaccination aggravated a pre-existing injury, petitioners must also establish three further factors in addition to the above-discussed *Althen* factors. *See Loving v. Sec'y of Health & Human Servs.*, 86 Fed. Cl. 135, 144 (Fed. Cl. 2009) (combining the first three *Whitecotton* factors for claims regarding aggravation of a Table injury with the three *Althen* factors for off table injury claims to create a six-part test for off-Table aggravation claims); *see also W.C. v. Sec'y of Health & Human Servs.*, 704 F.3d 1352, 1357 (Fed. Cir. 2013) (applying the six-part *Loving* test.). The additional *Loving* factors require petitioners to demonstrate aggravation by showing: (1) the vaccinee's condition prior to the administration of the vaccine, (2) the vaccinee's current condition, and (3) whether the vaccinee's current condition constitutes a "significant aggravation" of the condition prior to the vaccination. *Id*.

V. Discussion

As explained above, although petitioner suggests that her sensory variant GBS was initially caused by her 2011 flu vaccination, her operative claim under her amended petition is that her later 2014 flu vaccination significantly aggravated that condition. Accordingly, this case turns on the six-part *Loving* test for determining whether a petitioner has experienced a vaccine-caused significant aggravation of a pre-existing

condition. Each of those six factors are discussed below. Counterintuitively, although petitioner's claim is for damages related to the 2014 significant aggravation of her sensory variant GBS, this case has focused heavily on whether the prior initial onset of petitioner's condition could be temporally related to her earlier 2011 vaccination.

Petitioner suggests that she suffered an initial bout of sensory variant GBS in the autumn of 2011 after receiving a flu vaccine; however, the condition was not diagnosed at that time and it was not until she experienced her alleged significant aggravation of the condition in 2014 that she claims she was able to piece her history together and ultimately receive a diagnosis of sensory variant GBS from Dr. Khoshnoodi that could explain her entire history back to 2011. Absent a diagnosis of sensory variant GBS petitioner has not otherwise explained how her 2014 flu vaccine could have affected her condition. Importantly, however, respondent disputes that petitioner ever suffered sensory variant GBS and both Dr. Khoshnoodi and Dr. Engstrand considered a temporal relationship between petitioner's 2011 vaccination and onset of her condition as a key diagnostic consideration in their assessments. Thus, *Loving* prong one, addressing petitioner's pre-vaccination condition, garners the most discussion below.

Ultimately, although petitioner has demonstrated that the flu vaccine can in general cause a significant aggravation of sensory variant GBS, she has not demonstrated that she ever suffered sensory variant GBS or that the actual condition she suffered meaningfully worsened following her second flu vaccination in 2014. Thus, she did not present a logical sequence of cause and effect demonstrating those symptoms to be vaccine-caused.

a. *Loving* Prong One

The first analysis required by the *Loving* test is an assessment of the vaccinee's condition prior to the administration of the vaccine. Additionally, because petitioner bears the burden of proving the factual circumstances underlying her claim, this also presents a threshold question of whether petitioner ever suffered the condition underlying her claim, namely sensory variant GBS. *Lombardi v. Sec'y of Health & Human Servs.*, 656 F.3d 1343, 1352 (Fed. Cir. 2011) (explaining that "if the existence and nature of the injury itself is in dispute, it is the special master's duty to first determine which injury was best supported by the evidence presented in the record before applying the *Althen* test to determine causation of that injury."); *Broekelschen v. Sec'y of Health & Human Servs.*, 618 F.3d 1339, 1346 (Fed. Cir. 2010).

First, I explain the diagnostic considerations presented by the parties' experts. Second, I assess the medical record evidence relating to those factors. And then, finally, in light of that discussion, I address the reliability of the purported sensory variant GBS diagnosis presented by Drs. Engstrand and Khoshnoodi. For the reasons discussed below, the evidence does not preponderate in favor of a finding that petitioner suffered sensory variant GBS at any time prior to her 2014 influenza vaccination.

i. Diagnostic considerations

GBS, also known as acute inflammatory demyelinating polyradiculoneuropathy ("AIDP"), generally consists of an ascending motor paresis peaking within four weeks. (Shin J. Oh et al., *Sensory Guillain-Barre Syndrome*, 56 NEUROLOGY 82 (2001) (Ex. 39).) It can have a variable presentation and, in fact, several specific variants have been identified. (*Id*.; *see also* Ex. C, p. 1.) Over the years different diagnostic criteria have been proposed for GBS; however, in this case, both experts agree that the so-called Brighton Criteria are generally authoritative. (Ex. 29, p. 5; Tr. 62, 86-90; Ex. C, pp. 2-3; Tr. 409-10.) GBS is generally diagnosed based on clinical observation, with ancillary testing of cerebral spinal fluid and nerve conduction or EMG studies. (Christiaan Fokke et al., *Diagnosis of Guillain-Barre syndrome and validation of Brighton criteria*, 137 BRAIN 33 (2014) (Ex. C, Tab 1, p. 2).) The Brighton Criteria provides four levels of diagnostic certainty based on seven diagnostic considerations, including: bilateral flaccid weakness of the limbs, decreased or absent deep tendon reflexes, a monophasic course with nadir occurring within four weeks of onset, elevated protein in cerebral spinal fluid, nerve conduction findings, and the absence of an alternative diagnosis. (*Id*. (Table 1).)

Although sensory variant GBS cannot be distinguished from the more classic AIDP etiologically, the experts agree that it is characterized by a distinct presentation that favors sensory symptoms over motor symptoms. (Tr. 12-13, 410-16, 498-500.) In that regard Dr. Chaudhry stressed that the Brighton Criteria does not address the sensory variant of GBS, which is incredibly rare. (Tr. 409-10.) Nonetheless, he opined that the core diagnostic requirements of GBS remain relevant to sensory variant GBS with the exception of the flaccid paralysis or weakness that is required by the Brighton Criteria. (Tr. 409-16.) Thus, for example, a 1981 diagnostic criteria for sensory variant GBS by Asbury required sensory loss and areflexia with (1) rapid onset, (2) widespread and symmetric onset, (3) complete or near complete recovery, (4) elevated CSF protein, and (5) electrodiagnostic studies characteristic of a demyelinating process in the peripheral nerve. (Oh et al., *supra* at Ex. 39, p. 4 (citing Arthur K Asbury, *Diagnostic considerations in Guillian-Barre syndrome*, 9 ANNALS OF NEUROLOGY S1 (1981).)

Petitioner filed an article by Oh, et al, that examined eight reported cases of sensory variant GBS. (Oh et al., *supra* at Ex. 39, p. 1.) Although this article was initially filed in this case in connection with Dr. Khoshnoodi's written opinion, during the hearing both Dr. Engstrand and Dr. Chaudhry spoke approvingly of the diagnostic considerations contained in that article. (Tr. 90-91, 221-24; Tr. 410-16.) Oh, et al, identified nine diagnostic considerations, eight of which were observed in all eight of their patients and one that was identified in only half. They are: (1) acute onset of symmetric loss; (2) peak deficit achieved within 4 weeks; (3) diminished or absent reflexes; (4) normal motor strength; (5) nerve conduction evidence of demyelination in at least two nerves; (6) monophasic course; (7) no other known cause for neuropathy; (8) no family history of neuropathy; and (9) elevated CSF protein during the acute phase of the disease (half of cases). (Oh et al., *supra* at Ex. 39, p. 1.) The Oh authors

explained that some, but not all, of their diagnostic factors overlap with the diagnostic factors previously established by Asbury.

Both experts agreed that it is not necessary for a patient to demonstrate all nine of the Oh factors to be diagnosed with sensory variant GBS. Dr. Engstrand was a little hard to pin down with respect to specifics. She repeatedly indicated that she uses published criteria for diagnosis, but is not "hard and fast" about it. (Tr. 89-94.) However, she did specifically endorse reliance on acute onset, a peak deficit within four weeks, normal motor strength (with caveat), and a monophasic course. She disagreed that diminished or absent reflexes are necessary. (*Id*. at 93-94) She indicated that, overall, six of the nine points should be present as a threshold for a diagnosis. (*Id*. at 94.) Dr. Chaudhry somewhat similarly opined that the bare minimum for diagnosis should be an acute symmetric loss (sensory), a peak deficit within four weeks, areflexia, and a monophasic course. (Tr. 418-20.)

A significant point of disagreement is Dr. Chaudhry's opinion that absent or diminished reflexes and normal motor strength (i.e. no weakness) are definitively required for diagnosis. The latter point is relevant especially to the parties' contentions regarding *Loving* prong two and is discussed separately in that context. With respect to absent or diminished reflexes, Dr. Chaudhry is persuasive in identifying this as a significant and informative diagnostic consideration even if it is not absolutely dispositive. Areflexia was a specific requirement under the Asbury criteria and absent or diminished reflexes were also identified as a key diagnostic factor present in all eight of the Oh subjects. (Oh et al., *supra* at Ex. 39, pp. 2, 4.) Additionally, Dr. Chaudhry explained that reflexes are controlled by the sensory nerves, making the reflexes highly relevant to a sensory presentation. (Tr. 404.) In that regard, the Fokke paper filed by respondent indicates that "[p]atients with initially normal reflexes also less frequently had sensory deficits (46%) compared to patients with decreased reflexes (69%). (Fokke et al, *supra* at Ex. C, Tab 1, p. 5.) In any event, although clearly of the opinion that intact reflexes do not defeat a GBS diagnosis, when asked to describe GBS, Dr. Engstrand herself indicated that GBS patients "usually get decreased reflexes." (Tr. 85.) Moreover, even without considering the finding essential, Dr. Engstrand also acknowledged that she looks at whether reflexes are intact when diagnosing GBS. (Tr. 86.)

ii. <u>Petitioner's medical records do not support any sensory variant GBS diagnosis</u>

1. Many of petitioner's symptoms, including symptoms of neuropathy, predated her vaccination by years according to the medical history she provided her treaters

Although not one of the nine diagnostic considerations discussed above, both Dr. Khoshnoodi and Dr. Engstrand premised their diagnostic opinions on the presence of a temporal relationship between the onset of petitioner's condition and her September 2011 flu vaccination. (Tr. 36, 126-27.) Thus, the first issue presented by petitioner's

medical records is that during an extended course of seeking diagnosis and treatment for her condition, petitioner consistently and repeatedly placed the initial onset of her symptoms, including symptoms of neuropathy, as occurring around 2006 rather than at any point in the weeks or months following her 2011 vaccination. Petitioner's attempt to explain this pattern of reporting and instead establish a different onset based on testimonial evidence is unpersuasive.

When petitioner first presented for a rheumatological evaluation with Dr. Wilson for the joint pain that she asserts was later diagnosed as sensory variant GBS, she did not initially describe any specific acute onset of symptoms. She did note that she started having problems in 2003 that she attributed to weight gain and that she began having MRIs to investigate beginning in 2006. (Ex. 3, p. 4.) Several months later, on June 6, 2012, petitioner sought orthopedic care for multiple joint/musculoskeletal pain on a self-referral basis. (Ex. 6, p. 5.) In describing onset of her condition, she stated broadly that she had not been feeling well since 2006. She noted numerous colds since then and also stressed having had a cough from November of 2011 to January of 2012. (*Id.*) During a follow up encounter on June 20, 2012, petitioner focused specifically on her right foot pain without revisiting onset. (*Id.* at 13.) However, because this was described as neuropathic pain, petitioner was referred for a neurology evaluation. (*Id.* at 14-15.)

Petitioner first sought an evaluation from a neurologist (Dr. Wallace) on July 3, 2012. (Ex. 4, p. 5.) At that time she did describe symptoms beginning in the autumn of 2011. Specifically, she indicated that "last fall she developed pain in multiple joints and a tingling, aching, burning feeling in her right leg below the knee." (*Id.*) However, when she returned for a follow up appointment with Dr. Wallace on August 14, 2012, Dr. Wallace recorded the following history as highlighted below:

Today, I saw Barbara Goforth in the office for neurologic evaluation. I have reviewed your office notes. I had seen Ms. Goforth on July 3rd on referral from the orthopedic office and indicated at that time that I thought she had a predominantly small fiber neuropathy. The basis for that was the sensory findings on her examination. Also, I was unable to obtain a sural or medial plantar sensory response. At that time, she indicated that she had been followed by Dr. Wilson and then evaluated at the orthopedic office by Rheumatology because of a high sedimentation rate. She reported that her other studies had been negative. Today, she tells me that she has been having symptoms since 2006 and she enumerated them in detail. She says that in 2006 she developed a pins-and-needles feeling and burning in her lower legs. Those symptoms continued in 2007 and she saw Dr. Kona, an orthopedist in Farmville, who did MRI of her hip and legs which were negative. In 2007, she developed urinary frequency and the feeling that she wasn't emptying her bladder. The above symptoms continued through 2008. In 2009, she had the swine flu but did not have any specific neurologic symptoms associated with it. In 2010, she said the pins-and-needles feeling in her legs was more severe, but it had never gone away completely. She says she had blurring of vision and rapid eye movements. When asked to further describe the rapid eye movements she said that she would have rapid twitching in her left eyelid. She also developed a choking feeling and says that when the choking feeling occurred she would feel as if there were a heavy weight on her chest. She also noted difficulty swallowing. Those symptoms have persisted to the present, are intermittent, and she can't identify anything that makes them develop or improve. In November 2011, she had viral like symptoms and the choking symptom and shortness of breath feeling got worse. She saw a pulmonologist without a specific diagnosis being made. However, the pulmonologist got a sedimentation rate and that was 45. Thus, she was referred to Dr. Wilson who tried her on steroids. Rheumatology at the orthopedic office is trying to get her off steroids and she is now down to 5 mg but says she feels worse when she tries to reduce her steroids. She says that recently she has been having what she describes as extreme blurriness of her vision. When asked to describe it further, she says that her right eye seems worse than the left. She then described vertical diplopia which is present in the right eye

(Ex. 4, p. 3.) Dr. Wallace's records include his handwritten timeline of these symptoms that he discussed with petitioner (Ex. 4, p. 12) as well as a checklist of symptoms completed by petitioner (Ex. 4, pp. 9-11).

Petitioner acknowledged having and reporting the symptoms identified in Dr. Wallace's list; however, she had different explanations for why each of these prior symptoms was unrelated to the condition for which she was seeking treatment at that time.[12] (Tr. 235-38.) During direct examination, petitioner indicated that this list of symptoms arose from the advice of her friend Wendy, who suggested that any time petitioner went to a neurologist, she should "write down every symptom you had ever experienced." (Tr. 232.) Petitioner implied that her list was of a broad and general nature and that it was wrong of Dr. Wallace to take the list as indicative of her current complaints. (Tr. 332-33, 338-39.) She suggested that Dr. Wallace's erroneous list was the source of later, similar histories. (Tr. 232-36, 341.)

Respondent's cross examination revealed, however, that petitioner had been frustrated that Dr. Wallace was dismissive of her concern at the time that her symptoms might be attributable to multiple sclerosis. (Tr. 336-37.) Petitioner clarified on cross-examination that " . . . Wendy had told me, you know, you need to write down every symptom you've ever had *that could be related to MS* . . . And I said, well, in this year, I experienced this. And in this year, I experienced this. And I believe that's where he came up with this list from." (Tr. 337 (emphasis added).) Thus, contrary to her direct testimony, petitioner ultimately confirmed that she was not listing for Dr. Wallace disparate symptoms she understood to be explained by other unrelated conditions, but instead was seeking to present to Dr. Wallace a list of otherwise unexplained symptoms that she feared could be unified to present an evolving picture of multiple sclerosis dating back to 2006. Her specific purpose was to try to convince Dr. Wallace that she actually suffered MS, a point which is also noted in his medical record. (Ex. 4, p. 4.) But, in any event, even before she was evaluated by Dr. Wallace, petitioner had already indicated to Dr. Wilson and N.P. Yesalis, albeit with less specificity, that her condition dated back to at least 2006.[13] (Ex. 4, p. 3; Ex. 6, p. 5.)

[12] Specifically, with regard to Dr. Wallace's handwritten timeline: Dr. Wallace listed "2006 pins and needles legs, also burning." (Ex. 4, p. 12.) Petitioner testified this referred to her plantar fasciitis even though it references her legs and her plantar fasciitis affected her feet. (Tr. 235-36). Dr. Wallace listed urinary frequency and "doesn't feel empty" in 2007, but petitioner attributed that only to working long uninterrupted hours in surgery. (Ex. 4, p. 12; Tr. 236.) (2008 was noted to be "same" and 2009 was illegible to petitioner.) (*Id.*) For 2010, Dr. Wallace listed "Pins and needles, blurred vision, rapid eye movement, choking feeling." (Ex 4, p. 12; Tr. 236.) This time, petitioner attributed the pins and needles to Morton's neuroma, which she described as a growth under the toes that causes nerve pain. (Tr. 233-37.) She attributed the blurred vision to astigmatism and the rapid eye movement to caffeine intake. (*Id.* at 237.) Petitioner attributed the choking feeling to having a narrow esophagus. (*Id.* at 237-38.)

[13] Additionally, petitioner's specific reinterpretation of Dr. Wallace's notation of numbness and tingling of the leg dating back to 2006 is also particularly unpersuasive due to inconsistencies in her testimony. Petitioner attributed Dr. Wallace's reference to pins and needles dating back to 2006 to her plantar fasciitis (Tr. 230, 235-36); however, petitioner acknowledged that her plantar fasciitis affected her feet while this notation specifically identifies the pins and needles as affecting her legs. Moreover, Dr. Wallace's record indicates petitioner discussed a prior evaluation and MRI by a Dr. Kona in the context of reporting this symptom. (Ex. 4, p. 12.) On further questioning, petitioner acknowledged that Dr. Kona

Dr. Wallace felt petitioner had peripheral sensory polyneuropathy, but noted that petitioner was concerned that she may have multiple sclerosis. He referred her to another neurologist (Dr. Jones) for a second opinion and further evaluation. (Ex. 4, pp. 4-6; Ex. 5, p. 4.) When petitioner saw Dr. Jones on September 19, 2012, she again described similar symptomology dating back to 2006. (Ex. 5, p. 4.) Specifically, Dr. Jones recorded that:

> Patient outlines her symptomatology since 2006, when her right foot started burning; she also developing muscle spasms and "cold" paresthesias in her right calf as well as urinary frequency (no response to Detrol LA). About that time, she started having RLE "charley horses." In 2007, she has continued RLE and was seen by an orthopod who reportedly did an MRI of her right hip and back that were normal; she was given Ultram, which reportedly was without benefit. In 2009, she started getting "shocks" in her arms, a choking sensation when she ate, especially with solids, and her LE paresthesias extended to involve her LLE; given this, she stopped working her 2nd job. In 2010, her vision became blurred like her "eyes her glazed over" while her other symptoms continued. She also noticed that all of her symptoms were worse in the heat. In 2011, she "could see her eye jumping" (horizontally), and her vision became even blurrier. In March of 2012, she woke up one morning and had not control of her LE; she now has ambulatory dysfunction, including one fall while at work in the OR. In May, she had three weeks of numbness in her tongue, and she felt a band of stabbing band around her lower chest; reportedly, the ultrasound was negative but her CT showed intussception (for which she had an exploratory laparotomy). She also describes diplopia (horizontal and MONOCULAR in her right eye), "short-circuity" shocks in her LE, hyper-awareness of (and inappropriate pain with) sensory stimuli (hyperalgesia / allodynia), occasional "jerks in her hands"

(*Id.*)

Petitioner asserts that any of the subsequent histories that match or are similar to the timeline created by Dr. Wallace must represent mere carrying over of his list. (Tr. 232-36, 341.) However, this rationale is directly refuted by Dr. Jones' record. Dr. Jones explained "I actually have some of these neurology notes, which enumerated the aforementioned symptoms in detail; as some of the verbiage is very similar to what she says today, I suspect that she was reading the list of symptoms from the same notebook she brings with her today." (Ex. 5, p. 4.) Thus, Dr. Jones confirmed he was aware of the prior neurology record, but also that he separately discussed the same history with petitioner in person.

In January of 2014, petitioner self-referred to a third neurologist (Dr. Rodden). (Ex. 12, pp. 6-10.) Her chief complaint was "possible demyelinating disease" and her history was recorded as follows:

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treated her for suspected lower back issues and found evidence of osteophytes upon MRI. (Tr. 373.) Additionally, according to her witness, Dr. Aslanis, petitioner reported in 2011 having had plantar fasciitis dating back only to 2009 (Ex. 24, p. 1.) Conversely, petitioner sought to explain Dr. Wallace's reference to pins and needles in the legs in 2010 to a neuroma rather than plantar fasciitis. (Tr. 236-37.) Moreover, when petitioner first presented to Dr. Rodden in January of 2013, she reported that plantar fasciitis had been a previously suspected cause of her right foot pain that was not borne out by her subsequent history (Ex. 12, p. 6.)

Barbara Goforth is a 41 y.o. female who I am asked to see in consultation for possible demyelinating disease. In 2005 she had burning pain in her right foot and progressed up her knee. She said she felt like her leg felt wet but also burning. She also thought she might have plantar fascitis. This continued until 2007. Then it progressed to back pain. She had and MRI of her L spine with just osetophytes. She was put on pain meds but didn't like it so stopped them. She then developed pins and needles in her wrists. She also started to get this sensation in her chest, etc.

In 2009 she caught the swine flu while out of the country. 2011 she go chicken pox. She had a viral illness for several months until this past February. She was seen by rheum and treated for possible Lupus. She was put on plauqenil and prednisone and it didn't help.

(Ex. 12, p. 6.)

Subsequently, on November 19, 2014, petitioner filed the instant action. (ECF No. 1.) A day later, on November 20, 2014, when petitioner saw N.P. Burton (in Dr. Rodden's office) to report symptoms following her November 2014 flu vaccination, she reported for the first time that "[t]his started in 2011 following a flu shot . . ." (Ex. 12, p. 79.) At that time she reported a history as highlighted below:

evaluated for small fiber neuropathy or CIDP with biopsy. Patient states numbness, tingling and vibration starting in feet an moving up legs with also in hands and legs. This has been going going on since October 2011 with varying progression and symptoms of neuropathic symptoms, weakness of legs and difficulty walking. This started in 2011 following a flu shot with improvement of symptoms of numbness, and gait impairment with eventual improvement after 8-9 months with waling and gait but not neuropathic symptoms and then again when she got flu shot this year 6 weeks ago and is unsure if there is any relation to the flu shot and symptoms but her progression of symptoms started with this. Having SOB and coughing - feels like cant breath. Fell Sunday after long drive requiring assistance to get out of car and then when inside due to leg numbness. Feels like legs are jello. Muscle spasms - takes valium and Zanaflex which helps spasms but not weakness or "jello" feeling. She has to use arms to move legs. Extreme back pain - feels like going to break in half. Discussed at length that do not believe this to be a MS flare or exacerbation based on unchanged MRI and generalized presentation. Unsure of the relationship to the Flu shot at this time. Patient is extremely upset and worried and fearful that this will progress to her not being able to walk like last time in 2011-2012. She

(*Id.*)

Thereafter, petitioner repeated essentially the same revised history to both Dr. Mayr (her fourth neurologist) and Dr. Khoshnoodi (her fifth neurologist). (Ex. 48, pp. 1-2; Ex. 21, p. 1.) On March 23, 2015, petitioner reported to Dr. Khoshnoodi that she "was at her usual state of health until about 2011, working as a surgical nurse [assistant]. At that time, she received a flu shot and developed flu symptoms a few days later. Along with flu symptoms, she developed numbness and paresthesia all over her body . . ." (Ex. 21, p. 1.) At this initial visit, Dr. Khoshnoodi recorded that "[s]he is convinced that everything is caused by the flu shot and want to provide it so she can get compensated for it." (*Id.* at 1-2.) Based on the history provided, Dr. Khoshnoodi included sensory variant GBS caused by petitioner's 2011 flu vaccine in petitioner's differential diagnosis for the first time. (*Id.* at 4.) However, upon review of the complete medical records, Dr. Chaudhry opined for respondent that the above-discussed history provided by petitioner during her prior medical encounters demonstrates onset of neuropathic symptoms dating back to the 2005-2007 timeframe with no evidence of neuropathic changes happening near vaccination. (Tr. 456-57.) In contrast, Dr. Engstrand did not substantively address petitioner's repeated reports of symptoms predating her 2011 vaccination.

Based on the record as a whole, there is preponderant evidence that petitioner had relevant symptoms indicative of a pre-existing neuropathy predating her 2011 flu vaccination and with onset as early as 2006. Medical records generally constitute trustworthy evidence in that "[t]he records contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium."[14] *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). Moreover, it was not until three years after her 2011 vaccination that petitioner ever attributed any worsening of her condition to that vaccination despite receiving extensive care in the interim. Her later reports to N.P. Burton, Dr. Mayr, and Dr. Khoshnoodi, contradict her earlier records in reporting that she was asymptomatic prior to the autumn of 2011. *See e.g.*, *R.K. v. Sec'y of Health & Human Servs*., No. 03-632V, 2015 WL 10936124, at *76 (Fed. Cl. Spec. Mstr. Sept. 28, 2015) (holding that more remote histories of illness do not have sufficient indicia of reliability to be credited over conflicting contemporaneous medical records and earlier reported histories), *mot. rev. denied* 125 Fed Cl. 57 (2016), *aff'd* 671 Fed. Appx. 792 (Fed. Cir. 2016); *see also e.g.*, *Vergara v. Sec'y of Health & Human Servs*., 08-882V, 2014 WL 2795491, *4 (Fed. Cl. Spec. Mstr May 15, 2014) ("Special Masters frequently accord more weight to contemporaneously-recorded medical symptoms than those *recorded in later medical histories*, affidavits, or trial testimony" (emphasis added).).

To the extent any of petitioner's earlier records from 2012 suggest that she suffered worsening symptoms beginning in the autumn of 2011, those records place those symptoms in the context of her respiratory illness occurring around that time. (*Compare* Ex. 4, p. 5 (reporting without specificity symptoms beginning "last fall"); Ex. 4, p. 3 (noting "viral-like" symptoms in November 2011) and Ex. 6, p. 5 (earlier associating problems to numerous coughs since 2006 and noting a cough and laryngitis beginning November 2011).) However, her contemporaneous medical records place that illness at the beginning of December of 2011 at the earliest. Petitioner presented for care for acute sinusitis on December 6, 2011, and at that time noted onset to have been three days prior, though she denied any cough. (Ex. 18, p. 12.) When she presented for follow up care with Dr. Donker on February 20, 2012, she complained of a cough for "several weeks" and laryngitis for one week. (Ex. 2, p. 6.)

Moreover, contrary to her own recollection that she was too unwell to trick-or-treat in October of 2011 (Tr. 241-44), Dr. Aslanis recalled that petitioner did not struggle

[14] Conversely, it must also be noted that there is no presumption that medical records are accurate or complete as to all of a patient's conditions. *Kirby v. Sec'y of Health & Human Servs.*, 997 F.3d 1378, 1382-83 (Fed. Cir. 2021). Afterall, "[m]edical records are only as accurate as the person providing the information." *Parcells v. Sec'y of Health & Human Servs*., No. 03-1192V, 2006 WL 2252749, at *2 (Fed. Cl. Spec. Mstr. July 18, 2006). Importantly, however, "the absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance." *Murphy v. Sec'y of Health & Human Servs*., 23 Cl. Ct. 726, 733 (1991) (quoting the decision below), *aff'd per curiam,* 968 F.2d 1226 (Fed. Cir. 1992). Here, petitioner's records contain affirmative descriptions of symptom onset conflicting with her claim.

at work until around or after Thanksgiving of 2011. (Tr. 151.) She also recalled that petitioner's difficulty with her job, and the need to "break scrub," coincided with onset of her cough.[15] (Tr. 152.) Although witness testimony may be offered to overcome the weight afforded to contemporaneous medical records, such evidence must be "consistent, clear, cogent, and compelling." *Camery v. Sec'y of Health & Human Servs.*, 42 Fed. Cl. 381, 391 (1998) (citing *Blutstein v. Sec'y of Health & Human Servs.*, No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)).

It is also worth noting more generally that petitioner's explanation for why the medical records are inaccurate is largely premised on her ability to confidently attribute many of her symptoms to specific conditions. However, the record refutes any such confidence. Although she works in a medical setting, petitioner acknowledged in testimony that she does not interpret test results or diagnose conditions, does not work regularly with GBS patients, and does not have any professional knowledge of neuromuscular disease. (Tr. 324-27.) She also acknowledged that her own condition was not consistent with what she had learned about GBS in nursing school. (Tr. 343-44.) Moreover, prior to her 2014 vaccination, petitioner clearly underwent a multi-year search for a diagnosis to no avail. At turns rheumatic, orthopedic, and neurologic conditions were evaluated as explanations for her presentation. Petitioner's stated ability to distinguish the causes of her different symptoms is belied by her own treating physicians' inability to do so. Moreover, petitioner's fact witness, Ms. Brooks, specifically testified that petitioner sought guidance from her because she did not know what could explain her symptoms. (Tr. 281-83.) She explained that petitioner often called her with questions about various neurological diseases. (Tr. 282.) They also initially considered the possibility petitioner's presentation was due to a back injury. (Tr. 282-83.)

2. Petitioner's post-vaccination clinical course was not consistent with GBS

The second issue presented by petitioner's medical records is that during the period following her 2011 vaccination, her presenting symptoms and physical examinations were not consistent with sensory variant GBS.

The first medical record post-dating petitioner's September 2011 flu vaccination is a rheumatology referral by Dr. Donker dated November 3, 2011. (Ex. 2, p. 3.) The referral indicated petitioner's complaint was "pain at multiple sites – unknown cause." (*Id.*) Petitioner was not able to see the rheumatologist (Dr. Wilson) until March 14,

[15] Notably, if I were to accept Dr. Aslanis's testimony in connection with the December 6, 2011, and February 20, 2012, medical records as providing preponderant evidence that petitioner experienced a post-vaccination onset of numbness and tingling after Thanksgiving and in connection with her cough, then that would raise a question as to whether her initial claim should be considered as timely. Importantly, however, such a conclusion would place onset of petitioner's symptoms beyond what Dr. Engstrand considered an appropriate temporal relationship to her earlier vaccination. (Tr. 76.) Moreover, for all the reasons discussed throughout this decision, petitioner is not persuasive in contending that her condition is explained as sensory variant GBS or that it was caused or aggravated by either of her vaccinations. Accordingly, there is no reason to revisit the timeliness of her original claim.

2012. (Ex. 27, p. 9.) At that time, petitioner complained of joint pain, redness, swelling and stiffness in numerous joints. She also described fatigue and weakness in her hands. Petitioner described her symptoms as "worsening." (*Id*.) Dr. Wilson's Review of Systems indicated that petitioner was experiencing numbness and tingling; however, his physical exam revealed symmetrical reflexes were present along with normal sensation. (*Id*. at 10-11.) She also had normal gait, normal Romberg test (measures balance), and normal strength. (*Id*. at 11.) The assessment was polyarthralgia, but with a wide differential diagnosis. (*Id*. at 12.)

As petitioner's first treatment record for what she now asserts is sensory variant GBS, this record is notable for being significantly at odds with the above-discussed diagnostic factors for sensory variant GBS. Whereas the diagnostic criteria look for acute onset of symmetric loss and a peak deficit within four weeks, petitioner did not describe any acute onset of her complaints, still had normal sensation on physical exam, and described her symptoms as still worsening five months after the alleged onset of her condition. She also had intact reflexes. Thus, Dr. Chaudhry persuasively opined that petitioner's March 15, 2012 encounter was not consistent with either GBS or residual effects of GBS. (Tr. 407-08.) Although Dr. Engstrand stressed the presence of numbness and tingling, she agreed that petitioner's neurologic exam was otherwise normal at this encounter. (Tr. 171-73.) Dr. Engstrand also agreed that petitioner's prior medical encounters of December 6, 2011, and February 20, 2012, included no indication petitioner had any neurologic symptoms. (Tr. 167-71 (discussing Ex. 18, p. 12, Ex. 2, pp. 4, 6-7).) During the hearing, Dr. Khoshnoodi, the treating physician who introduced the suggestion of sensory variant GBS into petitioner's later medical history, confirmed that he never reviewed any of petitioner's medical records except for those of Dr. Rodden and, thus, would not have known about this encounter or been aware of these exam findings. (Tr. 124-25.)

Petitioner stressed in her testimony that N.P. Yesalis's later June 20, 2012 prescription of gabapentin caused her to understand for the first time that she had been experiencing nerve pain rather than joint pain. (Tr. 304-06, 309.) She indicated that she had never experienced nerve pain before and therefore was not able to distinguish it from joint or muscle pain. (Tr. 309.) For this reason, petitioner asserts that notations of joint pain or arthralgia in her earlier record should be reinterpreted in hindsight as reports of nerve pain. (*Id*.) This is unpersuasive for several reasons. First, petitioner was inconsistent on this point. As noted above, in seeking to explain Dr. Wallace's timeline of symptoms, she asserted that she had, in fact, previously experienced nerve pain in 2010 that she attributed to a neuroma. (Tr. 233-37.) Moreover, petitioner is recorded by N.P. Yesalis as self-describing her right foot pain as neuropathic pain at the same encounter during which petitioner was first prescribed gabapentin. (Ex. 6, p. 14.) This contradicts her stated rationale for being unable to distinguish her complaints. Additionally, Dr. Chaudhry explained that gabapentin is used for many pain syndromes, including both neuropathies and polyarthralgia, so petitioner's new understanding of her pain is based on a questionable premise. (Tr. 426-27.) Finally, the report of joint pain was not limited to pain, but was also accompanied by reports of joint stiffness, swelling, and redness. (Ex. 27, p. 9.)

Petitioner first sought an evaluation from a neurologist (Dr. Wallace) on July 3, 2012. (Ex. 4, p. 5.) Petitioner described her symptoms as "fluctuating." (*Id.*) Dr. Wallace observed that petitioner had normal strength and tone on physical examination. Deep tendon reflexes were normal and symmetrical. Vibratory sensation was mildly diminished on her toes and she had decreased sensation to pinprick in a stocking distribution. (*Id.*) Dr. Wallace diagnosed an asymmetric[16] sensory peripheral neuropathy, possibly due to an unknown underlying inflammatory illness. (*Id.*)

Dr. Wallace's July 3, 2012 record is again inconsistent with much of the diagnostic criteria for sensory variant GBS. Although petitioner did describe onset occurring in the autumn of 2011, this record indicates, four months after her encounter with Dr. Wilson, that her course had been fluctuating and confirms that her condition continued to progress (Dr. Wallace identified sensory loss whereas Dr. Wilson had previously observed normal sensation), suggesting again that it did not peak within four weeks and was not monophasic. She still had normal and symmetrical reflexes and those sensory symptoms that she did have were asymmetric. Dr. Engstrand acknowledged that the fluctuating and intermittent symptoms described by petitioner at this visit are not consistent with GBS. (Tr. 185-89.) She also acknowledged the presentation to be asymmetric and with intact reflexes. (*Id.*)

According to petitioner, her symptoms attributable to sensory variant GBS would have been resolving after about this point. (Tr. 254-54.) Dr. Chaudhry also stressed, however, that petitioner's condition continued to evolve after she began seeking treatment and beyond when she suggests her symptoms resolved. Her medical records reflect a sudden inability to move her legs beginning in March of 2012 and onset of tongue numbness in May of 2012. (Tr. 449-52, 467-68.) Additionally, Dr. Jones first recorded reduced reflexes in December of 2012 (Ex. 5, p. 9) and Dr. Rodden first recorded right sided weakness in January of 2014 (Ex. 12, p. 9). In addition to weakness being inconsistent with the sensory variant of GBS, Dr. Chaudhry also indicated that this asymmetric weakness would not be consistent with the symmetric presentation of GBS more generally. (Tr. 429-30.) According to Dr. Chaudhry, onset of diminished reflexes as of December 2012 and new weakness in January of 2014, two years into the alleged course of the condition, are not consistent with a monophasic course of GBS. (Tr. 405, 421-22, 464-65.) Additionally, he observed that petitioner's presentation included some indicators of central nervous system involvement, including oligoclonal bands in her spinal fluid and sub-optic neuritis. (Tr. 408-09.) Petitioner also had an MRI study performed on August 7, 2012, which showed white matter signals in the brain that were nonspecific but compatible with multiple sclerosis. (Ex. 16, pp. 31-32.)

[16] Only petitioner's right lower extremity was tested by NCS and EMG; however, Dr. Wallace also stressed that petitioner's symptoms were asymmetric. (Ex. 4, p. 5.)

3. Petitioner's NCS/EMG results and oligoclonal bands were not consistent with GBS

The third issue presented by petitioner's medical history is the lack of any available confirmatory findings upon relevant diagnostic testing. Among the nine diagnostic criteria discussed above, two assess specific tests that are available to aid in diagnosis of GBS, namely nerve conduction and EMG studies and cerebral spinal fluid testing. In petitioner's case, neither demonstrated any evidence that petitioner suffered GBS.

With regard to cerebral spinal fluid testing, petitioner underwent a spinal tap on January 8, 2013. (Ex. 5, pp. 23-29.) It showed no indication of elevated protein. However, all agree that because this test was performed beyond what petitioner alleges to have been the acute phase of petitioner's condition, that negative result is not diagnostically useful. (Tr. 47-48, 125-26, 404, 536-38.) Nonetheless, Dr. Engstrand contends that the finding of six oligoclonal bands is diagnostically significant for GBS. (Tr. 44-45, 190-92.) Dr. Chaudhry persuasively explained, however, that while oligoclonal bands found in serum can be diagnostically significant for GBS, oligoclonal bands found in spinal fluid are not. Oligoclonal bands in spinal fluid point to inflammation of the central nervous system rather than the peripheral nerves affected by GBS. (Tr. 408.) For his part, Dr. Khoshnoodi, the treating physician who included sensory variant GBS in his differential diagnosis, likewise indicated that petitioner's oligoclonal bands were not diagnostic of GBS. (Tr.145-46.)

Additionally, in the course of her medical history following the 2011 vaccination, petitioner has undergone four separate nerve conduction and EMG studies: July 3, 2012 (Ex. 4, p. 7), January 13, 2015 (Ex. 12, pp. 140-45), March 24, 2015 (Ex. 21, p. 6), and March 3, 2016 (Ex. 37, pp. 20-21). According to Dr. Chaudhry, while these studies showed evidence of sensory peripheral neuropathy, they do not show evidence of demyelination or reveal the cause of the neuropathy. (Ex. C, p. 2; Tr. 435-46.) Focusing on petitioner's first, July 3, 2012, NCS/EMG, Dr. Chaudhry explained that demyelination is demonstrated on nerve conduction by measuring latency and calculating velocity, because it is the myelin that determines how fast nerve impulses travel. (Tr. 435-39.) According to Dr. Wallace's study results, petitioner's reported velocities on July 3, 2012, were 49 meters per second for the peroneal nerve and 46 meters per second for the tibial nerve against references ranges of 44-57 meter/sec and 41-58 meter/sec respectively, both within normal range. (Ex. 4, p. 7.) Accordingly, they showed no slowing. (Tr. 435-439, 443, 507-08.)

Dr. Engstrand stressed two later notations that suggested Dr. Wallace's study demonstrated slowed latency of the peroneal nerve, which she opined is consistent with GBS. (Tr. 15-25 (discussing Ex. 37, p. 31 (April 21, 2016 record of Dr. Cochran discussing NCS/EMG conducted by Dr. Wallace); and Ex. 36, p. 71 (July 8, 2016 record of Dr. Phillips discussing NCS/EMG conducted by Dr. Wallace)).) Importantly, however, Dr. Engstrand did not herself interpret or raise any specific reference to the underlying data, relying instead only on notations themselves. Review of Dr. Wallace's actual

results and references ranges, coupled with Dr. Chaudhry's explanation, shows Drs. Cochran's and Phillips's statements to be incorrect. Significantly, both notations appear as representations by petitioner in the history of present illness section of the medical records. Moreover, both records indicate the physicians did not review the results themselves. Dr. Cochran explicitly stated so. (Ex. 37, p. 31.) Dr. Phillips indicated he reviewed outside studies, but identified only petitioner's later January 2015 NCS/EMG and noted it to be consistent with sensory neuronopathy. (Ex. 36, p. 73.)

Dr. Engstrand also explained that two of petitioner's subsequent NCS/EMG studies showed prolonged latency of the median nerve of the upper extremity. (Tr. 15-25 (discussing Ex. 12, p. 141 (January 13, 2015 NCS/EMG); Ex. 37, p. 18 (March 3, 2016 record of Dr. Lim); Ex. 37, pp. 20-21 (March 3, 2016 NCS/EMG)).) Dr. Chaudhry explained, however, that the prolonged latency of the median nerve demonstrated on the January 13, 2015, and March 3, 2016, studies cited by Dr. Engstrand represent focal demyelination that is explained by carpal tunnel syndrome. (Tr. 440-46.) This is consistent with how these studies were interpreted by Dr. Lim at the time. (Ex. 37, p. 21.) Moreover, petitioner had a documented history of carpal tunnel syndrome. (Tr. 484-85.) Additionally, Dr. Chaudhry stressed that Dr. Sohail specifically indicated in evaluating the earlier of these two studies that these results included nothing to suggest CIDP, which necessarily indicates a lack of demyelination apart from the separately discussed carpal tunnel syndrome. (Tr. 440-43 (discussing Ex. 12, p. 140-41).)

On the whole, Dr. Engstrand's opinion and testimony regarding NCS/EMG was less persuasive than Dr. Chaudhry's. Dr. Engstrand's opinion was rarely more specific than to assert that any evidence of slowing constitutes evidence of demyelination, a point disputed by Dr. Chaudhry and not fully supported by the literature filed in this case. (Tr. 437-39 (*see also* Oh et al., *supra* at Ex. 39, p. 3 (explaining that "[t]hough a slow NCV was observed in posterior tibial and peroneal nerves in five patients and in median and ulnar nerves in three patients, evidence of demyelination was observed in only two patients.") Dr. Chaudhry, however, provided significantly more explanation for his opinion. Moreover, whereas Dr. Engstrand was accepted without objection as an expert in neurology, Dr. Chaudhry was accepted without objection as expert in neuromuscular disease and electrodiagnostic medicine as well as neurology. (Tr. 11, 399.)

The remaining question is whether the absence of sensory responses demonstrated by petitioner's NCS/EMG studies is otherwise diagnostic of sensory variant GBS. Notably, although Dr. Khoshnoodi never opined that petitioner's NCS/EMG demonstrated demyelination, he nonetheless opined that petitioner's absent sensory responses upon NCS were diagnostically relevant. In his earlier letter to the special master, Dr. Khoshnoodi indicated that petitioner's NCS/EMG was indicative of sensory axonal neuropathy. (Ex. 22; Ex. 37.) Dr. Khoshnoodi offered two rationales for accepting petitioner's NCS/EMG findings as suggestive of sensory variant GBS. First, he indicated that these findings objectively confirm petitioner's subjective sensory loss complaints and permanent nerve damage. He further and relatedly indicated that absent sensory signals with intact motor signals are compatible with sensory variant

GBS.[17] (Tr. 125-26, 136.) Second, Dr. Khoshnoodi suggested that, because petitioner's NCS/EMGs were performed later, it is possible they were too late to detect demyelinating features that may have been present earlier. (Tr. 121.)

Classically, under the Brighton Criteria, NCS findings consistent with one of the subtypes of GBS is required to achieve only the highest level of diagnostic certainty (Fokke et al, *supra* at Ex. C, Tab 1, p. 2 (Table 1).) In that context, it has been observed that "[i]n current clinical practice the value of subtyping by nerve electrophysiology is uncertain" and "there are no definite agreed-upon diagnostic electrophysiological criteria for the diagnosis of [GBS]." (*Id*. at 9-10.) As noted above, however, Dr. Chaudhry explained that the Brighton Criteria do not address sensory variant GBS. (Tr. 409-10.) With specific regard to sensory variant GBS, the literature of record in this case explains that the presence of demyelination has a specific diagnostic role in identifying the condition. Oh, et al, explains that the prior Asbury criteria for sensory variant GBS *required* electrodiagnostic evidence of demyelination of the peripheral nerve. (Oh et al., *supra* at Ex. 39, p. 4.) They also observed that all eight of their study subjects demonstrated demyelination in at least two nerves. (*Id*.)

Dr. Khoshnoodi is correct that in some cases Oh et al., observed no demyelination in the motor nerves and also observed no sensory nerve conduction potential in half the cases; however, the presence of some evidence of demyelination, either sensory, motor, or mixed, remained an important diagnostic consideration present in all eight cases. (Oh et al., *supra* at Ex. 39, p. 3.) The authors explained that it is the presence of demyelination that allows sensory variant GBS to be distinguished from acute sensory neuronopathy, which is a distinct condition affecting the dorsal root ganglia rather than the peripheral nerves. (*Id*. at 4) In contrast to his testimony, Dr. Khoshnoodi included sensory neuronopathy in his differential diagnosis within his medical record and recommended an MRI to investigate whether an inflammatory process was affecting petitioner's dorsal root ganglion, which was never done. (Ex. 21, pp. 4-5.) Dr. Chaudhry likewise endorsed that recommendation. (Tr. 432, 446.) During the hearing, Dr. Khoshnoodi ultimately acknowledged that petitioner's EMG/NCS results, taken in isolation, would implicate a broad differential diagnosis including several different neuropathies. (Tr. 134.) Consistent with Dr. Chaudhry's opinion, he also agreed that neuropathy, sensory neuropathy, and peripheral neuropathy, are viable diagnoses following a NCS like petitioner's. (Tr. 134-35.) Dr. Khoshnoodi also ultimately acknowledged that there is no way of knowing whether these abnormalities were present prior to July of 2012. (Tr. 144.) Accordingly, his suggestion that demyelination could have been present earlier is speculative.

[17] Following on from Dr. Khoshnoodi's opinion, Dr. Engstrand equated a loss of sensory response with, in effect, very severe slowing, characterizing the finding as being "more than slow," implying that the absent sensory responses are further evidence of demyelination. (Tr. 189.) That is not an accurate description of Dr. Khoshnoodi's testimony. Dr. Khoshnoodi testified that "[w]e did not detect any sensory signals, so we could not really judge anything about them being slow or delayed, because they were absent." (Tr. 121.) Moreover, Dr. Engstrand's opinion on this point is unpersuasive for reasons discussed below. Peripheral demyelination is not the only pathological explanation for why sensory response may be absent.

iii. Dr. Khoshnoodi's and Dr. Engstrand's diagnostic opinions were not reliably reached

As explained in greater detail above, the nine diagnostic considerations for sensory variant GBS as considered by Oh, et al, are: (1) acute onset of symmetric loss; (2) peak deficit achieved within 4 weeks; (3) diminished or absent reflexes; (4) normal motor strength; (5) nerve conduction evidence of demyelination in at least two nerves; (6) monophasic course; (7) no other known cause for neuropathy; (8) no family history of neuropathy; and (9) elevated CSF protein during the acute phase of the disease (half of cases). (Oh et al., *supra* at Ex. 39, p. 1.) For the reasons discussed above, petitioner's medical history from 2011 through 2012 potentially implicates only points (4), (7), and (8). That is, although she developed signs of weakness beginning in 2014, she did have normal strength during 2012. She had neuropathy that was not otherwise explained and did not report any family history of neuropathy. Although she did later have diminished or absent reflexes, this was not recorded until December of 2012, more than a year after the alleged onset of her sensory variant GBS. This is far short of the six factors Dr. Engstrand suggested would be necessary to support a sensory variant GBS diagnosis. (Tr. 94.)

Moreover, as a treating and diagnosing physician, Dr. Khoshnoodi acknowledged sensory variant GBS is typically diagnosed by acute onset of symptoms and a peak deficit within four weeks. (Tr. 123.) There is also agreement among the experts in this case regarding the diagnostic importance of an acute onset, a peak deficit achieved within four weeks, and a monophasic course. (Tr. 89-94, 418-20.) However, none of these factors were demonstrated in this case following petitioner's 2011 vaccination. Additionally, Dr. Chaudhry was persuasive in stressing the diagnostic importance of symmetric loss and areflexia. (Tr. 418-20.) Neither of these factors was present. To the extent an extended or relapsing course of a GBS-like presentation could in some cases conceivably be explained as the chronic counterpart to GBS, CIDP or chronic inflammatory demyelinating polyneuropathy, Drs. Engstrand, Khoshnoodi, and Chaudhry all agreed that petitioner did not have CIDP. (Tr. 211-13, 443, 546; Ex. 38, p. 2)

Dr. Engstrand stressed symptoms such as fatigue, urinary frequency, headache, shortness of breath, blurred vision, dizziness, and swallowing difficulty. (Tr. 14-15.) Contrasting her view with Dr. Chaudhry's competing opinion, she indicated she is aware of no literature that would indicate these symptoms are not associated with GBS. (*Id.*) While it may be true that some or all of these symptoms could potentially be seen among GBS patients, that does not make them diagnostic of the condition. Nor are any of these symptoms unique to GBS. Without evidence supporting the core diagnostic considerations relevant to sensory variant GBS, reliance on these symptoms is not persuasive with regard to diagnosis.

Additionally, although not a part of the diagnostic criteria, a factor critical to both Drs. Engstrand's and Khoshnoodi's diagnostic opinions was the purported fact that

petitioner experienced onset of symptoms temporally related to her 2011 vaccination. (Tr. 36, 127.) In fact, Dr. Khoshnoodi testified that the timing is "very important really. Everything is about the timing of her symptoms in this diagnosis . . . that's really the key to this diagnosis." (Tr. 127.) He confirmed that if the timing of onset was different than what was reported to him, then his diagnostic opinion would change. (Tr. 127-28.) However, for all the reasons discussed above, Drs. Engstrand's and Khoshnoodi's assumptions regarding onset are incorrect.

When first asked about the history taken by Dr. Rodden, Dr. Khoshnoodi initially recalled that Dr. Rodden had recorded symptoms dating back to 2005 that may be attributable to MS. (Tr. 129.) However, he then acknowledged that he was unaware that petitioner had not reported a post-vaccination symptom onset to Dr. Rodden until more than three years after the fact. (Tr. 130.) He also indicated that he was unaware petitioner had provided a different history to Dr. Rodden at an earlier encounter, stating "[m]y understanding was that her symptoms had started in 2011." (*Id.*) Dr. Khoshnoodi confirmed that his assumption regarding an acute post-vaccination onset was based on petitioner's report and that he had no records from the purported acute phase of her illness. (Tr. 127.) In fact, the only prior records Dr. Khoshnoodi reviewed were those of Dr. Rodden. (Tr. 124-25.) Dr. Engstrand similarly indicated during testimony that she was not aware that petitioner reported symptoms dating back to 2005/2006 on multiple occasions. (Tr. 181, 184-85.) After reading Dr. Wallace's above-discussed August 14, 2012 timeline of symptoms into the record, Dr. Engstrand's only response was to indicate without elaboration that some unspecified symptoms were different than her later presentation. (Tr. 182-84.)

Accordingly, to the extent Drs. Engstrand and Khoshnoodi based their diagnostic opinions on false assumptions as to onset, they are unpersuasive. *Burns v. Sec'y of Health & Human Servs.*, 3 F. 3d 415 (Fed. Cir. 1993) (holding that "[t]he special master concluded that the expert based his opinion on facts not substantiated by the record. As a result, the special master properly rejected the testimony of petitioner's medical expert."); *see also Rickett v. Sec'y of Health & Human Servs.,* 468 Fed. Appx. 952, 958 (Fed. Cir. 2011) (holding that "it was not error for the Special Master to assign less weight to Dr. Bellanti's conclusion regarding challenge-rechallenge to the extent it hinged upon Mr. Rickett's testimony that was inconsistent with the medical records."); *Dobrydnev v. Sec'y of Health & Human Servs.,* 566 Fed. Appx. 976, 982–83 (Fed. Cir. 2014) (holding that the special master was correct in noting that "when an expert assumes facts that are not supported by a preponderance of the evidence, a finder of fact may properly reject the expert's opinion") (citing *Brooke Group Ltd. v. Brown & Williamson Tobacco Corp.*, 509 U.S. 209, 242 (1993)); *Bushnell v. Sec'y of Health & Human Servs.,* No. 02-1648V, 2015 WL 4099824, at *12 (Fed. Cl. Spec. Mstr. June 12, 2015) (finding that "because Dr. Marks' opinion is based on a false assumption regarding the onset of J.R.B.'s condition, and the incorrect assumption of a "stepwise regression" after each vaccine administration, it should not be credited.")

The Federal Circuit has recognized that "treating physicians are likely to be in the best position to determine whether 'a logical sequence of cause and effect show[s] that

the vaccination was the reason for the injury.'" *Capizzano v. Sec'y of Health & Human Servs.,* 440 F.3d 1317, 1326 (Fed. Cir. 2006) (quoting *Althen v. Sec'y of Health and Human Servs.,* 418 F.3d 1274, 1280 (Fed. Cir. 2005)). This logic has also been applied 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"), *mot. for rev. denied*, 122 Fed. Cl. 86 (2015), *aff'd*, 645 F. Appx. 1002 (Mem.) (Fed. Cir. 2016). However, the opinions of treating physicians are not binding on special masters. 42 U.S.C. 300aa§ 13(b)(1); *see also Snyder v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 745 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"). Rather, when considering medical opinion evidence special masters are required to determine whether it has a reliable basis. *Moberly v. Sec'y of Health & Human Servs.,* 592 F.3d 1315, 1326 (Fed. Cir. 2010) ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence.")

Additionally, the deference given to treating physician opinions is premised on their first-hand observation of a patient's condition. Here, Dr. Khoshnoodi was not a treating physician until nearly four years after her 2011 vaccination and acknowledged that he did not have access to the relevant contemporaneous medical records. *Nuttall v. Sec'y of Health & Human Servs*., 122 Fed. Cl. 821, 832 (2015) (indicating that "[t]he reasoning underlying the finding that opinions of treating physicians should be given particular weight does not apply when, as here, the treating physician only saw the patient after the injury and based his opinion on the same evidence as relied upon by the retained experts."), *aff'd* 640 Fed. Appx. 996 (Mem.) (Fed. Cir. 2016).

Although it is undisputed that petitioner has suffered some form of neurologic injury inclusive of a sensory polyneuropathy, considering the record as a whole there is not preponderant evidence that petitioner's condition can be diagnosed as sensory variant GBS. Accordingly, there is likewise not preponderant evidence that petitioner suffered any pre-existing sensory variant GBS prior to her November 2014 influenza vaccination.

b. *Loving* Prong Two

The second *Loving* prong addresses petitioner's current, or post-vaccination, condition. Petitioner received the vaccination at issue on November 2, 2014. (Ex. 26.)

During the hearing, petitioner indicated that following her 2014 vaccination she experienced worsened pins and needles sensation, weakness, and difficulty walking. (Tr. 249-51.) She stressed that she fell nine times subsequent to her 2014 vaccination. (*Id.*) She suggested she may have experienced falls subsequent to her 2011 vaccination, but stressed she experienced no falls during 2014. (*Id.*) She also indicated that after the 2014 vaccine she had worse difficulty with her swallowing and with dry

mouth.[18] (*Id.*) She additionally had significant back pain. (*Id.*) She reiterated that her 2011 symptoms resolved after about eight or nine months of her prior vaccination, but suggested that the symptoms "came back with a vengeance" after the 2014 vaccination. (Tr. 253-54.)

Petitioner stressed that she has continued to have residual effects including nerve pain, heat intolerance, and gastroparesis, but indicated that her 2014 symptoms reached a nadir after about the third week of November. (Tr. 254-56.) Similarly, Dr. Engstrand testified that petitioner's worst period was between two to six weeks post-vaccination, which coincides with what she identified as the relevant period for causally implicating the vaccination in petitioner's condition. (Tr. 71, 75.) Dr. Engstrand testified that petitioner's condition "didn't fluctuate that much" following this initial period, allowing her to opine that this constituted a second monophasic course consistent with diagnostic standards for GBS. (Tr. 70-72.) Similarly, Dr. Khoshnoodi, who first saw petitioner on March 24, 2015, confirmed that he saw petitioner only after the acute phase of her alleged sensory variant GBS. (Tr. 131.) During the six weeks following her November 2, 2014 vaccination, petitioner had only one medical encounter.

On November 20, 2014, petitioner sought follow up care for numbness and tingling in all extremities and weakness in her legs that was beginning to impair her gait. (Ex. 12, p. 79.) Petitioner reported that she had experienced similar symptoms following her 2011 flu vaccination, but they had resolved within eight or nine months of vaccination. (*Id.*) She attributed her recent symptoms to her 2014 vaccination. (*Id.*) Petitioner's physical examination on November 20, 2014, indicated normal tone, bulk, and strength in her upper extremities bilaterally, but with reduced strength of 4/5 in her bilateral lower extremities. She had light and sharp touch sensations in her bilateral upper extremities, but decreased temperature sensation in her right upper extremity. Sharp touch was present in her lower extremities bilaterally, but she had decreased light touch and absent temperature sensation bilaterally in the lower extremities. She had normal proprioception, but an abnormal gait with wide exaggerated steps. Deep tendon reflexes were 2+ in the upper extremities and 1+ in the lower extremities. (Ex. 12, p. 82.)

Dr. Engstrand opined that this appointment was significant for demonstrating bilateral leg weakness affecting gait, polyneuropathy, bilateral arm weakness, and back pain. She opined that the history of present illness is consistent with a temporal relationship to the November 2, 2014 vaccination. (Tr. 56-57.) On cross examination, Dr. Engstrand confirmed that her opinion is that petitioner's symptoms, especially her leg weakness and increased tingling and difficulty breathing, represented a worsening of symptoms. (Tr. 213-16.) Her opinion was based on her understanding that petitioner's condition had been improving prior to the 2014 vaccination, though she

[18] Additional symptoms, such as difficulty breathing, dry mouth, and potential autonomic dysfunction, were variously mentioned throughout the hearing, including by Drs. Khoshnoodi and Engstrand; however, none of these are within the core diagnostic considerations for GBS. Absent clearer evidence of GBS, these symptoms do not warrant extensive discussion. Petitioner has not linked any of these features to her alleged vaccine-injury other than in the context of a GBS diagnosis.

could not recall any medical record that supported that assumption. She opined that petitioner's weakness and falling following the 2014 vaccination showed that her condition had worsened. (Tr. 57-59, 217-18.)

Dr. Engstrand is not persuasive in opining that leg weakness is compatible with sensory variant GBS, at least not in this case. Initially, Dr. Engstrand noted that no weakness is required for a diagnosis of sensory variant GBS. (Tr. 53-54.) This is because, as both parties' experts agree, it is the predominance of sensory rather than motor loss that characterizes the sensory variant of GBS. (Tr. 13, 30, 409-10.) Thus, Dr. Chaudhry explained that the Brighton criteria requirement of ascending paralysis precludes direct application of the Brighton criteria to sensory variant GBS. (Tr. 409-10.) In that regard, Oh, et al, similarly explained that "[a]mong the classic diagnostic criteria of GBS, progressive motor weakness of more than one limb has been a feature. Thus, none of our cases meet these criteria." (Oh et al., *supra* at Ex. 39, p. 4.) Accordingly, Oh, et al, included "normal motor strength" among their diagnostic criteria. (*Id*. at 1.) Moreover, all eight of their validating test subjects demonstrated normal motor strength. (*Id*.)

Dr. Engstrand further testified, however, that there is no such thing as a "pure" sensory variant GBS. (Tr. 54.) This assertion finds some support in that Oh, et al, argued in favor of designating subjects with sensory variant GBS even when their NCS results showed demyelination of the motor nerves. (Oh et al., *supra* at Ex. 39, p. 4.) Critically, however, the authors explained this designation was premised on a clinical presentation fitting the criteria for sensory variant GBS, which they constructed to include normal motor strength. (*Id*.) The authors reasoned that sensory nerve demyelination is much more difficult to detect and that electrodiagnostic data does not always correlate with neuropathic symptoms. (*Id*.) Contrary to what was seen by Oh, et al, as discussed above, petitioner's EMG/NCS data demonstrated only sensory loss without motor involvement. Moreover, Dr. Engstrand actually went much further. She contended that weakness is not only compatible with sensory variant GBS, but in fact may be common or "typical" of the condition. (Tr. 54-56.) Dr. Engstrand referenced some unspecified literature she had read that supports this point; however, on the current record this assertion appears nonsensical. Explaining the absence of weakness appears to be the entire point of identifying a sensory variant of GBS.

Importantly, contrary to Dr. Engstrand's opinion, Dr. Khoshnoodi, the treating physician that diagnosed sensory variant GBS, was careful to distinguish between gait and balance difficulties as signs of abnormal sensation from true extremity weakness. (Tr. 119.) In fact, invocation of sensory variant GBS arose in this case to explain why petitioner's earlier post-2011 presentation did not include weakness. Especially because petitioner's NCS/EMG did not show any motor demyelination such as was seen in some Oh, et al, subjects, Dr. Engstrand did not adequately explain how onset of weakness could be viewed as any part of her prior condition. Dr. Khoshnoodi testified that there is no objective weakness in sensory variant GBS and that if petitioner "truly had weakness" it would change his diagnostic opinion. (Tr. 119, 143.) Despite her contention that weakness is a typical feature of sensory variant GBS, Dr. Engstrand fully

endorsed Dr. Khoshnoodi's testimony and offered no competing explanation to support the idea that onset of weakness in petitioner's case contributes to rather than confounds the diagnosis. (Tr. 219.) In that regard, Dr. Chaudhry suggested that motor weakness in the absence of NCS/EMG evidence of motor nerve involvement suggests a condition of the central nervous system.[19] (Tr. 469-70.)

Additionally, there is evidence of record to suggest that this was not a new symptom. Dr. Rodden first documented right-sided weakness (4/5) on physical examination in January of 2014 and documented this weakness throughout 2014. (Ex. 12, p. 9 (as of 1/23/14), 23 (as of 3/19/14), 46 (as of 6/26/14), 61 (as of 9/15/14).) Thus, Dr. Chaudhry opines that petitioner's physical examinations demonstrate weakness throughout 2014 and prior to the vaccination at issue. (Tr. 467-70.) Moreover, contrary to petitioner's testimony, Dr. Rodden documented in June of 2014 that petitioner had fallen three times in connection with a recent "flare" of her condition. (Ex 12, p. 45.) Onset of motor weakness during this timeframe is not consistent with a post-vaccination significant aggravation, nor with a monophasic course. (Tr. 469-70.)

Ultimately, Dr. Khoshnoodi opined that only subjective complaints support petitioner's belief that she suffered a significant aggravation of her condition following the 2014 vaccination. (Tr. 128.) Importantly then, petitioner's later medical records reveal that she has had a pattern of relapsing or exacerbated symptoms of her peripheral neuropathy corresponding to times of sickness or stress. (Ex. 33, pp. 25-27.) Her physician indicated that this is expected and not an indication that her condition is worsening. (*Id.*) Additionally, contrary to petitioner's assertion that she had previously recovered and her condition worsened only post-vaccination, Dr. Rodden's medical records show petitioner's condition declining throughout much of 2014 and prior to her November vaccination. On March 19, 2014, petitioner reported to Dr. Rodden that she was having excessive fatigue, "lots of numbness," and electrical shocks for three days. (Ex. 12, p. 21.) She also reported that her numbness was causing her to be unable to put on her surgical mask. (*Id.*) On June 26, 2014, petitioner is described as having had "a flare" since her prior visit, including an affirmative report of right-side weakness and three falls, stating that she felt her symptoms were "coming back." (*Id.* at 45.) She also reported shoulder and hip pain, stomach problems, and heat intolerance. (*Id.*) Dr. Rodden felt petitioner was having a "significant decline." (*Id.* at 47.) On September 15, 2014, petitioner reported that she had begun to experience daily "electric shocks" in her right arm. (*Id.* at 60.) Overall, Dr. Rodden characterized petitioner's condition as stable, but with symptoms progressing. (*Id.*) Lower extremity weakness is documented at this encounter. (*Id.* at 61.) As with her later records, Dr. Rodden notes that petitioner's symptoms worsen with stress. (*Id.* at 60.)

[19] Dr. Engstrand initially opined that petitioner's January 13, 2015 nerve conduction study, which demonstrated slowing of the median nerve, demonstrates new nerve damage following the 2014 vaccination. She described it as an "important" finding. (Tr. 15-18.) On cross-examination, however, she agreed the results were similar to her prior study and that she was relying instead on petitioner's clinical presentation to demonstrate the worsening of her condition. (Tr. 216-17.) In any event, the significance of petitioner's January 13, 2015 NCS/EMG to her sensory variant GBS diagnosis has already been addressed.

Based on the record as a whole, petitioner's condition still did not constitute sensory variant GBS in the post-2014 vaccination period and also does not appear to have been worse than her condition throughout 2014.

c. *Loving* Prong Three

A significant aggravation is "any change for the worse in a preexisting condition which results in markedly greater disability, pain, or illness accompanied by substantial deterioration of health." 42 U.S.C. § 300aa-33(4). Petitioner does not need to prove that her condition is worse than the expected outcome of her pre-existing condition, but need only compare her pre- and post-vaccination conditions. *Sharpe v. Sec'y of Health & Human Servs*., 964 F.3d 1072, 1081-82 (Fed. Cir. 2020). Here, petitioner alleges she suffered a significant aggravation of pre-existing sensory variant GBS. (ECF No. 38, p. 1.) For the reasons discussed above, however, there is not preponderant evidence petitioner ever had sensory variant GBS, either before or after her 2014 flu vaccination. Accordingly, petitioner's suggestion that her 2014 post-vaccination symptoms represented worsening symptoms of that condition cannot succeed. Petitioner has not presented any other diagnosis or explanation for her pre-existing condition nor has she explained, in the absence of a sensory variant GBS diagnosis, how her post-vaccination symptoms can be related to her pre-existing symptoms.

Additionally, as the treating physician that initially proposed a sensory variant GBS diagnosis, Dr. Khoshnoodi's opinion in this case fails to provide preponderant evidence underlying the claim that petitioner experienced a significant aggravation of that condition. As explained above, Dr. Khoshnoodi's medical records reflect only a suggestion of "residual effects" of sensory variant GBS among his differential diagnosis. (Ex. 21, p. 4.) His initial letter to the court addressed post-2011 diagnosis only. (Ex. 22.) In a subsequent letter he additionally asserted that petitioner's 2014 vaccination "result[ed] in worsening of her symptoms." (Ex. 37, p. 1.) Upon further questioning from the previously assigned special master, he identified only petitioner's subjective complaints as evidence of worsened symptoms. (Ex. 38, p. 2.) Additionally, Dr. Khoshnoodi stressed that his opinion was predicated on petitioner's description of onset and that he would not maintain his diagnostic opinion if onset was different than what was represented to him. (Tr. 127-28.) He also confirmed in testimony that his assessment as to petitioner's significant aggravation was based on his trust in petitioner to accurately report her subjective complaints and that he did not see petitioner during the acute phase of her alleged significant aggravation. (Tr. 131.) Ultimately, during the hearing, Dr. Khoshnoodi testified:

> Q: Is it your opinion that in 2014, she had a second phase of sensory variant GBS?
>
> A: I do not know. No.

(Tr. 139.)

Thus, petitioner's suggestion that she experienced a significant aggravation of sensory variant GBS does not ultimately find preponderant support among petitioner's many treating physicians. As explained above, petitioner sought treatment for three years and from four different neurologist before Dr. Khoshnoodi proposed sensory variant GBS in his differential diagnosis. None of those physicians offered any similar diagnosis. Although some of petitioner's treating physicians, such as Dr. Rodden, appear to have subsequently accepted Dr. Khoshnoodi's sensory variant diagnosis, it is not possible to glean from the records that they would have arrived at or maintained that diagnosis without Dr. Khoshnoodi's initial opinion. (Ex. 20, pp. 47-49 (noting prior sensory variant GBS diagnosis made at Johns Hopkins and assessing sensory variant GBS with "workup ongoing").) Petitioner's other treating neurologists continued to question both the diagnosis itself and the idea that petitioner's condition was vaccine related. On July 8, 2016, Dr. Phillips wrote to Dr. Bonagiri following a neurological consultation with petitioner. (Ex. 37, pp. 50-53.) He recounted petitioner's prior diagnostic assessments in detail, including Dr. Khoshnoodi's "presumed diagnosis [of a] GBS-like disease process," but nonetheless characterized petitioner as presenting with "a complicated constellation of neurological symptoms for which the diagnosis has been unclear." (*Id*. at 50.) Dr. Phillips acknowledged the suspicion raised by petitioner's reported history of a temporal association to her vaccinations, but cautioned that "we cannot say for certain that her vaccinations were the definite triggers for her clinical syndrome." (*Id*. at 53.) Subsequently, in September of 2018, Dr. Shipley characterized petitioner's history of GBS as only "questionable" or "possible." (Ex. 33, pp. 4-5.)

d. *Loving* Prongs Four, Five and Six (also the *Althen* test)

As noted above, petitioner's amended petition asserts a significant aggravation claim. (ECF No. 38.) However, in her post-hearing brief, she contends more broadly that her November 2, 2014 flu vaccination either initially caused or significantly aggravated her sensory variant GBS.[20] (ECF No. 123, p. 1.) In either event, *Loving* prongs four through six follow the *Althen* test for demonstrating causation in fact. Under either approach, the questions posed by this analysis are whether the petitioner presented a medical theory causally connecting the vaccination and the injury, in this case sensory variant GBS; whether a logical sequence of cause and effect demonstrates the vaccine causally contributed to the injury in this specific case; and, whether there is a medically reasonable temporal relationship between the vaccine and injury.

Here, there is no genuine dispute regarding the general medicine. In placing GBS on the Vaccine Injury Table relative to the flu vaccine, respondent had already recognized a causal relationship between the flu vaccine and GBS under at least some

[20] Petitioner also stresses a prior order from the previously assigned special master noting that an additional question is whether petitioner also had autonomic issues. (ECF No. 123, p. 1.) Notably, however, during the hearing any mention of autonomic symptoms by either Dr. Engstrand or Dr. Khoshnoodi were limited to discussing potential sequela of GBS (Tr. 88-89, 106, 119, 125.) Accordingly, petitioner has not substantiated any separate claim for autonomic dysfunction.

circumstances. National Vaccine Injury Compensation Program: Revisions to the Vaccine Injury Table, 82 Fed. Reg. 6294-01, 6295 (Jan. 19, 2017) (explaining that "the Secretary found that there was compelling, reliable, and valid medical and scientific evidence of an association between the 2009 H1N1 vaccine and GBS . . . To date, the H1N1 antigen has been included in all seasonal influenza vaccines beginning with the 2010-2011 flu season.") Additionally, Dr. Chaudhry testified on behalf of respondent that, although they have different presentations, it is not currently possible to etiologically distinguish sensory variant GBS from other forms of GBS. (Tr. 498-500.) And, although Dr. Chaudhry expressed uncertainty bordering on skepticism regarding the available evidence causally linking GBS to the flu vaccine, he did ultimately testify that under certain circumstances he would be willing to causally attribute a case of sensory variant GBS to a prior flu vaccine.[21] (Tr. 494-500.) Because *Loving* prong four/*Althen* prong one relates to general causation only, this effectively places this particular prong beyond dispute. *See Pafford v. Sec'y of Health & Human Servs.*, 451 F.3d 1352, 1355-56 (Fed. Cir. 2006) (accepting the special master's interpretation of *Althen* prongs one and two as asking "can it?" and "did it?" respectively). 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, 548-49 (Fed. Cir. 1994).

With respect to *Loving* prong five, petitioner must show that her vaccination affected her own condition. *Sharpe*, 964 F.3d at 1082, 1085-86. Here, Dr. Engstrand's testimony was a bit confusing. During direct examination, she testified that her opinion was that a logical sequence of cause and effect existed between petitioner's November 2014 vaccination and her injury, both because she developed new symptoms and because "her other symptoms that she had before got worse." (Tr. 75-76.) In response to my question, she indicated that her opinion was that petitioner experienced two monophasic courses of sensory variant GBS, one in 2011 and one in 2014 (Tr. 72); however, she ultimately confirmed that her opinion was that a pre-existing condition was significantly aggravated by the vaccination. (Tr. 76-77.) On cross examination, Dr. Engstrand again confirmed her opinion that petitioner had residual effects of pre-existing sensory variant GBS at the time of her November 2014 flu vaccination, but also responded affirmatively when asked if her opinion was that petitioner "*developed* GBS or sensory variant GBS *after* her 2014 vaccine." (Tr. 84 (emphasis added).) For his part, Dr. Khoshnoodi ultimately disclaimed during the hearing that petitioner developed sensory variant GBS following her 2014 vaccination or experienced a second phase of the condition following that vaccination. (Tr. 139.)

In any event, under *Loving* prong five/*Althen* prong two, petitioner's failure to substantiate her allegations with respect to *Loving* prongs one through three is fatal to her claim. Importantly, analysis of the first three *Loving* prongs demonstrates not only that petitioner was never definitively diagnosed with sensory variant GBS, but also that

[21] Specifically, Dr. Chaudhry offered his own hypothetical, testifying that "if you were to ask me that if it was all --let's say, she had flu shot and few days later came to the hospital with numbness and tingling, she had absent reflexes, normal strength, high CSF protein, and then things would stay the same or improved, and she was seen and documented to have, and it was after the flu vaccine, I would say that likely that was the cause, if other causes were excluded including nutritional and toxic." (Tr. 499.)

her presentation did not fall within the diagnostic criteria for that condition at any time either before or after her 2014 flu vaccination. Further, she did not demonstrate that her post-vaccination condition represented a worsening of that condition. Absent that diagnosis, she has not otherwise suggested how her vaccination could have affected her condition. Thus, these findings necessarily preclude her from demonstrating a logical sequence of cause and effect implicating her November 2014 vaccination as a factor contributing to her current condition, either as an initiating cause or a significant aggravator. On the whole, and for all the reasons discussed above, Dr. Chaudhry was more persuasive in opining that petitioner experienced a continuous, fluctuating, and progressive course of an undiagnosed neurological condition spanning from years prior to years after her 2014 flu vaccination without any readily discernable connection to her vaccination.

With respect to timing under *Loving* prong six, Dr. Engstrand indicated that injury onset of up to six weeks post-vaccination is consistent with a causal relationship between vaccination and injury. (Tr. 76.) This is consistent with the timing demonstrated by the Vaccine Injury Table. 42 C.F.R. § 100.3(a). For his part, Dr. Chaudhry allowed that the injury table is instructive. (Tr. 500-06.) However, in light of all of the above, even if petitioner did have some documented complaints occurring within the relevant timeframe, this is effectively immaterial. *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).

VI. Conclusion

There is no dispute in this case that petitioner suffers a neurologic injury that has profoundly affected her life. She has my sympathy and I do not question her sincerity in bringing this claim. However, for all the reasons discussed above, I find that petitioner has not met her burden of proof in this case. She has not provided preponderant evidence that she suffers sensory variant GBS. She has not provided preponderant evidence that her condition was initially caused by either her 2011 or 2014 influenza vaccination. Nor has she provided preponderant evidence that her condition was significantly aggravated by her 2014 influenza vaccination. Therefore, this case is dismissed.[22]

IT IS SO ORDERED.

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

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