In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 19-1307V
Filed: December 18, 2025

TIMOTHY WILLIAMS,

Petitioner,

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,

Respondent.

Special Master Horner

Emily Beth Ashe, Anapol Weiss, Philadelphia, PA, for petitioner.
Meghan Murphy, U.S. Department of Justice, Washington, DC, for respondent.

DECISION[1]

On August 28, 2019, petitioner, Timothy Williams, filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10, *et seq.* (2012),[2] alleging that he suffered myasthenia gravis ("MG") and chronic inflammatory demyelinating polyneuropathy ("CIDP")[3] caused-in-fact by an influenza ("flu") vaccination that he received on September 16, 2016. (ECF No. 1, p. 1.) On March 29, 2021, petitioner filed an amended petition to additionally allege inflammatory polyradiculopathy. (ECF

[1] Because this document contains a reasoned explanation for the action taken in this case, it must be made publicly accessible and will be posted on the United States Court of Federal Claims' website, and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the document 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, upon review, I agree that the identified material fits within this definition, I will redact such material from public access.

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

[3] Although this decision consistently uses the acronym CIDP, in some instances varying terms were used to refer to this condition. The petition refers to CIDP as being a polyneuritis and Dr. Steinman made some references to "chronic inflammatory neuropathy" or "chronic demyelinating neuropathy" while Dr. Price at one point referenced "chronic inflammatory demyelinating polyradiculoneuropathy."

No. 44, p. 1.) For the reasons set forth below, I conclude that petitioner is *not* entitled to an award of compensation.

I. Applicable Statutory Scheme

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

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 ex rel. Sevier v. Sec'y of Health & Human Servs.*, 940 F.2d 1518, 1525 (Fed. Cir. 1991).

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 1278-79; *Hines*, 940 F.2d at 1525. Under that standard, 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. He need not show that the vaccination was the sole cause but must demonstrate that the vaccination was at least a "substantial factor" in causing the condition at issue and was a "but for" cause. *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999). Thus, petitioner must supply "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury[.]" *Althen*, 418 F.3d at 1278 (quoting *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992)). Ultimately, petitioner must satisfy what has come to be known as the *Althen* test, which requires: (1) a medical theory causally connecting the vaccination and the injury; (2) a

logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury.[4] *Id*.

Because the Vaccine Act requires a petitioner to present a claim for compensation for a "vaccine-related injury or death," § 300aa-11(c); § 300aa-13(a)(1)(A); *Stillwell v. Sec'y of Health & Human Servs.*, 118 Fed. Cl. 47, 56 (2014), *aff'd per curiam*, 607 F. App'x 997 (Fed. Cir. 2015), a petitioner "must specify his vaccine-related injury and shoulder the burden of proof on causation." *Broekelschen v. Sec'y of Health & Human Servs.*, 618 F.3d 1339, 1346 (Fed. Cir. 2010). Accordingly, where the identity and nature of the vaccine-related injury is in dispute, the Federal Circuit has held that it is "appropriate for the special master to first determine what injury, if any, [is] supported by the evidence presented in the record before applying the *Althen* test to determine causation. *Lombardi v. Sec'y of Health & Human Servs.*, 656 F.3d 1343, 1352-53 (Fed. Cir. 2011). However, "the function of a special master is not to 'diagnose' vaccine-related injuries." *Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1382 (Fed. Cir. 2009). Instead, special masters shall determine "based on the record evidence as a whole and the totality of the case, whether it has been shown by a preponderance of the evidence that a vaccine caused the [petitioner's] injury." *Id.* (quoting *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 549 (Fed. Cir. 1994)).

Ultimately, 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). Medical records are generally viewed as particularly trustworthy evidence because they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). However, medical records and/or statements of a treating physician's views do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. § 300aa-13(b)(1). A petitioner may rely upon circumstantial evidence. *See Althen*, 418 F.3d at 1280. Moreover, 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

[4] The Vaccine Act also permits petitioners to recover damages for any vaccine-caused "significant aggravation" of a pre-existing condition. The Act defines significant aggravation as "any change for the worse in a preexisting condition which results in markedly greater disability, pain, or illness accompanied by substantial deterioration of health." § 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 establish three additional factors. *See Loving v. Sec'y of Health & Human Servs.*, 86 Fed. Cl. 135, 144 (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 significant 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 significant 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. *Loving*, 86 Fed. Cl. at 144.

1279-80. While scientific certainty is not required, that expert's opinion must be based on "sound and reliable" medical or scientific explanation. *Boatmon v. Sec'y of Health & Human Servs*., 941 F.3d 1351, 1359 (Fed. Cir. 2019).

Cases in the Vaccine Program are assigned to special masters who are responsible for "conducting all proceedings, including taking such evidence as may be appropriate, making the requisite findings of fact and conclusions of law, preparing a decision, and determining the amount of compensation, if any, to be awarded." Vaccine Rule 3(b)(1). Special masters must ensure each party has had a "full and fair opportunity" to develop the record but are empowered to determine the format for taking evidence based on the circumstances of each case, including having the discretion to decide cases without an evidentiary hearing. Vaccine Rule 3(b)(2); Vaccine Rule 8(a); Vaccine Rule 8(d). Special masters are not bound by common law or statutory rules of evidence but must consider all relevant and reliable evidence in keeping with fundamental fairness to both parties. Vaccine Rule 8(b)(1). The special master is required to consider "all . . . relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." § 300aa-13(b)(1). The special master is required to consider the entirety of the evidentiary record, draw plausible inferences, and articulate a rational basis for the decision. *Winkler v. Sec'y of Health & Human Servs.*, 88 F.4th 958, 963 (Fed. Cir. 2023) (citing *Hines*, 940 F.2d at 1528).

II. Procedural History

As noted above, the initial petition alleged either MG or CIDP caused-in-fact by petitioner's vaccination. (ECF No. 1.) In September of 2019, petitioner filed initial medical records and an affidavit in support of his claim. (ECF Nos. 7-8; Exs. P1-P9.) Throughout the rest of 2019 and the beginning of 2020, petitioner filed additional medical records and a statement of completion. (ECF Nos. 10, 12, 14, 16-17, 19-21; Exs. P10-P18.)

Respondent filed a Rule 4(c) Report on August 7, 2020. (ECF No. 26.) Respondent contended that petitioner failed to support his allegation of vaccine injury with preponderant evidence, stressing that petitioner had not submitted an expert report, petitioner's symptoms preceded vaccination by between six months to five years, and the lack of reliable evidence showing a causal association between the flu vaccine and MG and/or CIDP in the existing medical literature. (*Id.* at 9-10.) Thereafter, petitioner filed additional medical records and another statement of completion by the end of 2020. (ECF Nos. 35-36; Exs. P19-P20.)

In March of 2021, petitioner filed additional medical records and another statement of completion. (ECF Nos. 41-42; Exs. P21-P23.) Petitioner filed an expert report by neurologist and neuroimmunologist Lawrence Steinman, M.D., with supporting

medical literature, on March 29, 2021. (ECF Nos. 43; Ex. P24.) Dr. Steinman opined that petitioner's condition represented an inflammatory polyradiculopathy, rather than either CIDP or MG. (Ex. P24, p. 6.) Thereafter, petitioner filed an amended petition, alleging inflammatory polyradiculopathy in addition to the previously alleged MG and CIDP. (ECF No. 44.)

Respondent then filed responsive expert reports by neurologist Raymond S. Price, M.D., and immunologist John T. Bates, Ph.D., along with supporting medical literature. (ECF Nos. 47-48; Exs. A-D.) Dr. Price opined that petitioner suffered MG, but not CIDP or inflammatory polyradiculopathy. (Ex. A, pp. 9-13.) Dr. Bates's opinion contested Dr. Steinman's theory of vaccine causation vis-à-vis inflammatory polyradiculopathy. (Ex. C.) The parties then exchanged further reports by their experts. (ECF Nos. 50, 52, 54-55; Exs.P25-P26; Exs. E-F.)

An entitlement hearing was scheduled to commence on July 23, 2024. (ECF No. 59.) Before the hearing, petitioner filed additional medical records and medical literature. (ECF Nos. 60, 62-64; Exs. P27-P36.) In a joint pre-hearing submission, the parties agreed that petitioner did not suffer CIDP, narrowing the diagnostic dispute to MG versus inflammatory polyradiculopathy. (ECF No. 69.)

The two-day entitlement hearing commenced on July 23, 2024. (*See* Transcript of Proceedings ("Tr."), at ECF Nos. 80-81.) At the close of the hearing, the parties confirmed that the evidentiary record was complete. (ECF No. 76.) However, I advised the parties that, although petitioner had initially pleaded a vaccine-caused MG, I felt that petitioner had effectively abandoned any prosecution of such a claim. (Tr. 333-34.) I instructed petitioner to explain in post-hearing briefing what basis, if any, he had for continuing to assert a claim for vaccine-caused MG. (*Id.* at 334-35.) Thereafter, on July 26, 2024, petitioner filed a status report indicating that he was "releasing his claim" that his flu vaccine caused MG. (ECF No. 77.)

Petitioner filed his post-hearing brief on October 7, 2024 (ECF No. 83); respondent filed his responsive post-hearing brief on November 2, 2024 (ECF No. 84); and petitioner filed his reply on November 18, 2024 (ECF No. 85). Accordingly, this case is now ripe for resolution of entitlement.

Petitioner argues that he has met his burden of proof with respect to a vaccine-caused inflammatory polyradiculopathy regardless of whether he additionally suffered MG. (ECF Nos. 83, 85.) Respondent argues, however, that petitioner only ever suffered MG and not inflammatory polyradiculopathy. (ECF No. 84, pp. 15-18.) Because I agree with respondent, and because petitioner did not pursue any claim that his vaccine caused or aggravated MG, it is not necessary to reach any *Althen* or *Loving* analysis in this case.

III. Factual History

a. Medical Records

Before receiving the vaccine at issue in this case, petitioner had a medical history significant for sinus problems, coronary artery disease, and hypertension. (*E.g.*, Ex. P2, p. 158; Ex. P13, p. 11.) Additionally, he suffered from chronic low back pain with radiculopathy and was treated with Depo-Medrol injections. (*E.g.*, Ex. P2, p. 165; Ex. P11, pp. 311-23.) On December 8, 2014, petitioner reported that, despite conservative treatment with pain and anti-inflammatory medication, his back pain was so severe that he was unable to walk more than 100 feet before having to stop. (Ex. P8, pp. 250-52.) He additionally reported numbness in his right thigh that caused his leg to give way, interfering with his ability to work. (*Id.* at 252.) Petitioner was treated with Depo-Medrol injections and NSAIDs for hip and back pain up until the vaccination at issue in this case. (Ex. P8, pp. 186-93, 201, 205-08, 211-13, 228-30.) Petitioner also received annual flu vaccinations in 2013, 2014, and 2015. (Ex. P2, pp. 94, 172.)

On September 16, 2016, petitioner followed up with his primary care physician, William Pickard, M.D., for back pain and sinus problems. (Ex. P2, pp. 93-96.) Dr. Pickard noted that petitioner's back pain, which was associated with his lumbar radiculopathy, was improved and stable at that time. (*Id.* at 93.) However, during this appointment, petitioner reported back and joint pain, as well as sinus pressure. (*Id.* at 95.) His physical exam was positive for low back pain, as well as sinus congestion pain and pharyngitis. (*Id.* at 95-96.) Petitioner received the flu vaccination at issue at this appointment. (*Id.* at 94; Ex. P1, p. 1.)

On September 20, 2016, petitioner returned to Dr. Pickard with reports of excessive fatigue, which he suspected could be due to Lyme disease as he had recently spent time in the woods. (Ex. P2, p. 90.) However, he denied a rash and subsequent Lyme disease testing was negative. (*Id.* at 90, 182.) His physical exam continued to show pharyngitis and low back pain but was otherwise normal. (*Id.* at 92-93.)

The next day, September 21, 2016, petitioner presented to neurologist Mohammad Sadeghi, M.D. (Ex. P3, pp. 1-8.) On his intake form, petitioner indicated that he presented for evaluation of possible multiple sclerosis symptoms. (*Id.* at 25.) He reported a five-year history of intermittent, aching pain that "starts in my arm and wrist on left side, then goes to the left hip with a burning sensation and then from calf down to foot on left side." (*Id.* at 25-26.) His pain intensified when sitting but improved when walking. (*Id.* at 26.) Petitioner also reported back pain, numbness and weakness in his arm and leg, tingling in hands and feet, limping, facial weakness and tremors, difficulties with speech and swallowing, cough, extreme fatigue, decreased concentration, double or blurry vision, and lightheadedness. (*Id.* at 27-29.) He noted that he experiences his back pain "[q]uite a bit." (*Id.* at 28.) Petitioner described his leg numbness, weakness, and pain as moderate, while noting that his arm weakness was moderate with only minimal pain and numbness. (*Id.*)

At his appointment with Dr. Sadeghi, petitioner reported a history of insomnia with worsening fatigue over the past four years. (Ex. P3, pp. 5, 7.) Petitioner indicated that he was started on testosterone injections to address his fatigue, which helped for a time, but he noticed worsening fatigue about six months prior, followed by new onset of blurry vision. (*Id.*) He described one "major" episode at night, as well as frequent episodes while watching television. (*Id.*) He also reported intermittent numbness of the left side of his face, arm, and leg; increased phonation; difficulty swallowing; and slurred speech. (*Id.*) On physical exam, petitioner's speech was "somewhat slurred" and nasal but coherent, and his sensory exam, coordination, and gait were normal. (*Id.* at 6, 8.) Petitioner's strength was normal and intact in his bilateral upper and lower extremities, with the exception of some mild fatigability in petitioner's right upper extremity. (*Id.*) Dr. Sadeghi ordered testing for myasthenia gravis ("MG") and prescribed Mestinon. (*Id.*)

Petitioner underwent an MRI of his brain on September 28, 2016. (Ex. P4, p. 14.) The study showed no acute intracranial abnormality and mild nonspecific white matter changes most commonly secondary to chronic small vessel ischemia. (*Id.*) On October 6, 2016, petitioner presented to Dr. Sadeghi for a review of his MRI and follow up for possible MG. (Ex. P3, p. 46.) He reported improvement in his symptoms since he began taking Mestinon. (*Id.*) Specifically, he reported that he did not feel as fatigued and denied further episodes of diplopia and ptosis. (*Id.*) However, petitioner did note that he "takes up to 3 or 4 tablets" of Mestinon when working the night shift as his symptoms recur at the end of the day. (*Id.*) On physical exam, petitioner had diplopia and fatigability upon sustained upward gaze. (*Id.* at 47.) However, strength in his bilateral upper and lower extremities was normal with no appreciable fatigability. (*Id.*) Petitioner's sensory examination, gait, and coordination were normal. (*Id.*)

Petitioner had a follow up appointment with his primary care provider on October 14, 2016. (Ex. P2, p. 87.) Dr. Pickard noted that petitioner had recently been diagnosed with MG but was doing much better after starting Mestinon. (*Id.* at 89.) Petitioner reported joint and back pain, as well as sinus problems. (*Id.*) Petitioner's low back pain, sinus congestion pain, and pharyngitis were confirmed on physical exam. (*Id.* at 90.) However, his exam was otherwise unremarkable. (*Id.*)

On October 27, 2016, petitioner presented to gastroenterologist Dr. Nezar Shobassy. (Ex. P5, p. 2.) Petitioner reported his MG diagnosis and explained that he was having indigestion and difficulty swallowing. (*Id.*) His physical examination was normal. (*Id.*) Dr. Shobassy scheduled petitioner for an abdominal ultrasound, an esophagogastroduodenoscopy ("EGD"), and a colonoscopy. (*Id.*) Petitioner's EGD was suggestive of Barrett's esophagus; however, a biopsy was performed which was negative for Barrett's esophagus with no evidence of dysplasia or malignancy. (Ex. P5, pp. 1, 3-4, 6.)

On November 2, 2016, petitioner had a follow up appointment with his primary care physician for his lumbar radiculopathy. (Ex. P2, p. 84.) While physical exam continued to show joint and back pain, Dr. Pickard noted that petitioner's back pain was

somewhat improved. (*Id.* at 86-87.) At this appointment, petitioner requested a neurology referral for a second opinion. (*Id.* at 84, 87.) Additionally, Dr. Pickard ordered MG antibody panels after documenting that petitioner's neurologist "lost" his prior laboratory results. (*Id.* at 87.) During a follow up encounter on November 18, 2016, Dr. Pickard noted that petitioner was still undergoing a neurology workup. (*Id.* at 81.) That same day, petitioner's lab results, including an MG panel and testing for acetylcholine receptor binding antibodies and muscle-specific kinase ("MuSK") antibodies, returned negative. (*Id.* at 178-81.) However, it is noted in the record that a negative result does not conclusively rule out MG. (*Id.* at 179.)

On November 28, 2016, petitioner presented for further evaluation by neurologist, Robert Glenn Smith, M.D. (Ex. P6, p. 3.) Dr. Smith noted petitioner's MG diagnosis and description of "2 different types of problems" involving weakness. (*Id.* at 4.) Petitioner reported "many years" of severe fatigue "when he has overworked himself," with a recovery period of hours to overnight before returning to his baseline. (*Id.*) This problem was compounded by his chronic low back pain and history of radicular pain, and he noted an increase in the severity of his symptoms over the past two years. (*Id.*) However, petitioner's "more recent severe activity dependent fatigue" appeared to be a separate issue. (*Id.*) He documented an episode of weakness and difficulty breathing, requiring up to two days of sleep before he felt normal. (*Id.*) Petitioner reported that his arms and legs "feel like lead" and become very fatigued with activity. (*Id.*) He described another episode, during which petitioner experienced leg cramps from climbing stairs or ladders and low back pain without radicular pain. (*Id.*) He also reported bilateral ptosis with episodic muscle twitching, and worsening choking on foods and liquids, especially when fatigued. (*Id.*) Petitioner reported further weakness in his shoulders and neck. (*Id.*) Although petitioner reported diplopia, Dr. Smith opined that this could be related to petitioner's cataracts, rather than actual binocular diplopia. (*Id.*) Petitioner reported improvement in his ptosis, diplopia, and fatigue with Mestinon, as well as a reduction in pain with Celebrex. (*Id.*) While Dr. Smith did not have petitioner's lab results to review, he recorded that, according to petitioner, the initial and repeat MG panels were both negative. (*Id.*) Dr. Smith further recorded that petitioner "has a history of sciatica, possibly most affecting dermatome L5, and S1," as well as "a history of singultus, associated with his increase in episodic choking." (*Id.*)

Physical exam revealed mild, bilateral ptosis affecting the right more than the left; mild atrophy of the tongue; and minimally reduced strength in petitioner's left hip flexor, knee extensor, and ankle evertor. (Ex. P6, pp. 6-7.) His reflexes were normal and intact, with the exception of reduced reflexes in petitioner's knees bilaterally. (*Id.* at 7-8.) Petitioner's sensory exam, coordination, and gait were all normal. (*Id.* at 8.) Petitioner's differential diagnosis was MG, though Dr. Smith observed that additional testing was necessary to verify the diagnosis. (*Id.* at 9.) He therefore ordered an EMG, CT of the chest with contrast, and basic metabolic panel. (*Id.*)

Petitioner underwent an EMG/nerve conduction study on December 7, 2016. (Ex. P19, p. 2.) There was no evidence of decrement on repetitive nerve stimulation.

(Ex. P19, p. 8.) The EMG revealed evidence of "inactive, mild to moderate severity denervation with partial reinnervation of muscles normally in functional contact with motor neurons at multiple lumbar levels, consistent with an inactive polyradiculopathy." (Ex. P19, p. 8.) The upper extremity muscles tested were "much less affected electrically, with denervation changes noted mostly in C7 and possible C8 innervated muscles." (*Id.*) The nerve conduction study revealed normal distal latencies and conduction velocities in the tested upper and lower extremity motor nerves with the exception of right ulnar distal latency and mild slowing of the left median motor velocity. (*Id.*) F-wave latencies were essentially normal. (*Id.*) Dr. Smith's impression of petitioner's EMG/nerve conduction study findings was

> [w]idespread inactive motor denervation with partial reinnervation in the tested lower extremity, with significantly less denervation reinnervation changes observed in the tested upper extremity. Likewise, reduction in recruitment was observed in both upper and lower extremities tested. Inactive denervation changes were observed in the thoracic paraspinal muscles and in the genioglossus muscles.
>
> Relatively normal nerve conduction studies, though early demyelination changes possibly may be observed in right median and ulnar nerve responses.

(*Id.* at 10.) These findings suggested the presence of a "longstanding, progressive, relatively inactive, polyradiculopathy" without significant neuropathy. (*Id.*) Although the differential diagnosis was broad and included inflammatory, infectious, paraneoplastic, POEMS, MUGAS, vascular, metabolic, or genetic etiologies, it was noted that many of these etiologies had been excluded and that petitioner's upper motor neuron findings were not consistent or present in most of these diseases. (*Id.*) Additionally, petitioner's chest CT showed "[a] couple of lung nodules bilaterally likely post inflammatory." (Ex. P23, p. 1.)

On January 17, 2017, petitioner followed up with Dr. Smith to review his test results. (Ex. P6, pp. 22-23.) Dr. Smith recorded that petitioner presented with a symptom complex suggestive of a neuromuscular junction disorder with improvement of ptosis and weakness with Mestinon. (*Id.* at 23.) In documenting petitioner's history, Dr. Smith recorded:

> Since last seen, he [has] undergone EMG, which did not confirm decrement on repetitive stimulation, but noted the presence of a polyradiculopathy. While no obvious neuropathic findings were observed, the patient did have significant side-to-side variation in his left versus right median nerve, with slight slowing in conduction velocity noted on the tested left side. Similar observation was noted in comparing the left and right sensory median results. Other than prolonged motor distal latency of the ulnar nerve on the left, without sensory latency abnormality, no other significant abnormalities were noted on nerve conduction studies.

(*Id.*) Petitioner reported that he was able to stay off Mestinon for 36 hours prior to his EMG study. (*Id.* at 23-24.) However, his weakness and fatigue had worsened since his last visit. (*Id.* at 23.) Although his symptoms improved with Mestinon, Dr. Smith documented that petitioner was unable to completely control his activity dependent weakness. (*Id.*)

Petitioner's physical examination again showed mild, bilateral ptosis affecting the right more than the left, mild tongue atrophy, minimally reduced strength in the left lower extremity, as well as mild tenderness over the cervical spine without reduced range of motion or muscle spasm and tenderness over the lumbosacral spine with mildly reduced range of motion. (Ex. P6, pp. 26-27.) His sensory exam, coordination, and gait were all again normal, and his reflexes were normal and intact with the exception of reduced reflexes in his knees, bilaterally. (*Id.* at 28.) X-ray of the thoracic and cervical spine both showed degenerative changes. (Ex. P23, pp. 2-3.) Dr. Smith documented that the x-ray of petitioner's cervical spine revealed "C6/C7 degenerative/spondylitic changes" with minimal changes observed in the thoracic spine. (Ex. P6, p. 29.) Dr. Smith's assessment was MG with exacerbation, CIDP, bilateral lumbar radiculopathy, chronic bilateral low back pain without sciatica, polyradiculopathy, and a possible cervical radiculopathy at C7/C8. (*Id.* at 29-31.) In addition to continuing with Mestinon, petitioner was started on prednisone and CellCept, and further testing was ordered. (*Id.* at 25, 29-31.)

On January 25, 2017, petitioner underwent a successful lumbar puncture, which revealed no abnormalities. (Ex. P23, p. 4; Ex. P29, p. 389.) Given that there was no evidence of any significant brachial blood brain barrier or CSF inflammation, Dr. Smith opined that most of the differential diagnoses for polyradiculopathy could be excluded on the basis of the lumbar puncture results. (Ex. P6, p. 31.)

Petitioner's next follow up appointment with Dr. Smith was on March 30, 2017. (Ex. P6, pp. 44, 47-48.) Since his last visit, petitioner reported improvement in his generalized weakness, fatigue, ptosis, double vision, and beathing. (*Id.*) His response to Mestinon was described as "variable." (*Id.*) Dr. Smith noted that petitioner called the office in January of 2017 to report persistent muscle weakness that continued to progress. (*Id.*) At that time, petitioner's work was significantly limited by weakness and associated shortness of breath. (*Id.*) However, petitioner reported "significant improvement" after introducing prednisone and CellCept. (*Id.*) Petitioner also reported continued episodic difficulty swallowing and "choking spells," though it was noted that "symptom onset predates the onset of other myasthenic symptoms." (*Id.*) He described his fatigue and weakness as constant. (*Id.*) Although he experienced an erratic sleep pattern due to his shift work, he reported "feeling excessively fatigued even after full night/day sleep." (*Id.*) He also reported occasional transient double vision at the end of his workday. (*Id.*) However, he denied shortness of breath after day-to-day activities, focal weakness in arms and legs, or a limitation of his activity at work or at home due to weakness. (*Id.*)

Petitioner's physical exam showed mild ptosis, affecting the right more than the left; mildly reduced range of motion in his lumbar spine; slightly reduced strength in his left deltoid, bicep, hip flexor, and knee extensor; and reduced strength in the left ankle evertor. (Ex. P6, pp. 51-52.) Petitioner's reflexes were normal with the exception of reduced reflexes in his triceps, bilaterally. (*Id.* at 52.) Petitioner's sensory examination, coordination, and gait were all normal. (*Id.* at 53.)

Dr. Smith continued to list MG as a diagnosis. (Ex. P6, p. 55.) However, CIDP was removed from petitioner's problem list. (*Id.* at 44.) Dr. Smith noted that, although petitioner's EMG showed no evidence of decrement on repetitive nerve stimulation and his antibody testing was negative, petitioner has had "significant response" to his treatment regimen. (*Id.* at 55.) Dr. Smith suggested a reevalution for MG antibodies. (*Id.*) Additionally, Dr. Smith noted petitioner's long shifts that alternated between day and night shifts, highlighting that petitioner had finished his night shift early that morning but had not yet slept by the time he presented for his afternoon appointment, and that petitioner reported significant fatigue, despite normal strength testing that was stable when compared to his prior testing. (*Id.*) Dr. Smith prescribed modafinil on a trial basis for "shift work sleep disorder." (*Id.*) He also documented concern for undiagnosed obstructive sleep apnea and advised petitioner to undergo a sleep study. (*Id.*)

To evaluate for a clinical diagnosis of vertebrobasilar insufficiency, Dr. Smith recommended that petitioner undergo an MRA of the head and neck. (Ex. P6, p. 55.) Petitioner's subsequent MRA on April 7, 2017, was unremarkable. (Ex. P23, pp. 5-6.) Petitioner followed up with his primary care physician on April 26, 2017, and May 26, 2017. (Ex. P2, pp. 62-68.) At both encounters, petitioner reported low back pain. (*Id.* at 65, 68.) Additionally, his primary care provider noted that petitioner's energy levels were improving on his current medication regimen and that his insomnia was also better. (*Id.* at 68.)

On June 14, 2017, petitioner had a follow up appointment with Dr. Smith. (Ex. P6, p. 67.) Although petitioner reported experiencing both good and bad days, he described fewer episodes of swallowing problems. (*Id.* at 70.) He also could not recall when he last experienced double vision, but continued to report occasional blurred vision when fatigued. (*Id.*) Petitioner continued to take CellCept, Mestinon, and prednisone. (*Id.*) He denied fluctuating weakness, despite taking prednisone on alternating days. (*Id.*) Additionally, petitioner reported experiencing significant improvement in function when taking modafinil to address his shift work sleep disorder. (*Id.* at 67, 77.) Dr. Smith documented that petitioner's symptoms of double vision, ptosis, and shortness of breath had largely resolved. (*Id.* at 77.) Additionally, he recorded that petitioner was negative for the acetylcholine receptor and the MuSK antibodies and without evidence of decrement on repetitive nerve stimulation. (*Id.*) However, Dr. Smith noted that petitioner had a polyradiculopathy on needle exam and that the possibility of a distal inflammatory motor polyneuropathy producing symptoms similar to MG could not be ruled out. (*Id.*) Dr. Smith assessed petitioner with MG, cervical radiculopathy at C7, polyradiculopathy, and bilateral lumbar radiculopathy, as

well as insomnia and shift work sleep disorder. (*Id.*) Petitioner continued to follow up with his primary care physician throughout the rest of the year. (Ex. P2, pp. 44-62.)

Petitioner's next follow up appointment with Dr. Smith was on December 20, 2017. (Ex. P6, p. 90.) Petitioner reported that "he continues to have more good days than bad with function continuing to improve, though in a more limited fashion." (*Id.*) He described difficulty with endurance, despite apparently normal immediate strength, and increased fatigue when walking or standing for extended periods. (*Id.*) Specifically, petitioner was only able to walk about 50 yards and could stand for no more than 5 minutes before needing to rest. (*Id.*) Dr. Smith documented that petitioner had "previously been noted to have a polyradiculopathy superimposed upon his myasthenia gravis, with clinical findings suggestive of the C7 and/or C6 radiculopathy affecting the upper extremities (C7/C8 by EMG)." (*Id.*) He also noted that "[s]econdary to [petitioner's] polyradiculopathy and proximal weakness, our concern about CIDP as a component of his overall process (with additional involvement of the neuromuscular junction) is increasing." (*Id.* at 97.)

On physical exam, petitioner had minimal ptosis in the right side only, mildly reduced range of motion in his lumbar spine, and normal strength in his extremities with the exception of 4+/5 strength in his left ankle evertor. (Ex. P6, pp. 94-95.) Petitioner's reflexes were normal and intact with the exception of reduced reflexes in his triceps, bilaterally. (*Id.* at 95.) His sensory examination, coordination, and gait were normal. (*Id.*) Dr. Smith assessed petitioner with CIDP, MG with exacerbation, bulbar MG, chronic bilateral low back pain without sciatica, cervical radiculopathy at C7, and vertebrobasilar TIAs. (*Id.* at 97.)

Dr. Smith ordered a repeat EMG, which petitioner underwent on January 17, 2018. (Ex. P6, p. 97; Ex. P20, p. 1.) The repeat EMG revealed:

> Inactive, chronic polyradiculopathy, affecting all tested extremities on needle exam. The patient's denervation disease does not appear to have progressed significantly since last visit; there may be some mild evidence of improvement proximally in the upper extremities and thoracic paraspinal muscles.
>
> Nerve conduction studies no longer document evidence of demyelination, with improvement in previously noted slowing of several motor distal latencies and/or conduction velocities.

(Ex. P20, p. 7.) Dr. Smith remarked that these findings were consistent with an "inactive, demyelinating motor polyradiculopathy," and that the possible etiologies remained "broad." (*Id.* at 8.)

Petitioner continued to see his primary care physician throughout 2018. (Ex. P2, pp. 6-44; Ex. P8, pp. 82-95.) On June 14, 2018, petitioner had a follow up appointment with Dr. Smith for fatigable weakness and MG. (Ex. P6, p. 131.) Dr. Smith explained

that petitioner's "neuromuscular junction abnormality has been complicated by the presence of an additional polyradiculopathy, a component of which appears not to have a structural basis." (*Id.*) Since his last encounter, petitioner's insurance had denied treatment of his MG with either IVIG or rituximab. (*Id.*) While petitioner noted that his weakness was better controlled after increasing his prednisone dosage, he reported that he continued to experience intermittent weakness, shortness of breath, and paroxysmal nocturnal dyspnea-like episodes. (*Id.*) Additionally, petitioner noted fatigue at the end of the day with episodes of tremulousness in his legs after showering and hand tremors. (*Id.*) Despite evidence that treatment with steroids and Mestinon was "aiding his level of function," petitioner reported that he did not believe CellCept was offering any symptomatic relief for his MG and requested a different medication. (*Id.*) Petitioner's physical exam was consistent with prior exams except for a 6 Hz tremor that was previously described as an essential tremor. (*Id.* at 136.) Dr. Smith recommended reassessing petitioner's acetylcholine receptor and MuSK antibody titers. (*Id.* at 131.) Petitioner was assessed in pertinent part with acetylcholine receptor antibody double negative bulbar MG, lumbosacral radiculitis, CIDP, cervical radiculopathy at C7, bilateral lumbar radiculopathy, and shift work sleep disorder. (*Id.* at 140.) Dr. Smith noted that a significant portion of the appointment was spent discussing whether petitioner's polyradiculopathy was playing a role in his fatigable weakness. (*Id.*)

Petitioner continued to see his primary care provider throughout 2019. (Ex. P8, pp. 45-82; Ex. P11, pp. 150-170.) Additionally, petitioner underwent physical therapy from January 2019 to February 2019 and from June 2019 to August 2019 to improve his generalized weakness. (*See* Ex. P7.)

Petitioner's next follow up appointment with Dr. Smith was on March 20, 2019. (Ex. P29, p. 374.) In documenting petitioner's history, Dr. Smith noted that petitioner was previously diagnosed with acetylcholine receptor and MuSK antibody negative MG, although petitioner's clinical symptoms were more typical for acetylcholine receptor antibody positive disease. (*Id.*) He also noted that petitioner had "evidence on EMG of a superimposed neuropathic process, initially recognized as a demyelinating polyradiculoneuropathy." (*Id.*) Based on repeat EMG findings, Dr. Smith explained that petitioner's polyradiculoneuropathy had been stable on Mestinon, CellCept, and prednisone. (*Id.*) However, petitioner's fatigable weakness worsened, and he was started on methotrexate to help stabilize his motor function. (*Id.*) At the time of the encounter, petitioner reported that he was still experiencing significant problems with fatigable weakness, noting that he typically was unable to function after a day of work, as well as episodes of blurred vision and shortness of breath. (*Id.*) Petitioner's shortness of breath primarily occurred at night when he was lying flat and resolved after several minutes of standing. (*Id.*) That said, Dr. Smith also noted that petitioner experienced shortness of breath almost exclusively on days that he does not take prednisone, suggesting that he may be experiencing diaphragmatic weakness when his medication wears off. (*Id.*) Additionally, petitioner's visual blurring would be present when he woke up in the morning prior to taking his first dose of medication. (*Id.*) Dr. Smith noted that petitioner's undiagnosed sleep apnea was superimposed on these two issues. (*Id.*) Petitioner also reported experiencing radicular and allodynic pain affecting

his shoulders and neck, as well as occasional difficulty maintaining a grip on objects. (*Id.*) Dr. Smith documented that petitioner was suffering from distal peripheral neuropathy and noted that his severe allodynic pain occurred mainly at night limiting petitioner's sleep. (*Id.*) Finally, Dr. Smith noted that petitioner was also suffering from "shiftwork disorder with mental clouding" and experienced incomplete improvement with medication. (*Id.*)

Based on these findings, Dr. Smith concluded that (1) petitioner still had substantial problems with his MG, and that the condition was not yet adequately controlled on the days that he takes prednisone; (2) that petitioner's radiculopathy and distal, painful neuropathy were both negatively impacting his life and affecting his ability to complete activities of daily living; and (3) that petitioner's shift work sleep disorder, presumably superimposed on his other problems, was making it more difficult for petitioner to function throughout the day. (Ex. P29, p. 374.) On physical exam, petitioner had minimal ptosis on the right side only, a 6 Hz tremor, slightly decreased strength in the hip flexors and left ankle evertor, and reduced reflexes in the triceps, knees, and ankles. (*Id.* at 379-80.) Petitioner's sensory examination was abnormal with decreased pinprick sensation in the bilateral upper and lower extremities and decreased vibration sensation in the bilateral lower extremities. (*Id.* at 381.) Additionally, petitioner had a positive Baylor and lateral Adson's tests, bilaterally. (*Id.* at 380.) Although his request for IVIG treatment for petitioner was denied the year prior, Dr. Smith planned to request IVIG again. (*Id.* at 375, 384.) Petitioner began his IVIG treatment in June of 2019. (*See* Ex. P18.)

Petitioner began seeing pulmonologist Sujatha Goli, M.D., on August 15, 2019, for evaluation of symptoms related to his MG, including shortness of breath and choking, as well as treatment for sleep apnea. (Ex. P15, p. 1.) Dr. Goli ordered a pulmonary function test, arterial blood gas test, a chest x-ray, and a swallow study. (*Id.* at 2.) Petitioner's labs and chest x-ray were largely unremarkable, and the swallow study did not reveal any evidence of aspirations. (*Id.* at 4.) However, a home sleep study showed severe sleep apnea. (*Id.* at 4, 12.)

On September 22, 2019, petitioner was admitted to the hospital after presenting to the emergency department with reports of a sudden onset of palpitations, which he described as feeling like his heart was racing, and associated shortness of breath. (Ex. P12, pp. 6, 8.) What was likely a new onset of atrial fibrillation was detected, and petitioner was treated with a Cardizem drip. (*Id.*) He was discharged on September 24, 2019. (*Id.* at 6.)

Petitioner followed up with Dr. Smith on May 28, 2020. (Ex. P28, p. 438.) Dr. Smith summarized petitioner's history as "acetylcholine receptor antibody negative, [M]u[SK] antibody negative, LR P4 antibody? [MG] (with clinical symptoms more typical for acetylcholine receptor antibody positive disease) and evidence o[n] EMG of a superimposed neuropathic process, initially recognized as a demyelinating polyradiculoneuropathy." (*Id.*) He noted that petitioner was responding well to IVIG and had experienced an improvement in overall motor function; however, despite petitioner's

improvement with IVIG therapy, Dr. Smith documented that petitioner continued to take Mestinon, methotrexate, and steroids, in combination with IVIG. (*Id.*) Additionally, Dr. Smith explained that petitioner had developed acute lower back pain on January 27, 2020, and had participated in aquatic therapy through March of 2020. (*Id.*) At the time of the visit, petitioner reported that his "back pain is still relatively severe and continues to be a problem," as well as some residual balance problems. (*Id.*)

At this encounter, Dr. Smith assessed petitioner with MG and CIDP, among other conditions, such as lumbosacral radiculitis, bilateral lumbar radiculopathy, chronic bilateral low back pain, cervical radiculopathy at C7, and shift work sleep disorder. (Ex. P28, p. 450.) He documented that petitioner "has multiple problems, caused by different diseases." (*Id.*) Dr. Smith noted that petitioner's MG was finally improving with IVIG therapy but indicated that petitioner needed to increase the frequency of his treatments to maintain better control. (*Id.*) Additionally, Dr. Smith remarked that while treatment with steroids had helped petitioner control his neuromuscular disorder symptoms, his distal symmetric polyneuropathy had worsened with steroid use. (*Id.*) Further, Dr. Smith documented that "[t]he fact that his previous neuropathic findings in ulnar and median nerve is actually improved on immunomodulatory therapy has suggested that his EMG quantitated and clinically identified motor neuropathic findings [that] are likely inflammatory in nature." (*Id.*) However, he noted that a marker antibody to explain petitioner's myasthenic signs and symptoms had not yet been detected. (*Id.*) Dr. Smith instructed petitioner to determine if his insurance would cover testing for LRP4 antibodies and indicated that petitioner may still need a cell-based assay for acetylcholine receptor antibody testing. (*Id.*)

To address petitioner's low back pain, Dr. Smith ordered an MRI of the lumbosacral spine. (Ex. P28, pp. 450-51.) He also noted that petitioner had not experienced further episodes of left neck pain. (*Id.* at 451.) Petitioner was instructed to consider restarting aquatic therapy and advised on back exercises for older individuals with radiculopathies that he could do at home. (*Id.*) Petitioner began physical therapy in September of 2020 and continued to pursue treatment until September 2023. (Ex. P27; Ex. P31.) During some of these visits, petitioner reported being diagnosed with Guillain-Barré Syndrome ("GBS"). (Ex. P31.)

On November 9, 2020, petitioner underwent an MRI of the lumbar spine, which revealed "no acute osseous lesion," normal spinal contents and paraspinal structures, "[m]oderate compromise of the spinal canal at L3-4 [that] is secondary to ligamentous hypertrophy and spondylotic change without disc herniation or compromise of the neural foramina," and "[m]oderately severe compromise of the spinal canal at L4-5 . . . due to ligamentous hypertrophy, spondylotic change and a central, 7 mm disc herniation which compresses the thecal sac and does not affect the neural foramina." (Ex. P28, pp. 453-54.) Petitioner had another follow up appointment with Dr. Smith on November 19, 2020. (*Id.* at 423.) Dr. Smith reviewed petitioner's MRI and explained that petitioner's "newest clinical symptoms are suggestive of lumbosacral stenosis with neurogenic claudication." (*Id.*) Petitioner continued to report back pain and fatigable weakness upon walking and standing. (*Id.*) Dr. Smith suggested that, "[g]iven that his aquatic

therapy only provided transient benefits, and that his white count [when] most recently checked was elevated with mild left shift, assessment by his [primary care physician] would be appropriate to verify lack of an infectious process coincident with his current low back symptoms." (*Id.*) Additionally, Dr. Smith noted that surgical intervention was likely necessary, given the severity of his lumbosacral canal stenosis. (*Id.* at 423, 436-37.) While petitioner could undergo a repeat EMG, Dr. Smith advised that petitioner's canal stenosis would be affecting multiple roots, and thus the EMG would basically just reveal worsening of his polyradiculopathy. (*Id.* at 437.) With respect to petitioner's MG, Dr. Smith documented that petitioner's condition was well-controlled after increasing the frequency of his IVIG treatments and expressed his intent to pursue LRP4 antibody testing if covered by petitioner's insurance. (*Id.* at 427, 436.)

Petitioner's next follow up appointment was on April 1, 2021. (Ex. P28, p. 708.) Dr. Smith reported that petitioner had "symptoms of CIDP, but additional fatigable weakness complaints previously diagnosed as acetylcholine receptor antibody negative, [M]u[SK] negative, LRP4 [antibody]? [MG]." (*Id.*) He explained that petitioner continued to be treated with steroids, methotrexate, Mestinon, and IVIG. (*Id.*) Dr. Smith continued to stress that petitioner needed to undergo LRP4 antibody testing, if possible. (*Id.* at 721.) He also indicated that petitioner would need to continue with his current therapy for treatment of CIDP. (*Id.*)

On September 13, 2021, petitioner returned to Dr. Smith. (Ex. P28, p. 832.) Petitioner reported experiencing significant life stressors since his last visit, including his wife being ill and his daughter unexpectedly passing away. (*Id.*) He indicated that he had to take a few days off in early June after experiencing a flare of dyspnea, weakness, and fatigue. (*Id.*) Additionally, petitioner reported that he resumed aquatic therapy in mid-June, but that he had to end at least one session early due to weakness, and that his physical therapy evaluation confirmed significant leg weakness. (*Id.*) Petitioner's diltiazem dose was reduced, which helped with the dyspnea but did not improve his weakness. (*Id.*) However, at the time of the encounter, petitioner reported that he was feeling stronger. (*Id.* at 833.) Dr. Smith documented that petitioner's "[l]ast EMG actually suggested stabilization of the disease and possible slight improvement at least in demyelinating features. However, if he fails to improve, we will need to readdress at least markers that are potentially involved in this process." (*Id.* at 832.) He noted that LRP4 antibody testing should be more affordable as it was now available through multiple laboratories. (*Id.*) No diagnosis was listed for this appointment; however, it was noted that petitioner would be reevaluated in March of 2022. (*Id.* at 846.)

Petitioner saw Dr. Smith again on March 14, 2022. (Ex. P28, p. 1012.) Dr. Smith documented that petitioner had

> fatiguable weakness associated with radicular pain and other problems suggestive of overwork, previously followed by Dr. Sadeghi for myasthenia gravis, which she diagnosed in 2016. On [Mestinon], the patient has noted improvement in fatigable weakness and diplopia. However, he has been

> negative for any markers of myasthenia gravis. Further, EMG-tested in 2016-showed no evidence of decrement on repetitive stimulation. In fact, there was no evidence of neuropathy, although there was a polyradiculopathy noted on testing. Subsequent studies in 2017 [and] 2018 did not show any significant progression in neuro changes, again only documenting evidence of a polyradiculopathy.

(*Id.*) With respect to petitioner's underlying disease, Dr. Smith explained that petitioner "does not meet criteria" for either MG or CIDP, but noted that petitioner has "a polyradiculopathy and fatigable weakness that appears to respond to IVIG." (*Id.*) He noted that petitioner had not experienced any significant worsening in his strength except in the most proximal muscles. (*Id.* at 1026.) While possibly indicative of a progression of petitioner's underlying disease, Dr. Smith documented that petitioner experienced a worsening in clinical symptoms around the time he became due for his next IVIG treatment, which suggested that any progression was likely due to the treatment wearing off. (*Id.* at 1012, 1026.) However, petitioner declined to increase the frequency of his IVIG treatments and indicated his preference to increase his Mestinon dose as needed in the event of worsening fatigue. (*Id.* at 1012.) Dr. Smith listed MG, CIDP, neurogenic claudication due to lumbar spinal stenosis, bilateral lumbar radiculopathy, cervical radiculopathy at C7, and shift work sleep diagnoses among the diagnoses for the encounter. (*Id.* at 1026.)

On October 31, 2022, petitioner had his next follow up appointment with Dr. Smith for "CIDP MG overlap," as well as fatigable weakness associated with radicular pain and other problems suggestive of overwork. (Ex. P29, p. 126.) Petitioner reported occasionally feeling weak before his IVIG infusion, but his symptoms were otherwise essentially stable and controlled on Mestinon, methotrexate, and IVIG. (*Id.* at 126-27, 141.) Petitioner also reported that he occasionally experiences "right hand tingling and 'shock' like pain from the wrist down." (*Id.* at 127.) Dr. Smith noted that these symptoms were consistent with a right median neuropathy, specifically carpal tunnel syndrome. (*Id.* at 126, 141.) Petitioner's positive right Phalen's sign and prior EMG also supported a finding of carpal tunnel syndrome. (*Id.*) Given the mildness of his symptoms, petitioner was advised to treat conservatively with acetaminophen and a splint. (*Id.*) MG and CIDP were listed as petitioner's diagnoses for the encounter. (*Id.* at 141.)

Petitioner's next appointment with Dr. Smith was on March 27, 2023, for fatigable weakness and CIDP MG overlap. (Ex. P28, p. 1479.) In recording petitioner's history, Dr. Smtih continued to stress that, despite his prior MG diagnosis and improvement on Mestinon, petitioner had tested negative for antibody markers for MG and his EMG showed no evidence of decrement on repetitive stimulation. (*Id.*) However, Dr. Smith noted that petitioner's Mestinon "may not have been completely 'out of his system' with his ingestion of the medication within 24 hours of his [EMG] testing." (*Id.*) He also documented that petitioner's polyradiculopathy and evidence of demyelination revealed by his initial EMG had somewhat improved by the time of his most recent needle exam assessment in early 2018. (*Id.*) Petitioner continued to report experiencing weakness

prior to his next IVIG therapy session. (*Id.*) Accordingly, Dr. Smith concluded that, "[a]t present, his polyradiculopathy with fatigable weakness is therefore adequately but incompletely controlled by current IVIG treatments." (*Id.*) Additionally, Dr. Smith noted that once petitioner started therapy, petitioner's "demyelinating polyneuropathy 'resolved' with normalization of the previously demyelinating neuropathic features, indicating improvement on therapy." (*Id.*) Petitioner again refused to increase the frequency of his IVIG therapy. (*Id.*) CIDP, MG, bilateral lumbar radiculopathy, neurogenic claudication due to lumbar spinal stenosis, sensory polyneuropathy, and shift work sleep disorder were listed among petitioner's diagnoses for the encounter. (*Id.* at 1493.)

In April of 2023, petitioner presented for chiropractic care, with major complaints of cervical, thoracic, and lumbo pelvic pain, which petitioner indicated first occurred after his flu vaccination in 2016. (Ex. P32, p. 3.) Petitioner reported that he was subsequently diagnosed with GBS, MG, and CIDP. (*Id.*) He explained that he experiences periodic flares of varying intensity that last anywhere from a day to a week. (*Id.*) Petitioner described his discomfort as an ache in the cervical, thoracic, and lumbo pelvic areas with tingling in his left hand and a burning sensation in his thighs, as well as weakness in his left arm and thigh. (*Id.*) The chiropractic provider described petitioner's gait as abnormal, noting that he tended to drag his left foot, and observed that he had difficulty breathing. (*Id.*) Petitioner reported that his pain is aggravated by standing for more than hour and is less bothersome when he is seated or laying down. (*Id.*) On physical exam, petitioner's left ilium appeared higher than his right, and his right shoulder was higher than his left. (*Id.*) Additionally, he had reduced cervical range of motion with discomfort during testing, as well as reduced thoraco-lumbar range of motion with discomfort along the entire spine on flexion and pain in the lumbo pelvic, lower thoracic, and scapular areas on lateral flexion. (*Id.*) Petitioner was diagnosed with "[c]hronic severe cervical sprain and strain with associated cervical subluxation complex, accompanied by moderate to severe myalgia and cervicalgia," "[c]hronic severe thoracic sprain and strain with associated thoracic subluxation complex, accompanied by moderate to severe thoracic and costal myalgia and pain;" "[c]hronic severe lumbo pelvic sprain and strain with associated lumbo pelvic subluxation complex, accompanied by severe myalgia and lumbo pelvic pain;" and "[c]hronic mild to moderate sprain/strain in left shoulder and arm with associated upper extremity subluxation complex accompanied by weakness and tingling." (*Id.* at 3-4.) After petitioner initiated regular chiropractic care in November of 2023, he was able to maintain "an overall decrease in his pain levels." (*Id.* at 4.) Petitioner continued chiropractic treatment through June of 2024. (Ex. P32.)

Based on the records submitted in this case, petitioner continued to receive IVIG through April of 2024. (Ex. P30.)

b. Testimony

Petitioner filed a sworn affidavit and provided testimony during the entitlement hearing. (Ex. P9; Tr. 7-34.) Petitioner avers that, prior to the subject vaccination, he

"had no history of neurological disorders." (Ex. P9, ¶ 3.) He maintains that he received the flu vaccine at issue in this case on September 16, 2016. (*Id.* ¶ 4; Tr. 13.)

During the hearing, petitioner provided testimony regarding the nature of his employment at an oil refinery. (Tr. 8-12, 31.) He describes the intense physical demands of his job, explaining, for example, that some tasks involved climbing towers in excess of 200 feet via ladder and noting that he was sometimes required to "work endless days of 12-hour shifts." (*Id.* at 9-12, 31.) Petitioner admits that, prior to receiving the flu vaccination at issue, he suffered from low back pain. (*Id.* at 13-14, 29-34.) He recalls receiving some injections for his low back pain and concedes that he followed up with his primary care provider, Dr. Pickard, for continuing low back pain. (*Id.* at 14, 29-31.) However, petitioner states that he was able to work through it and avers that his low back pain did not prevent him from doing his job or any of his hobbies. (*Id.* at 14, 32-34.)

Petitioner summarizes his medical course as contained in his medical records. (Ex. 9, ¶¶10-28; Tr. 16-28.) He maintains that he started experiencing fatigue and weakness approximately three days after receiving his flu vaccine. (Tr. 14-16.) Petitioner states that he followed up with Dr. Pickard the next day and requested to be tested for Lyme disease as he had spent the prior weekend at his lake house. (*Id.* at 16-17; Ex. 9, ¶ 10.) He recalls his neurology appointment with Dr. Sadeghi on September 21, 2016, stating that he remembers experiencing fatigue, weakness, blurred vision, trouble swallowing, slurred speech, tingling from his head to his foot, and left sided pain at the time of that encounter. (Tr. 18; Ex. 9, ¶ 11.) Petitioner avers that prior to receiving his flu vaccine on September 16, 2016, he had never experienced blurred or double vision, difficulty speaking or swallowing, or tingling from his face down to his foot. (Tr. 18-19, 21-22.) Dr. Sadeghi prescribed Mestinon and ordered blood tests for myasthenia gravis ("MG"), which came back negative. (*Id.* at 23.) After becoming frustrated with Dr. Sadeghi's office, petitioner explains that he sought a second opinion from Dr. Smith in November of 2016. (*Id.* at 24.) Petitioner avers that Dr. Smith eventually diagnosed him with polyradiculopathy. (*Id.* at 24-26.) However, he also states that Dr. Smith thought petitioner had MG, kept him on the treatment, and tested him for MG again, which came back negative. (*Id.* at 25.) Throughout the course of his care, Dr. Smith continued to prescribe petitioner Mestinon and started him on prednisone, CellCept, physical therapy, and eventually IVIG treatment. (Ex. 9, ¶¶ 19, 22; Tr. 26.) As of the date of the hearing, he was still taking Mestinon and prednisone, undergoing IVIG therapy, and attending physical therapy. (Tr. 26-28.) He avers that prior to his flu vaccine, he had not taken prednisone or any other steroids and had never undergone IVIG treatment. (*Id.* at 27-28.)

Petitioner describes how he continues to suffer from constant and severe pain, fatigue, weakness, and numbness. (Ex. 9, ¶ 29.) These symptoms have limited his ability to walk, work, perform many activities of daily living, and participate in many activities he previously enjoyed, such as fishing and hunting. (*Id.* ¶¶ 29-30.)

IV. Expert Opinions and Qualifications[5]

a. Petitioner's Expert, Lawrence Steinman, M.D.[6]

Dr. Steinman submitted three expert reports and testified during the entitlement hearing. (Exs. P24-P26; Tr. 35-122, 321-33.) He was proffered, without objection, as an expert in neurology and neuroimmunology. (Tr. 6-7, 38.)

According to Dr. Steinman, the three most likely diagnoses considered by petitioner's treating physicians' included MG, CIDP, and inflammatory polyradiculopathy. (Ex. P24, p. 6.) However, Dr. Steinman notes that he and Dr. Price agree that petitioner does not suffer from CIDP. (Ex. P24, p. 6; Ex. P26, p. 2; Tr. 102.) Therefore, the two competing diagnoses in this case are MG and inflammatory polyradiculopathy. (Tr. 44-45.) Dr. Steinman states that it is possible for inflammatory polyradiculopathy and MG to exist as comorbid diagnoses in rare cases. (Ex. P24, pp. 1, 6 (citing Saskia Bolz et al., *CIDP, Myasthenia Gravis, and Membranous Glomerulonephritis – Three Autoimmune Disorders in One Patient: A Case Report*, 18 BMC NEUROLOGY 1 (2018) (Ex. P24, Tab 4)); *see also* Tr. 119-20.) However, he maintains that in this case, it is more likely than not that petitioner suffers from one or the other, rather than both conditions. (Tr. 119-20.) While he concedes that a diagnosis of MG cannot be definitively ruled out and notes that this is a complex case, Dr. Steinman opines that inflammatory polyradiculopathy is more likely than not the correct diagnosis in this case. (Tr. 44-45; Ex. P24, pp. 1, 6; Ex. P26, p. 2.)

Dr. Steinman contends that petitioner does not suffer from MG. (Tr. 46; Ex. P24, p. 1.) To support his opinion, he points to petitioner's antibody and electrodiagnostic testing results. (Tr. 45, 100.) He stresses that petitioner tested negative for the acetylcholine receptor and muscle-specific kinases ("MuSK") antibodies, which he maintains are "rarely" negative "in people who actually still have myasthenia gravis." (*Id.* at 45.) Additionally, Dr. Steinman emphasizes that petitioner's treating neurologist, Dr. Smith, conducted comprehensive EMG studies which revealed no evidence of decrement on repetitive nerve stimulation. (*Id.* at 54, 100; Ex. P24, p. 6.) While he

[5] Because I have concluded, for the reasons discussed below, that the diagnostic issue presented by the parties is dispositive, summaries of the experts' opinions are limited to their views regarding diagnosis and do not address their opinions relative to whether the flu vaccine at issue in this case can or did cause an inflammatory polyradiculopathy.

[6] Dr. Lawrence Steinman received his medical degree from Harvard University. (Ex. P24, Tab 1, p. 1.) He completed a surgery internship and two residencies, one in pediatrics and one in pediatric and adult neurology, at Stanford University Hospital. (*Id.*; Tr. 36.) He also completed fellowships at the Weizmann Institute of Science, the Aharon Katzir-Katchalsky Center, and the National Institutes of Health. (Ex. P24, Tab 1, p. 1.) He currently works at Stanford University as a professor of Neurology and Neurological Sciences, Pediatrics, and Genetics. (*Id.*) He is board certified in psychiatry and neurology. (*Id.* at 2.) Dr. Steinman was elected to the Institute of Medicine of the National Academy of Sciences in 2009. (Ex. P24, pp. 2-3; Tr. 40-41.) He has cared for hundreds of patients with neuroinflammatory diseases, including inflammatory polyradiculoneuropathy and MG, and he has written several papers on MG. (Ex. P24, pp. 1-2; Tr. 36.) He has published roughly 600 journal articles. (Ex. P24, Tab 1, pp. 5-49.)

concedes that it is possible for an individual to suffer from "double antibody negative, EMG negative" MG, he opines that he puts "[v]ery low weight" on that possibility in petitioner's case. (Tr. 100.)

While he notes that petitioner does not satisfy the antibody or electrodiagnostic tests to support a diagnosis of MG, Dr. Steinman opines that petitioner "was treated with the appropriate drugs." (Ex. P25, p. 12.) Dr. Steinman acknowledges that petitioner was started on Mestinon, an effective and approved treatment for MG. (Tr. 45.) Although he concedes that Mestinon is not a medication prescribed to treat inflammatory polyradiculopathy, Dr. Steinman emphasizes that Dr. Smith "escalated" petitioner's therapy by prescribing steroids and CellCept to suppress inflammation, which he suggests favors a diagnosis of inflammatory polyradiculopathy. (*Id.* at 45-46.) However, Dr. Steinman acknowledges that medications, such as prednisone and CellCept, would also benefit patients suffering from MG. (*Id.* at 46, 53.) Dr. Steinman argues that the fact that Dr. Smith added four "highly impactful" medications to petitioner's regimen over the course of his treatment indicates that petitioner was not responding well to Mestinon, which cuts against a finding that MG is the most appropriate diagnosis. (*Id.* at 323-24.)

Moreover, Dr. Steinman stresses that Dr. Smith, an expert on neuromuscular disease, "weighed all of the evidence and cast aside a diagnosis of myasthenia gravis in favor of inflammatory polyradiculopathy." (Tr. 45.) He places high value on the opinion of Dr. Smith as the treating physician and agrees with Dr. Smith that inflammatory polyradiculopathy, rather than MG, is the correct diagnosis. (*Id.* at 46, 99.) Specifically, Dr. Steinman opines that "abandoning the diagnosis of myasthenia gravis is the best course of action" in this case. (*Id.* at 53.) While he acknowledges that he cannot point to a record where Dr. Smith explicitly used the word "inflammatory" when diagnosing petitioner with a polyradiculopathy, Dr. Steinman contends that based on the context, particularly his decision to treat petitioner with potent anti-inflammatory medications, such as prednisone and CellCept, Dr. Smith thinks petitioner has an inflammatory polyradiculopathy. (*Id.* at 104-05, 117.) He concedes that Dr. Smith continued to list MG as a diagnosis in the record for petitioner's most recent neurology encounter; however, Dr. Steinman states that he thinks it is very clear that Dr. Smith does not believe petitioner suffers from MG and argues that just because MG is listed as a diagnosis within an electronic medical record does not mean that Dr. Smith endorses MG as an appropriate diagnosis. (*Id.* at 101.)

In addition, Dr. Steinman opines that petitioner does not suffer from CIDP. (Ex. P24, p. 6; Ex. P25, p. 13; Tr. 90, 102, 105, 108.) He explains that petitioner does not have evidence of a demyelinating neuropathy. (Ex. P24, p. 6; Tr. 103.) Specifically, Dr. Steinman stated that petitioner's electrodiagnostic studies did not reveal evidence of demyelination, which would be expected for an individual suffering from CIDP. (Tr. 102, 112.) Again, while he acknowledges that Dr. Smith continues to list CIDP as a diagnosis in petitioner's most recent medical records, Dr. Steinman indicates it is very clear that Dr. Smith does not think petitioner has CIDP. (*Id.* at 56, 101-02, 108.)

According to Dr. Steinman, inflammatory polyradiculopathy has been described with an autoimmune response to a paranodal protein called contactin-1. (Ex. P24, p. 6 (citing Kathrin Doppler et al., *Destruction of Paranodal Architecture in Inflammatory Neuropathy with Anti-Contactin-1 Autoantibodies*, 86 J. NEUROLOGY NEUROSURGERY & PSYCHIATRY 720 (2015) (Ex. P24, Tab 6)); Ex. P26, p. 2.) Additionally, he notes that antibodies to another paranodal protein, neurofascin, have been reported in patients with inflammatory polyradiculopathy. (Ex. P25, pp. 12-13 (citing Atay Vural et al., *Autoantibodies Against the Node of Ranvier in Seropositive Chronic Inflammatory Demyelinating Polyneuropathy: Diagnostic, Pathogenic, and Therapeutic Relevance*, 9 FRONTIERS IMMUNOLOGY 1 (2018) (Ex. P25, Tab 5); Masato Kadoya et al., *IgG4 Anti-Neurofascin155 Antibodies in Chronic Inflammatory Demyelinating Polyradiculoneuropathy: Clinical Significance and Diagnostic Utility of a Conventional Assay*, 301 J. NEUROIMMUNOLOGY 16 (2016) (Ex. P25, Tab 6)); Ex. P26, p. 2.)

Relying on a study by Doppler et al., he explains that contactin-1, an antigen at the node of Ranvier, is targeted in inflammatory polyradiculopathy. (Tr. 62, 66 (discussing Doppler et al., *supra*, at Ex. P24, Tab 6).) He states that inflammatory polyradiculopathy does not involve demyelination because contactin-1 is at the node of Ranvier, not in the myelin. (*Id.* at 103 (discussing Doppler et al., *supra*, at Ex. P24, Tab 6).) Dr. Steinman explains that inflammatory polyradiculopathy involves axonal damage and potentially axonal loss. (*Id.* at 108.) Because contactin-1 is not limited to nerve roots and is found in the central and peripheral nervous systems, Dr. Steinman opines that it is difficult to determine whether inflammatory polyradiculopathy involves a generalized process affecting the whole spine or discrete lesions at various points on the spine. (*Id.* at 116-17.)

In Doppler et al., high reactivity to contactin-1 by enzyme-linked immunosorbent assay ("ELISA") was observed in four patients with chronic inflammatory demyelinating polyradiculopathy and in none of the patients with GBS. (Ex. P24, p. 6 (citing Doppler et al., *supra*, at Ex. P24, Tab 6).) Dr. Steinman notes that the four patients in Doppler et al. presented with a typical clinical picture, namely acute onset of disease and severe motor symptoms, and three patients developed an action tremor. (*Id.* (citing Doppler et al., *supra*, at Ex. P24, Tab 6).) In these four patients, disruption of paranodal architecture was observed via immunofluorescence-labelling of paranodal proteins of dermal myelinated fibers, and histology testing revealed axonal damage with no classical signs of demyelination. (*Id.* (citing Doppler et al., *supra*, at Ex. P24, Tab 6).) Therefore, the authors of the Doppler et al. study concluded that "anti-contactin-1-related neuropathy constitutes a presumably autoantibody-mediated form of inflammatory neuropathy with distinct clinical symptoms and disruption of paranodal architecture as a pathological correlate. Anti-contactin-1-associated neuropathy does not meet morphological criteria of demyelinating neuropathy and therefore, might rather be termed a 'paranodopathy' rather than a subtype of demyelinating inflammatory neuropathy." (*Id.* at 7 (citing Doppler et al., *supra*, at Ex. P24, Tab 6).)

Dr. Steinman opines that the clinical picture of the four patients examined in Doppler et al. is closest to petitioner's clinical picture. (Ex. P24, p. 6; Tr. 116 (citing

Doppler et al., *supra*, at Ex. P24, Tab 6).) He states that the four patients examined in Doppler et al. were found to have a non-demyelinating inflammatory polyradiculopathy. (Tr. 66, 103-05.) Dr. Steinman opines that petitioner did not suffer from demyelination and agrees underlying pathophysiology at petitioner's nerve root is not demyelinating. (*Id.* at 103, 111-13.) Therefore, Dr. Steinman contends the diagnosis in Doppler et al. supports his opinion that inflammatory polyradiculopathy is the diagnosis that fits best in this case. (Ex. P24, p. 6, Tr. 104-05, 116 (citing Doppler et al., *supra*, at Ex. P24, Tab 6).)

Specifically, Dr. Steinman opines that petitioner suffered from a significant aggravation of an inflammatory polyradiculopathy on the weekend he received the flu vaccine. (Ex. P24, p. 6; Tr. 39, 114-15.) He argues that petitioner's symptoms following vaccination, specifically the symptoms in his face, numbness and tingling, and his difficulty swallowing and speaking, are more likely than not a manifestation of a new radiculopathy that developed after he received the flu vaccination at issue. (Tr. 50.) Dr. Steinman explains that petitioner's preexisting radiculopathy seemed amplified post-vaccination with new areas of the nervous system, specifically cervical and bulbar nerve roots, being implicated. (*Id.* at 43-44, 50.)

Although Dr. Steinman concedes that he would expect an individual suffering from inflammatory polyradiculopathy to have elevated cerebrospinal fluid ("CSF") protein, he explains that petitioner was on anti-inflammatories at the time the CSF testing was completed which could have kept his CSF albumin levels within normal range. (Tr. 117-18.) He argues that all of the symptoms that petitioner presented with post-vaccination can be explained by inflammatory polyradiculopathy. (*Id.* at 118.) Furthermore, Dr. Steinman opines that the EMG and nerve conduction study results and the antibody testing results reinforce his preferred diagnosis of inflammatory polyradiculopathy because these tests are negative for CIDP and MG. (*Id.* at 111-12, 118-19.) Accordingly, he agrees that the diagnosis of inflammatory polyradiculopathy is arrived at in this case by process of elimination. (*Id.* at 118.) Additionally, Dr. Steinman agrees that inflammatory polyradiculopathy is an umbrella term for a variety of different conditions, such as CIDP and GBS, as opposed to a specific clinical entity with strict diagnostic criteria. (*Id.* at 115-16.)

While the medical records indicate that petitioner suffered from lumbar radiculopathy prior to receiving the flu vaccine at issue, Dr. Steinman emphasizes that there is no reference to any cervical radiculopathy in the pre-vaccination records. (Tr. 42-43.) He places the onset of petitioner's nerve pain and dysfunction in his cervical spine after the flu vaccination at issue. (*Id.* at 43.) Dr. Steinman distinguishes petitioner's preexisting lumbar radiculopathy from his post-vaccination clinical presentation, stating that the anatomy of petitioner's preexisting radiculopathy was confined to the lumbar area and extended, at most, into the thoracic area with no evidence of cervical or bulbar involvement. (*Id.* at 43-44, 49-50.) He notes that petitioner's lumbar radiculopathy primarily manifested as back pain, and that petitioner's pre-vaccination records do not reference many of the symptoms that petitioner complained of after vaccination, including tingling in his face and double vision. (*Id.* at

44.) While acknowledging that petitioner had fatigue prior to vaccination, Dr. Steinman contends that petitioner complained of fatigue "much more vigorously" post-vaccination. (*Id.* at 43-44.)

Dr. Steinman disagrees with Dr. Price's opinion that the evidence of chronic polyradiculopathies observed on petitioner's first EMG indicates that petitioner sustained a nerve root axonal injury prior to receiving the flu vaccination at issue, with some of the lumbar radiculopathies being secondary to his preexisting low back pain experienced in late 2014. (Ex. P25, pp. 12-13; Tr. 47-51.) He fails to understand how Dr. Price can assert that petitioner's EMG could pinpoint pathology back to two years prior. (Ex. P25, p. 13; Tr. 51.) Dr. Steinman explains that an EMG captures one point in time. (Tr. 49.) Accordingly, there is no way to substantiate Dr. Price's theory that, had petitioner undergone an EMG in late 2014 or early 2015, it would have demonstrated chronic polyradiculopathies with axon loss. (*Id.*) Dr. Steinman stresses petitioner's complaints following vaccination go above the lumbar and thoracic areas, while his complaints pre-vaccination never went above his low back. (*Id.* at 49, 52.) Specifically, he states that post-vaccination, petitioner had complaints of new symptoms, including blurry vision, double vision, difficulty swallowing, and numbness and tingling in the face and arm, and contends that these symptoms could not be attributed to a lumbar radiculopathy. (*Id.* at 52; Ex. P25, p. 13; Ex. P26, p. 1.)

b. Respondent's Expert, Raymond S. Price, M.D.[7]

Dr. Price submitted two expert reports and provided testimony during the entitlement hearing in this case. (Exs. A, E; Tr. 123-273.) He was proffered as an expert in neurology without objection. (Tr. 6-7.)

Dr. Price provided an extensive review of petitioner's medical course. (Ex. A, pp. 2-9.) He notes that petitioner's pre-vaccination medical history was significant for chronic low back pain with a lumbar radiculopathy. (Ex. A, p. 2; Tr. 140-41, 249-50.) Specifically, Dr. Price stresses petitioner's encounter with his primary care doctor in December of 2014, at which time petitioner reported that his back pain had not improved with pain medication and anti-inflammatories, complained of numbness in his right thigh causing his leg to give out, and noted that he could not walk for more than 100 feet without stopping and was thus not able to work. (Tr. 140-41 (discussing Ex. 8, p. 252).) He opines that petitioner's reported history and complaints are certainly

[7] Dr. Raymond S. Price received his medical degree from the University of Pennsylvania in 2004, before going on to complete an internship in internal medicine, a residency in neurology, and a fellowship in clinical neurophysiology with a neuromuscular focus at the University of Pennsylvania. (Ex. B, p. 1.) He is board certified in psychiatry and neurology, electrodiagnostic medicine, and neuromuscular medicine, and he maintains an active medical license in Pennsylvania. (*Id.* at 2.) He currently works as an associate professor in Clinical Neurology at the University of Pennsylvania School of Medicine, and as both the Neurology Residency Director and the Neurohospitalist Division Co-Director in the Department of Neurology at the University of Pennsylvania. (*Id.* at 1.) He has published 16 peer-reviewed research articles and reviews; 41 editorials, reviews, and chapters; and one book. (*Id.* at 11-15.) Additionally, he has cared for hundreds of patients with radiculopathies, neuropathies, and neuromuscular junction diseases, including patients with both CIDP and MG. (Ex. A, p. 1.)

suggestive of underlying lumbar spine disease. (*Id.* at 140.) Given the intense and physically demanding nature of petitioner's job and his reported symptomology, Dr. Price opines "[i]t's more likely than not by 99.9 percent that" petitioner's chronic back pain, leg numbness, and difficulty walking were "secondary to spine degenerative changes as opposed to some latent inflammatory polyradiculopathy." (*Id.* at 140-41.) Additionally, Dr. Price emphasizes that later records indicate that petitioner's chronic back pain with radicular pain improved with physical therapy and chiropractic maneuvers, which are treatments very commonly used to treat degenerative spine disease but are not treatments for a preexisting inflammatory polyradiculopathy. (*Id.* at 153, 155-56 (discussing Ex. P2, p. 192).)

Like Dr. Steinman, Dr. Price addresses the three diagnoses potentially at issue: MG, CIDP, and inflammatory polyradiculopathy. (Ex. A, pp. 9-13.) However, he opines that only MG is implicated in this case. (*Id*.) He explains that autoimmune MG[8] is a disease affecting the neuromuscular junction. (Ex. A, p. 9; Tr. 127.) Dr. Price explains that MG can be classified based on the presenting clinical symptoms, noting that ocular MG symptoms are localized to the eye whereas generalized MG can impact the eye, as well as other muscular components in the body, such as the throat and extremities. (Tr. 127-28.) Additionally, MG can be subdivided based on the presence or absence of certain antibodies. (*Id.* at 128; *see also* Ex. A, p. 9.) Most patients affected by MG have antibodies against the acetylcholine receptor or the muscle-specific tyrosine kinase ("MuSK"); however, some individuals have seronegative MG, meaning they test negative for both known antibodies. (Ex. A, p. 9 (citing Emma Ciafaloni, *Myasthenia Gravis and Congenital Myasthenic Syndromes*, 25 CONTINUUM: MUSCLE & NEUROMUSCULAR DISORDERS 1767 (2019) (Ex. A, Tab 1)); Tr. 128-29, 132-33.) Therefore, the fact that an individual may test negative for a "known" MG antibody does not rule out the possibility of autoimmune MG. (Tr. 129.)

Ptosis, frequently asymmetric, and binocular diplopia are the most common presenting symptoms of MG. (Ex. A, p. 9 (citing Ciafaloni, *supra*, at Ex. A, Tab 1); *see also* Tr. 130.) Other classic symptoms of generalized MG include blurred vision; dysphagia; jaw closure weakness; facial weakness; speech that is slurred, nasal, or of reduced volume; and weakness of big muscles. (Ex. A, p. 9 (citing Ciafaloni, *supra*, at Ex. A, Tab 1); Tr. 130-31.) Dr. Price emphasizes that MG typically does not involve weakness in distal muscles, such as muscles in the hands, fingers, toes, and feet. (Tr. 131.) Rather, MG typically involves weakness in proximal muscles and tends to be localized to the shoulder and pelvic area. (*Id.*) Limb weakness often results in difficulty performing tasks, such as getting up from a seated position, walking prolonged distances, climbing stairs, and tasks that involve raising the arms above the head. (Ex. A, p. 9 (citing Ciafalonia, *supra*, at Ex. A, Tab 1); Tr. 131.)

[8] Dr. Price explained that in addition to autoimmune myasthenia gravis, there is also a genetic myasthenia referred to as congenital myasthenic syndrome, which is not associated with antibodies. (Tr. 128-29.) However, congenital myasthenic syndrome is not at issue in this case. (Tr. 127.) Therefore, all references to myasthenia gravis ("MG") in this decision refer to autoimmune MG.

Fluctuating and fatigable weakness of muscle groups that worsens with activity and improves with rest is the core clinical feature of MG. (Ex. A, p. 9 (citing Ciafaloni, *supra*, at Ex. A, Tab 1); Tr. 135-36.) Therefore, it is very common for individuals with MG to describe their symptoms getting worse at the end of the day. (Tr. 135.) Additionally, similar to individuals with other neurologic conditions such as CIDP, patients with MG can have a "relapsing-remitting course," meaning they can experience flare ups where their symptoms are worse or severe and periods where they experience low to no symptomology. (*Id.* at 136.)

Dr. Price explains that there are various approaches to diagnosing an individual with MG. (Tr. 131-35.) One method involves placing patients on an acetylcholinesterase inhibitor, such as Mestinon,[9] which is a medication that blocks the breakdown of acetylcholine in the neuromuscular junction. (*Id.* at 131-32.) Improvement on an acetylcholinesterase inhibitor is highly suggestive of a neuromuscular disease, such as MG. (*Id.* at 132.) Another method is serologic testing, which involves testing patients for known antibodies. (*Id.*) Approximately 70-80% of individuals with generalized MG will test positive for an acetylcholine receptor antibody. (*Id.*) Of the population of patients with generalized MG who are negative for the acetylcholine receptor antibody, around 5-10% are positive for the MuSK antibody. (*Id.*) However, approximately 15-20% of individuals with generalized MG test negative for both known antibodies, and are found to have seronegative MG. (*Id.* at 129, 133.) In the case of seronegative MG, Dr. Price notes that some physicians hypothesize that those cases involve different antibodies, such as the low-density lipoprotein c-receptor related protein 4 ("LRP4"). (Ex. A, p. 9; Tr. 133.) Providers can also try to diagnose generalized MG electrophysiologically via repetitive nerve stimulation. (Tr. 133.) If a reduction in the amplitude ("decrement") is observed when you repetitively stimulate the nerve within one second, that is suggestive of a neuromuscular disease, such as MG. (*Id.*) However, Dr. Price emphasizes that a decrement on repetitive nerve stimulation is only observed in approximately 60-70% of patients with MG. (*Id.* at 133-34, 168.) Lastly, providers can perform a single-fiber EMG of a weak muscle, which is the most sensitive diagnostic tool used to confirm the diagnosis of MG and is positive in approximately 97% of affected patients. (Ex. A, p. 9 (citing Ciafalonia, *supra*, at Ex. A, Tab 1)); Tr. 134, 222.) If a single-fiber EMG is normal in a clinically affected muscle, the diagnosis of MG is excluded. (Ex. A, p. 9 (citing Ciafalonia, *supra*, at Ex. A, Tab 1)); Tr. 134.) Dr. Price stresses that in his practice, it is not uncommon for him to see patients who are seronegative with no evidence of decrement on EMG and are only found to have MG based on a single-fiber EMG. (Tr. 134-35.)

In this case, Dr. Price opines that petitioner was correctly diagnosed with MG. (Ex. A, p. 9-11.) He emphasizes that when petitioner presented for his first neurology encounter on September 21, 2016, he reported symptoms consistent with generalized

[9] Mestinon is brand name preparation of pyridostigmine bromide, which acts by inhibiting destruction of acetylcholine to facilitate transmission of impulses across the neuromuscular junction and is used for symptomatic treatment of myasthenia gravis. *Mestinon*, DORLAND'S MEDICAL DICTIONARY ONLINE, https://www.dorlandsonline.com/dorland/definition?id=30704 (last visited Dec. 10, 2025); *Pyridostigmine bromide*, DORLAND'S MEDICAL DICTIONARY ONLINE, https://www.dorlandsonline.com/dorland/definition?id=42398 (last visited Dec. 10, 2025).

MG, including fatigue, difficulty with swallowing and speech, and episodic blurred vision that is associated with fatigue and happens at night. (Tr. 141-44; Ex. A, pp. 9-10 (discussing Ex. P3, p. 5).) Additionally, Dr. Price stresses that petitioner's physical examination revealed nasal, slurred speech and mild fatigability of the right upper extremity with repetitive effort despite normal strength, which are findings also consistent with generalized MG. (Tr. 144-45; Ex. A, pp. 9-10 (discussing Ex. P3, p. 6).) At that encounter, Dr. Sadeghi noted that petitioner reported that "[h]e was started on testosterone injections which did help with fatigue until 6 months ago when he noticed worsening fatigue then new onset blurred vision." (Ex. P3, pp. 5, 7.) Dr. Price opines that this documentation places the onset of petitioner's worsening fatigue and episodic blurred vision before September 16, 2016, which suggests that petitioner developed generalized MG prior to receiving the flu vaccination at issue. (Ex. A, p. 10; Tr. 142-44.)

Dr. Price concedes that some of the symptoms that petitioner reported at this encounter cannot be attributed to MG. (Ex. A, pp. 9, 11.) These symptoms include pain when lifting heavy weights, pain when walking more than half of a mile, left-sided burning pain, and intermittent numbness of the left face, arm, and leg. (*Id.* at 9-10.) However, Dr. Price attributes some of these symptoms to petitioner's preexisting lumbar radiculopathy. (*Id.* at 10.) Moreover, Dr. Price stresses that petitioner's sensory examination was normal, revealing no objective evidence of his reported sensory symptoms of facial tremor and numbness in his hands and feet. (Tr. 144-45.)

At his follow up appointment with Dr. Sadeghi on October 6, 2016, petitioner reported significant improvement in his symptoms since starting Mestinon, noting that he does not feel as fatigued and denying further episodes of double vision and ptosis. (Ex. A, p. 10; Tr. 148-49 (discussing Ex. P3, p. 46).) Diplopia and fatigability upon sustained upward gaze were observed on physical examination. (Ex. A, p. 10; Tr. 148-50 (discussing Ex. P3, p. 47).) These symptoms are all consistent with MG. (Ex. A, p. 10; Tr. 150.) Dr. Price states that petitioner's response to Mestinon is exactly what he would expect to see in a patient with MG. (Ex. A, p. 10; Tr. 148-49, 230.) He notes that petitioner reported taking more Mestinon when working long shifts to counterbalance the fact that his symptoms worsen as the day progresses. (Tr. 150.) Again, Dr. Price opines that this fluctuation in symptoms and fatigability as the day progresses is indicative of MG. (*Id.*) He explains that Mestinon is a medication that is specific to symptomatic treatment of MG, and stresses that "[i]t is not used in the treatment of virtually any other neurologic conditions." (*Id.* at 149; *see also id.* at 157, 217-18.) Specifically, Dr. Price emphasizes that Mestinon is not used to treat inflammatory polyradiculopathy or CIDP, and that he would not expect to see an improvement in a patient suffering from those conditions on Mestinon alone. (*Id.* at 149, 151.)

When petitioner first presented to Dr. Smith on November 28, 2016, he reported a history of symptoms consistent with generalized MG, including bilateral ptosis, choking on foods and liquids, neck weakness, weakness in his arms and legs with activity, shortness of breath, and diplopia. (Ex. A, p. 10; Tr. 153-56 (discussing Ex. 2, p. 192).) Dr. Price describes this history as "almost textbook for myasthenia gravis." (Tr. 156.) He also notes that petitioner's reported multi-year history of severe fatigue with

overexertion is also consistent with MG, which again suggests the possibility that petitioner developed MG prior to the vaccination at issue. (*Id.* at 153, 156.) Dr. Price also concludes that Dr. Smith's finding of bilateral, asymmetrical ptosis on exam is again consistent with MG. (Ex. A, p. 10; Tr. 157.) Moreover, Dr. Price stresses that his opinion that petitioner was correctly diagnosed with MG aligns with the opinions of his treating neurologists, Dr. Sadeghi and Dr. Smith, because MG was consistently listed as a diagnosis throughout the course of petitioner's treatment. (Ex. A, p. 10.)

Dr. Price recognizes that petitioner tested negative for both acetylcholine receptor binding antibody and MuSK antibody. (Ex. A, p. 10; Tr. 151-53 (discussing Ex. 2, pp. 179-80).) However, as Dr. Price explained, 20% of patients with generalized MG are seronegative. (Ex. A, p. 10; Tr. 128-29, 216, 224-25, 266-67.) While he concedes that petitioner's EMG studies were normal with no evidence of decrement on repetitive nerve stimulation, Dr. Price stresses that a decrement is only observed in 60-70% of patients with MG. (Tr. 168, 204.) Moreover, he notes that petitioner had been on Mestinon at the time of the study, which could have impacted sensitivity of the test. (*Id.* at 168.) Petitioner was also never tested for low density lipoprotein c receptor-related protein 4 antibodies ("LRP4"), nor did he ever undergo single fiber EMG testing, which is the most sensitive test for diagnosing MG. (Ex. A, p. 10; Tr. 133, 241.)

While Dr. Price stresses that petitioner's initial marked improvement on Mestinon, especially with respect to his bulbar symptoms, is indicative of MG (Ex. A, pp. 10, 13-14; Tr. 149-50, 157, 230), he acknowledges that petitioner subsequently experienced a regression in the management of some of his symptoms, specifically fatigue and weakness (Tr. 193-94, 196). He explains that generalized MG is typically not controlled indefinitely with symptomatic treatment alone. (*Id.* at 150-51, 193.) While a patient may experience an initial improvement in their symptoms after initiating Mestinon, the underlying problem in generalized MG is the immune system. (*Id.* at 150, 193.) Therefore, effective treatment requires symptomatic therapy with medications like Mestinon in combination with medications like prednisone, CellCept, methotrexate, and IVIG, that suppress or attempt to suppress the antibody production in MG, even if the specific antibody cannot be identified. (*Id.* at 150-51, 193-94.) Accordingly, he states that it is typically standard practice to start a patient with generalized MG on prednisone and a prednisone-sparing agent, such as CellCept, in addition to Mestinon. (*Id.* at 193-94.) Given petitioner's delay in starting prednisone, Dr. Price opines that it is not surprising that petitioner, after experiencing a period of initial improvement, started to notice that some of his symptoms were not responding as well to Mestinon and required immunosuppression. (*Id.* at 194.)

Moreover, Dr. Price opines that petitioner's persistent and excessive fatigue is likely multifactorial, and points to documentation in the medical records to support this contention. (Tr. 196-98, 208-09, 214-15, 223, 230.) While MG would contribute to petitioner's fatigue, Dr. Price notes that MG would not be expected to cause brain fogginess, and neither would inflammatory polyradiculopathy. (*Id.* at 197.) He stresses that Dr. Smith was concerned that petitioner was suffering from undiagnosed obstructive sleep apnea, which was ultimately later confirmed via a sleep study. (*Id.* at

197-98; Ex. A, p. 11.) Dr. Smith also diagnosed petitioner with work shift sleep disorder. (Tr. 198; Ex. A, p. 11.) Given that both of these sleep disorders are not autoimmune in nature, they would not respond to Mestinon, anti-inflammatories, or steroids. (Tr. 198.) Accordingly, Dr. Price opines that these untreated sleep disorders likely contributed to petitioner's persistent and excessive fatigue. (*Id.*) He also notes that Dr. Smith diagnosed petitioner with a distal symmetrical neuropathy in 2019, highlighting that his neuropathy included a severe pain component that limited petitioner's sleep thereby contributing to his fatigue. (*Id.* at 213-14.)

Dr. Price opines that petitioner does not suffer from CIDP. (Ex. A, pp. 11-12; Tr. 138, 159.) Dr. Price opines that petitioner's clinical presentation is not indicative of CIDP, noting that petitioner's "prominent bulbar symptoms" are not characteristic of CIDP. (Ex. A, p. 12.) Typically, a loss of reflexes is one of the first symptoms observed in a demyelinating polyneuropathy. (Tr. 159.) Dr. Price notes that petitioner's reflexes were symmetrical and intact throughout 2016, 2017, and 2018, and therefore not suggestive of CIDP. (*Id.* at 159 (discussing Ex. P2, p. 196), 236-37.) Furthermore, Dr. Price indicates that the brief episodic nature of petitioner's symptoms is inconsistent with CIDP. (Ex. A, p. 12.) Additionally, petitioner's lumbar puncture did not reveal albuminocytologic dissociation, which is typically seen in individuals with CIDP involving the nerve roots. (*Id.*)

Moreover, Dr. Price opines that petitioner's electrodiagnostic testing does not support a diagnosis of CIDP. (Ex. A, pp. 11-12.) While he recognizes that Dr. Smith found evidence of demyelination on petitioner's first EMG and considered a diagnosis of CIDP, Dr. Price stresses that petitioner did not satisfy any of the electrodiagnostic criteria for a diagnosis of CIDP on either of his two EMG studies. (*Id.* at 7, 11-12; Tr. 163-64, 204, 207, 209.) He notes that petitioner's nerve conduction studies were "mostly normal" with some "very minimal abnormalities." (Tr. 162.) Dr. Price disagrees with Dr. Smith's finding of possible early demyelinating changes on petitioner's initial EMG. (*Id.* at 162-64, 204.) Although he acknowledges that petitioner's distal latencies in his right ulnar nerve and some of his sensory nerves were slightly longer than expected, Dr. Price explains that the distal latencies were not prolonged enough to meet formal, definitive criteria for demyelination. (*Id.* at 163-64.) Therefore, he contends that Dr. Smith's finding of evidence of demyelination on petitioner's first EMG that showed signs of improvement on his second EMG represents an "overinterpretation" of petitioner's nerve conduction studies. (*Id.* at 209.) Dr. Price stresses that there were no demyelinating features on either of petitioner's nerve conduction studies. (*Id.* at 204.) He also notes that petitioner's F wave studies were normal with no evidence of proximal demyelination or axonal loss. (*Id.* at 164-65.)

Dr. Price also opines that petitioner's condition is inconsistent with inflammatory polyradiculopathy. (Ex. A, pp. 12-13.) He explains that inflammatory polyradiculopathy, a condition distinct from MG, is a disease involving multiple nerve root injuries. (Tr. 137.) The most common cause of a radiculopathy or polyradiculopathy is degenerative changes to the spine. (*Id.* at 137-38.) Other causes include infections and inflammatory processes, meaning "more of an autoimmune-type condition." (*Id.* at 138.)

Dr. Prices agrees that inflammatory polyradiculopathy represents a large umbrella category of various conditions, including CIDP and acute inflammatory demyelinating polyradiculopathy ("AIDP"). (*Id.*) Typical symptoms depend on which nerve roots and cranial nerves are impacted, as well as the cause of the inflammatory polyradiculopathy. (*Id.* at 139.) Dr. Price acknowledges that an individual can develop a cranial neuropathy that causes double vision, dysphagia, or weakness or numbness in particular areas. (*Id.*) However, he notes that those symptoms are usually "relatively fixed" with an inflammatory polyradiculopathy, and therefore likely would not fluctuate as the day progresses. (*Id.* at 139-40, 195.)

Dr. Price emphasizes that petitioner's brief episodes of predominantly bulbar symptoms and fatigable weakness on examination are not consistent with a diagnosis of inflammatory polyradiculopathy. (Ex. A, p. 12.) For example, he discusses notations in petitioner's medical records that indicate his symptoms would emerge or worsen at the end of his work shift. (*E.g.*, Tr. 195 (discussing Ex. P6, p. 48).) Dr. Price notes that these subjective reports demonstrate fatigability within the day, which is very typical for MG but atypical and not expected in patients with inflammatory polyradiculopathy. (*Id.*) He also stresses that petitioner's sensory examinations were normal. (*E.g.*, *id.* at 144-45.) Although petitioner reported some sensory symptoms at his first neurology encounter on September 21, 2016, including a facial tremor and numbness and tingling in his face, arm, and leg, Dr. Price highlights that there was no objective evidence of these sensory symptoms on exam. (*Id.* at 144-45, 159-60, 195, 229-30, 235, 264-66.) While he acknowledges that later medical records indicate that petitioner had objective evidence of a tremor, Dr. Price notes that the tremor was said to be an essential tremor, which is an inherited tremor unrelated to inflammatory polyradiculopathy. (*Id.* at 268-70.) Additionally, Dr. Price stresses that petitioner's lumbar puncture was normal and showed no evidence of pleocytosis or elevated cerebrospinal fluid protein, which would be inconsistent with inflammatory polyradiculopathy. (Ex. A, p. 12; Tr. 190-92, 230-31.)

While Dr. Price recognizes that petitioner's EMG conducted on December 7, 2016, did show "chronic denervation with increased motor unit amplitude and duration of the bilateral lower extremity muscles and C7/C8 muscles, with preservation of sensory responses consistent with lumbar radiculopathies and a C7/8 radiculopathy," he opines that the needle EMG findings are not consistent with an inflammatory radiculopathy beginning after September 16, 2016. (Ex. A, p. 12; *see also* Ex. E, p. 3; Tr. 173.) He maintains that the needle EMG findings suggest that petitioner had mild to moderate nerve root axonal injury prior to receiving his flu vaccination on September 16, 2016. (Ex. A, p. 12; Ex. E, p. 3; Tr. 173.) Dr. Price states that axonal loss has "a very predictable pattern of changes" demonstrated on needle EMG examination. (Tr. 170.) He notes that "[w]ith acute motor axonal loss from a radiculopathy of any cause, there will be denervation of muscle cells that were supplied by the injured axons." (Ex. E, p. 4.) When a muscle cell is denervated, "meaning it no longer has a neuromuscular junction or a nerve controlling it," the muscle cell will start turning itself on after about two to three weeks from when the injury occurred. (Tr. 170; *see also* Ex. A, p. 13; Ex. E, p. 4.) This spontaneous electrical activity is referred to as fibrillation potentials or positive sharp waves. (Tr. 170.) With a needle EMG examination, Dr. Price explains

that, when you put a needle close to a muscle that is denervated, these spontaneous electrical activities will be observed when the patient is asked not to move. (*Id.* at 171.) The denervated muscle cell will continue to electrically turn itself on for at least two years if it never gets reinnervated, or until collateral reinnervation is complete. (*Id.*; Ex. A, p. 13; Ex. E, p. 4.)

When a denervated muscle cell undergoes collateral reinnervation, meaning the muscle cell is now controlled by a remaining motor axon, the motor unit amplitude and duration will increase on EMG. (Ex. E, p. 4; Tr. 171-72.) Dr. Price explains that this finding typically begins to develop three to six months after the acute denervation. (Ex. E, p. 4; Tr. 172.) During the early stages of reinnervation, the motor units may have prolonged durations with normal amplitudes. (Ex. E, p. 4.) Therefore, if petitioner had developed inflammatory polyradiculopathy with axon loss beginning on September 16, 2016, his EMG on December 7, 2016 should have shown "fibrillation potentials or positive sharp waves without increased motor unit amplitudes." (Ex. A, p. 13; *see also* Ex. E, p. 4.)

Dr. Price stresses that petitioner's EMG study on December 7, 2016, demonstrated increased motor unit amplitude and duration, which are findings that indicate complete reinnervation of the denervated muscle and typically develop three to six months after the acute denervation. (Ex. A, pp. 12-13; Ex. E, p. 4; Tr. 172.) Furthermore, he emphasizes that petitioner's needle examination did not reveal fibrillation potentials or positive sharp waves, which are signs of subacute denervation. (Ex. A, p. 13; Ex. E, p. 4; Tr. 174-75.) Accordingly, Dr. Price opines that petitioner's EMG findings are consistent with chronic denervation and complete reinnervation occurring at least three to six months prior. (Ex. E, p. 4; Tr. 173-75; Ex. A, pp. 12-13.) Thus, Dr. Price argues that the findings of petitioner's EMG conducted on December 7, 2016, evidence that his radiculopathies predate petitioner's flu vaccine that he received on September 16, 2016. (Ex. A, p. 12; Ex. E, p. 3; Tr. 173-75.) He also emphasizes that Dr. Smith described petitioner's polyradiculopathy as longstanding and inactive, rather than subacute or recent, which he argues is consistent with his opinion that petitioner's polyradiculopathy clearly predates vaccination. (Tr. 182-83.) Dr. Price also opines that petitioner' second EMG/never conduction study is consistent with a chronic, more than six-month old polyradiculopathy without significant progression or evidence of worsening injury. (*Id.* at 203-06 (discussing Ex. P29, pp. 395-96).)

Dr. Price dismisses Dr. Steinman's reliance on Doppler et al. as evidence of diagnosis in this case. (Ex. A, p. 13; Tr. 233-39 (discussing Doppler et al., *supra*, at Ex. P24, Tab 6).) Specifically, Dr. Price argues that petitioner's clinical picture is not similar to the clinical picture of the four patients with anti-contaction-1-associated neuropathy in Doppler et al. (Ex. A, p. 13; Tr. 233-39.) In Doppler et al., the four patients were all initially diagnosed with GBS after presenting with an acute onset of rapidly progressing proximal and distal weakness and distal sensory symptoms. (Ex. A, p. 13; Tr. 234-35.) Dr. Price acknowledges that petitioner experienced some proximal weakness; however, he stresses that petitioner did not have progressive distal weakness. (Ex. A, p. 13; Tr. 235.) Additionally, while petitioner reported some numbness and tingling in his hands

and feet, Dr. Price observes that those symptoms were never objectively corroborated on physical exam. (Tr. 235.) Moreover, Dr. Price explains that GBS typically involves an acute presentation of severe ascending weakness with absent reflexes. (*Id.* at 236-37.) He argues that petitioner's reflexes were intact on exam when he first sought care in 2016 and 2017, and notes that petitioner never had severe weakness. (*Id.* at 237.) The four patients in Doppler experienced initial improvement with IVIG treatment, whereas petitioner experienced initial improvement on Mestinon. (*Id.* at 236.) Dr. Price highlights that there is no evidence in the medical records to suggest that petitioner's treating providers initially suspected he was suffering from GBS, nor do the authors in Doppler et al. suggest that myasthenia gravis was considered as an initial diagnosis for any of the four patients. (*Id.* at 238.)

Dr. Price also explains that petitioner's clinical course differs from the patients in Doppler et al. from an electrophysiological perspective. (Ex. A, p. 13; Tr. 237.) Prior to the detection of contactin-1 autoantibodies, the diagnosis for the four patients in Doppler et al. was modified from GBS to CIDP, a condition that Dr. Price and Dr. Steinman agree petitioner does not suffer from. (Ex. A, pp. 12-13.) Nerve conduction studies for all four patients revealed prolonged distal latencies, decreased nerve conduction velocity, and prolonged F wave latencies, supporting their modified diagnosis of CIDP. (*Id.* at 13; Tr. 237.) Dr. Price opines that petitioner's nerve conduction studies were normal with no evidence of demyelination. (Ex. A, p.13; Tr. 237.) Lastly, Dr. Price emphasizes that, while all four patients in Doppler et al. had elevated protein in their cerebrospinal fluid, petitioner's lumbar puncture was normal. (Ex. A, p. 13; Tr. 238.)

Dr. Price further notes that Kadoya et al. studied patients with CIDP; however, because petitioner does not suffer from a CIDP, a conclusion Dr. Steinman agrees with, Dr. Price opines that this study has no relevance to this case. (*Id.* (citing Kadoya et al., *supra*, at P25, Tab 6).) Additionally, both Vural et al. and Kadoya et al. studied patients with antibodies against human neurofascin155. (*Id.* at 2 (discussing Kadoya et al., *supra*, at P25, Tab 6; Vural et al., *supra*, at Ex. P25, Tab 5).) However, there is no evidence in the medical records that suggests petitioner was ever assessed for human neurofascin155 antibodies. (*Id.*) Moreover, Dr. Price stresses that petitioner's clinical presentation is not consistent with the patients in Kadoya et al. and Vural et al., which suggests that petitioner does not have antibodies against human neurofascin155. (*Id.* at 2-3.) Specifically, petitioner "did not have loss of proprioception sensation, or diffuse areflexia, or hyporeflexia as seen with a peripheral cause of a sensory ataxia or tremor." (*Id.* at 3.) Nor did the medical records show elevated cerebrospinal fluid protein, prolonged distal motor or f-wave latencies, or enhancement or enlargement of the spinal root on MRI. (*Id.*) Lastly, petitioner was treated with IVIG, not Rituximab. (*Id.*) Considering patients with human nuerofascin155 antibodies typically respond poorly to IVIG treatment, Dr. Price contends that petitioner's treating physicians were clearly not considering a nuerofascin155 antibody mediated condition. (*Id.*)

In sum, Dr. Price contends that it is "exceedingly more likely than not" that petitioner suffers from autoimmune, seronegative MG based on his clinical presentation. (Tr. 228, 241.) Based on documentation in the medical records, Dr. Price opines that

the onset of petitioner's MG was likely before petitioner received his flu vaccination on September 16, 2016. (*Id.* at 142-44, 153, 156.) In addition to MG, Dr. Price states that, based on his review of the medical records, petitioner also has bilateral lumbar radiculopathies caused by his spinal stenosis, sensory polyneuropathy likely secondary to prolonged steroid use, shift work sleep disorder, obstructive sleep apnea, and a chronic, longstanding, inactive polyradiculopathy that likely predates petitioner's flu vaccine at issue. (*Id.* at 228.) He maintains that petitioner's clinical presentation is not consistent with inflammatory polyradiculopathy or a contactin-1 mediated disease. (*Id.* at 232-33, 242.) Lastly, Dr. Price agrees with Dr. Steinman that petitioner does not suffer from CIDP as his EMG studies did not reveal evidence of demyelination. (*Id.* at 228, 231.)

c. Respondent's Expert, John T. Bates, Ph.D.[10]

Respondent's second expert, Dr. Bates, submitted two expert reports in this case and testified at the entitlement hearing. (Exs. C, F; Tr. 274-320.) He was proffered as an expert in immunology without objection. (Tr. 6-7.) Dr. Bates defers to Dr. Price on the issue of petitioner's diagnosis and addresses Dr. Steinman's theory of causation as it relates to inflammatory polyradiculopathy, the diagnosis endorsed by Dr. Steinman. (Ex. C, p. 3; Tr. 312-13.) Accordingly, his opinion is not further summarized.

V. Analysis

In this case, petitioner initially alleged that the flu vaccine at issue caused him to develop MG and CIDP. (ECF No. 1.) Following his expert's assessment, petitioner filed an amended petition alleging that the flu vaccine caused him to suffer MG, CIDP, and/or inflammatory polyradiculopathy. (ECF No. 44.) In their prehearing submissions, the parties indicated that they agree that petitioner's injury is not CIDP. (ECF No. 69, p. 1; ECF No. 70, p. 16, n.4; ECF No. 72, p. 15, n.5.) Additionally, both Dr. Steinman and Dr. Price testified that petitioner does not suffer from CIDP. (Tr. 108, 159.) However, the parties dispute whether MG or inflammatory polyradiculopathy is the best supported diagnosis in this case.

Petitioner asserts that he suffers from inflammatory polyradiculopathy, stressing that all of the documentation from his treating physicians and Dr. Steinman's expert opinion support the diagnosis. (ECF No. 70, pp. 16-19.) In his prehearing submissions, petitioner emphasizes that Dr. Steinman and his treating neurologist, Dr. Smith, agree that MG is not an appropriate diagnosis, and that they both opine that he is experiencing polyradiculopathy. (*Id.*; ECF No. 74, p. 2.) After the conclusion of the entitlement hearing, petitioner released any claim that he suffered vaccine-caused MG.

[10] Dr. John T. Bates received his Ph.D. in microbiology from the University of Alabama at Birmingham in 2005, before going on to complete two post-doctoral fellowships, one in the Department of Microbiology and Immunology at Wake Forest University School of Medicine in 2010 and another in the Vanderbilt Vaccine Center at Vanderbilt University School of Medicine in 2014. (Ex. D, p. 1.) He currently works as an assistant professor at the University of Mississippi Medical Center in both the Department of Medicine and the Department of Microbiology and Immunology. (*Id.* at 2.) He has authored 19 publications. (*Id.* at 2-4.)

(ECF No. 77.) In his post-hearing briefing, petitioner argues that his flu vaccination caused significant aggravation of his preexisting lumbar spine radiculopathy which he alleges developed into polyradiculopathy involving his cervical spine. (ECF No. 83, p. 2; ECF No. 85, p. 2.) Petitioner acknowledges that his treating neurologist diagnosed him with both MG and polyradiculopathy and maintains that an individual can suffer from both conditions. (ECF No. 85, pp. 2-3.)

Respondent asserts that petitioner was correctly diagnosed with MG, which is the best supported diagnosis in this case. (ECF No. 72, pp. 15-17; ECF No. 84, pp. 15-18.) He contends that petitioner's clinical presentation is inconsistent with inflammatory polyradiculopathy and thus cannot be preponderantly supported given the record evidence. (ECF No. 72, pp. 16-17; ECF No. 84, pp. 17-18.) Therefore, respondent maintains that, because petitioner cannot demonstrate that he suffers from inflammatory polyradiculopathy by a preponderance of the evidence, petitioner's claim fails as his proposed causal theory is based only on a diagnosis of inflammatory polyradiculopathy. (ECF No. 84, p. 18.)

For the reasons discussed below, I find that there is preponderant evidence that petitioner suffers from MG *rather than* an inflammatory polyradiculopathy.

a. Petitioner's symptoms are consistent with MG

Dr. Price emphasized that petitioner's symptoms were primarily bulbar with some proximal weakness which is "perfectly consistent" with MG. (Tr. 229; *see also id.* at 201, 267; Ex. A, pp. 13-14.) Specifically, he noted that petitioner's symptoms of worsening fatigue, ptosis, dysphagia, diplopia, blurred vision, slurred nasal speech, shortness of breath, and fatigable weakness with activity, support a diagnosis of MG. (Ex. A, pp. 9-11,13-14 (citing Ciafaloni, *supra*, at Ex. A, Tab 1); Tr. 141-45, 148-50, 153-57, 267.) While petitioner experienced some proximal weakness throughout his clinical course (*e.g.*, Tr. 196 (discussing Ex. P6, p. 52)), Dr. Price stressed that there was not much evidence of distal weakness in the medical records, which is consistent with MG (*Id.* at 229, 235). Moreover, throughout the hearing, Dr. Price testified that even when Dr. Smith started to suspect that petitioner suffered from CIDP, petitioner's clinical presentation remained consistent with MG. (*E.g.*, *id.* at 218-19, 221-23, 226.) While Dr. Price acknowledged that some of petitioner's subjective sensory complaints cannot be explained by MG, all of petitioner's objective findings are consistent with MG. (*Id.* at 265-66.)

Moreover, Dr. Price stressed that fluctuating and fatigable weakness of muscle groups that worsens with activity and improves with rest is the core clinical feature of MG. (Ex. A, p. 9 (citing Ciafaloni, *supra*, at Ex. A, Tab 1); Tr. 135-36.) As pointed out by Dr. Price, there are several notations in the medical records indicating that petitioner's symptoms fluctuated, worsening as the day progressed and with repetitive use. For example, at his initial neurology encounter on September 21, 2016, petitioner reported experiencing episodic blurred vision that was associated with fatigue and occurred at night. (Ex. P3, p. 5.) Additionally, at his encounter with Dr. Sadeghi on

October 6, 2016, petitioner reported needing to take an increased dose of Mestinon when he is working long shifts to counterbalance the fact that his symptoms recur at the end of the day. (*Id.* at 46.) This fluctuating and fatigable weakness was also objectively demonstrated on physical exam, with instances of diplopia and fatigability being observed with sustained upward gaze and mild fatigability of the right upper extremity with repetitive effort. (*Id.* at 6, 47.)

Ultimately, Dr. Price is persuasive in opining that petitioner's clinical presentation is consistent with a diagnosis of MG. (Tr. 267.) Dr. Steinman likewise agreed that petitioner's presenting symptoms post-vaccination were predominantly bulbar in nature. (*Id.* at 118.) He also acknowledged that petitioner's presenting symptoms, specifically double vision and trouble chewing and swallowing, are symptoms typically observed in patients with MG. (*Id.*) Moreover, petitioner's treating neurologist, Dr. Smith, classified many of the symptoms identified above as "myasthenic symptoms." (*E.g.*, Ex. P6, p. 48.) Therefore, both experts and petitioner's treating physician appear to agree that petitioner's presentation is consistent with MG.

b. Petitioner's antibody and electrodiagnostic testing results are not inconsistent with a diagnosis of MG

Petitioner points to his antibody and EMG testing as support for his argument that MG is not the most appropriate diagnosis in this case. (Tr. 45-46, 100, 118, 323; ECF No. 70, pp. 17-19; Ex. P24, p. 5.) Dr. Steinman testified that individuals who have MG "rarely" test negative for both the acetylcholine receptor and MuSK antibodies. (Tr. 45.) He also stressed that Dr. Smith conducted a comprehensive electrodiagnostic study, which was negative for MG. (*Id.* at 100.) While he conceded that there is "a small chance" that petitioner could have seronegative, EMG negative MG, he stated he would put "[v]ery low weight" on that. (*Id.*)

However, Dr. Price explained that a sizeable minority of individuals with autoimmune MG will lack a known antibody and have no evidence of decrement on repetitive nerve stimulation. (Tr. 128-29, 133-35, 241.) Specifically, Dr. Price testified that seronegative MG, meaning an individual is negative for both the acetylcholine receptor and MuSK antibodies, is observed in approximately 15-20% of cases of autoimmune MG. (*Id.* at 128-29, 216, 224-25, 266-67; Ex. A., pp. 9-10 (citing Caifaloni, *supra*, at Ex. A, Tab 1).) Additionally, Dr. Price explained that a decrement on repetitive nerve stimulation is only observed in about 60-70% of individuals with MG. (Tr. 133-34, 165-68.) He noted that the sensitivity of repetitive nerve stimulation depends upon the muscle being tested, indicating that if the muscle is not a weak muscle, then the sensitivity of the test declines. (*Id.* at 133-34.)

Moreover, petitioner was already on Mestinon at the time of his EMGs (Ex. P6, pp. 9, 91-92, 111-12), which may have impacted the sensitivity and quality of the testing. (Tr. 232.) Although petitioner was instructed to hold his Mestinon for 36 hours prior to his EMG (Ex. P6, p. 9), his treating neurologist, Dr. Smith, raised the possibility of a false negative in a later treatment record, noting that Mestinon may not have been

"completely 'out of [petitioner's] system'" at the time of his EMG testing (Ex. P28, p. 1479). Dr. Price explained that

> if you are trying to stress the neuromuscular junction with multiple repetitive stimulations within a second and the patient is able to keep more acetylcholine in the neuromuscular junction because of the presence of Mestinon, then you are not stressing it as much as you think and therefore you will have a false negative test.

(Tr. 232.)

Dr. Price testified that it is not uncommon for neurologists at academic medical centers to see patients who are seronegative and had a normal repetitive nerve stimulation who are only found to have MG by a single-fiber EMG of a weak muscle. (Tr. 134-34; *see also* Ex. A, p. 9.) A single-fiber EMG, the diagnostic test for MG with the highest sensitivity, and is positive in 97% of affected patients. (Ex. A, p. 9 (citing Caifaloni, *supra*, at Ex. A, Tab 1).) A normal single-fiber EMG in an affected muscle excludes MG as a possible diagnosis. (*Id.* (citing Caifaloni, *supra*, at Ex. A, Tab 1).) However, petitioner never underwent a single-fiber EMG. (*Id.* at 9-10; Tr. 222, 241.)

Dr. Price is persuasive in explaining why petitioner's antibody and electrodiagnostic testing results do not rule out MG as a likely diagnosis.

c. <u>Petitioner's improvement on Mestinon supports a diagnosis of MG</u>

Both parties acknowledge that petitioner reported experiencing "significant" improvement in his symptoms after starting Mestinon. (ECF No. 70, p. 2; ECF No. 72, p. 4 (citing Ex. P3, pp. 46-47).) At his neurology follow-up visit on October 6, 2016, petitioner reported that since initiating Mestinon, his fatigability improved, noting that he was able to perform most of his duties at work without issue, and he also denied further episodes of double vision and ptosis (Ex. P3, p. 46), which are all symptoms consistent with MG (Tr. 150). When asked whether this marked improvement in petitioner's symptoms after starting Mestinon is what he would expect to see in an individual with MG, Dr. Price testified, "Yes, a hundred precent." (*Id.* at 230.) Additionally, he emphasized that taking Mestinon alone would not provide any symptomatic relief in an individual suffering from inflammatory polyradiculopathy. (*Id.* at 151.)

Dr. Price explained that Mestinon, as an acetylcholinesterase inhibitor, is a medication specific to the symptomatic treatment of MG. (Tr. 149.) He stressed that "[i]t is not used in the treatment of virtually any other neurologic conditions[,]" including GBS, CIDP, sarcoidosis, or radiculopathies, and would therefore not be expected to provide any symptomatic relief in individuals with those diseases. (*Id.*) Moreover, Dr. Steinman agreed that Mestinon is an effective treatment for MG and conceded that it is not a medication used to treat inflammatory polyradiculopathy. (*Id.* at 45, 98.) He also opined that petitioner "was treated with the appropriate drugs." (Ex. P25, p. 12.) Given his concession that Mestinon is a drug that provides effective treatment for MG and not

a medication prescribed to treat inflammatory polyradiculopathy, Dr. Steinman failed to explain why petitioner was appropriately treated with Mestinon if he does not suffer from MG. Instead, Dr. Price is persuasive in opining that symptomatic improvement in an individual on Mestinon "would be highly suggestive of a neuromuscular junction disease," such as MG. (Tr. 132.)

During the hearing, Dr. Steinman emphasized that Dr. Smith added prednisone, IVIG, CellCept, and methotrexate to petitioner's medication regimen throughout the course of petitioner's treatment. (Tr. 323-24.) He argued that the fact that Dr. Smith added these "highly impactful" medications on top of Mestinon suggests that petitioner was not responding as well to Mestinon as respondent claims, thereby casting doubt on MG as the appropriate diagnosis. (*Id.*) However, Dr. Steinman also conceded that additional medications prescribed by Dr. Smith, specifically prednisone and CellCept, would benefit an individual suffering from MG. (*Id.* at 45-46.) During the hearing, Dr. Price persuasively testified that, because the underlying problem in autoimmune MG is the immune system, effective treatment often requires medications that address the underlying immune component in addition to symptomatic therapy like Mestinon. (*Id.* at 150-51.) Therefore, he explained that it is not uncommon for patients with MG to be prescribed medications such as prednisone, CellCept, methotrexate, and IVIG in combination with Mestinon. (*Id.* at 150-51, 262.) While Dr. Smith did add these medications due to a suspicion of CIDP, he never removed MG from petitioner's list of diagnoses or stopped Mestinon. Moreover, the fact that Dr. Smith noted that petitioner responded to IVIG treatment is not informative for the reasons discussed by Dr. Price.

Additionally, Dr. Price explained that generalized MG is not typically controlled indefinitely with symptomatic management alone. (Tr. 193.) While an individual with MG may initially show signs of improvement after starting symptomatic treatment like Mestinon, the individual's symptoms will typically worsen over time or respond less to Mestinon if the underlying autoimmune activity that is causing the MG is not treated. (Tr. 193-94.) Accordingly, Dr. Price emphasized that individuals with generalized MG are typically started on prednisone in addition to Mestinon. (*Id.*) However, in this case, petitioner did not start taking prednisone and CellCept until January of 2017, approximately 3 months after initiating Mestinon. (Ex. P6, pp. 22-25.) Given petitioner's delay in starting on an immunosuppressant, Dr. Price is persuasive in opining that it is not surprising that petitioner's symptoms worsened after initially showing signs of improvement after starting Mestinon.[11] (Tr. 194.)

[11] When petitioner reported experiencing significant fatigue and brain fog despite remaining on Mestinon in March of 2017, Dr. Smith diagnosed petitioner with shift work sleep disorder and expressed concern for obstructive sleep apnea, which petitioner was subsequently diagnosed with. (Ex. P6, p. 55; Ex. P15, p. 12.) Dr. Price testified that while MG certainly could have contributed to some of petitioner's fatigue, it would not explain his entire clinical presentation as MG does not cause brain fog. (Tr. 197.) Critically, he stressed that inflammatory polyradiculopathy also does not cause brain fog. (*Id.*) Therefore, Dr. Price explained that petitioner's two different sleep disorders were likely contributing, at least in part, to petitioner's extreme fatigue. (*Id.* at 197-98, 209.) Because these sleep disorders are not autoimmune conditions, they would not respond to Mestinon or immunosuppressants. (*Id.* at 198.) Petitioner failed to address these non-neurologic causes of fatigue when assessing petitioner's response to Mestinon. Accordingly, the symptomatic flares petitioner experienced, particularly with his fatigue, do not evidence that petitioner was not responding effectively to Mestinon.

In sum, petitioner's response to Mesitnon is strong evidence supporting a diagnosis of MG. Petitioner has not offered any compelling explanation for why he would have experienced this symptomatic improvement after starting Mestinon if his presentation was primarily due to inflammatory polyradiculopathy.

d. Petitioner mischaracterizes Dr. Smith's opinion regarding the most appropriate diagnosis in this case

As a treating provider, petitioner argues that Dr. Smith's findings should carry significant weight as to the appropriate diagnosis in this case. (ECF No. 70, pp. 17-19 (citing *Andreu*, 569 F.3d at 1375); *see also* ECF No. 83, pp. 6-7.) Petitioner contends that Dr. Smith "did not agree" that he was suffering from MG and diagnosed petitioner with polyradiculopathy. (ECF No. 70, p. 17.) Dr. Steinman testified that it is very clear that Dr. Smith does not think petitioner suffers from MG, and he agrees with him. (Tr. 98.) He stated that Dr. Smith weighed all the evidence and abandoned a diagnosis of MG in favor of inflammatory polyradiculopathy. (*Id.* at 45, 53.) Petitioner stresses the importance of the record for his follow-up encounter on March 14, 2022, in which Dr. Smith documented that, "[w]ith regards to his underlying disease, [petitioner] does not meet criteria either for myasthenia gravis or CIDP, but does have a polyradiculopathy and fatigable weakness that appears to respond to IVIG." (ECF No. 70, pp. 18-19 (emphasis omitted) (citing Ex. P28, p. 1012).)

Dr. Price suggested, however, that Dr. Smith's comment that petitioner did not meet the criteria for MG simply reflects that petitioner tested negative for MG-associated antibodies and had no decrement on repetitive nerve stimulation (Tr. 222-24), points addressed separately above as not being dispositive of diagnosis. Indeed, there are several records that include documentation by Dr. Smith expressing his frustration with the lack of antibody and electrodiagnostic evidence to confirm a diagnosis of MG. (*See, e.g.*, Ex. P6, pp. 77, 97, 131; Ex. P28, p. 438; Ex. P29, p. 374.) However, this does not actually indicate that Dr. Smith had concluded that MG is not an appropriate diagnosis. Dr. Smith also repeatedly documented that petitioner should consider undergoing testing for low density lipoprotein c receptor-related protein 4 antibodies ("LRP4") (Ex. P28, pp. 436, 721, 832), which, as explained by Dr. Price, demonstrates that Dr. Smith was still considering the involvement of a rare antibody for MG (Tr. 224-25). Moreover, despite his remark about petitioner not meeting the criteria, Dr. Smith still listed MG as one of petitioner's diagnoses on March 14, 2022. (Ex. P28, p. 1026.) In fact, Dr. Smith continued to include MG as a diagnosis through March 27, 2023, and no records for subsequent neurology encounters were filed in this case.[12] (*Id.* at 1493.) Indeed, petitioner testified that Dr. Smith continued to prescribe him Mestinon, noting that he was still taking the medication as of the date of the hearing. (Tr. 27.) As explained

[12] Notably, Dr. Steinman did not accept that ongoing inclusion of MG as a listed diagnosis in petitioner's electronic medical record is meaningful as to Dr. Smith's thinking. (Tr. 101.) I have considered this point but conclude that the continued presence of MG as a listed diagnosis does provide some evidence of Dr. Smith's thinking when viewed in the context of the record as a whole.

above, both parties' experts recognize that Mestinon is an effective treatment for MG and is not a medication used to treat inflammatory polyradiculopathy. (*Id.* at 45, 98, 149, 151.)

Accordingly, petitioner is not persuasive in arguing that Dr. Smith abandoned a diagnosis of MG.

e. The medical records do not preponderantly support a diagnosis of inflammatory polyradiculopathy

Petitioner argues that "[a]ll of the documentation from Petitioner's treating physicians, in the weeks and months after vaccination, support the diagnosis of inflammatory polyradiculopathy." (ECF No. 70, p. 16.) In that regard, the record for petitioner's initial EMG on December 7, 2016, does indicate that Dr. Smith interpreted petitioner's EMG findings to be consistent with a "longstanding, progressive, relatively inactive, polyradiculopathy." (Ex. P19, p. 10.) Moreover, both Dr. Steinman and Dr. Price agree that petitioner's EMG reveals evidence of a polyradiculopathy. However, a finding of a polyradiculopathy on EMG does not automatically warrant a conclusion that the polyradiculopathy is inflammatory. Dr. Price testified that a polyradiculopathy can develop from a variety of different causes, with the most common cause being degenerative spine disease. (Tr. 137-38.) Regarding petitioner's own polyradiculopathy, Dr. Price explained that petitioner's EMG results, history of back pain, and negative lumbar puncture, are consistent with a mechanical explanation. (*Id.* at 137-41, 190-92, 205-06.)

Dr. Steinman repeatedly testified that petitioner's treating neurologist, Dr. Smith, diagnosed petitioner with inflammatory polyradiculopathy. (*E.g.*, Tr. 45-46, 53.) However, after extensively reviewing the medical records filed in this case, the undersigned was unable to locate any record in which Dr. Smith specifically diagnosed petitioner with an inflammatory, non-demyelinating polyradiculopathy. Rather, careful review of the medical records reveals that Dr. Smith diagnosed petitioner with a polyradiculopathy without ever explicitly specifying that petitioner's polyradiculopathy was inflammatory, as conceded by petitioner in his post-hearing briefing. (ECF No. 83, p. 9.) Moreover, when questioned by the undersigned, Dr. Steinman was unable to point to a record in which Dr. Smith specifically employed the terminology "inflammatory polyradiculopathy." (Tr. 104-05.) Instead, Dr. Smith identified a broad list of possible etiologies for petitioner's polyradiculopathy, including structural, inflammatory, infectious, neoplastic, vascular, metabolic, and genetic etiologies. (Ex. P19, p. 10.)

Nonetheless, Dr. Steinman testified that, even if Dr. Smith never explicitly used the terminology "inflammatory polyradiculopathy," it is clear that he felt petitioner's polyradiculopathy was inflammatory in nature. (Tr. 105.) This is informed two additional points. First, Dr. Steinman points to the fact that Dr. Smith had petitioner on two anti-inflammatory drugs, a steroid and CellCept. (*Id.*) He contends that Dr. Smith's decision to prescribe petitioner these medications demonstrates that he thinks petitioner's polyradiculopathy is inflammatory. (*Id.*) Second, in the record for petitioner's encounter

on November 19, 2020, Dr. Smith noted that "[t]he fact that [petitioner's] previous neuropathic findings in his ulnar and median nerve is actually improved on immunomodulatory therapy has suggested that his EMG quantitated and clinically identified motor neuropathic findings are likely inflammatory in nature (CIDP)." (Ex. P28, p. 436.)

However, as discussed separately above, Dr. Steinman's observation regarding anti-inflammatory medications is undercut by his testimony acknowledging that steroids and CellCept are medications that would also benefit an individual suffering from MG. (Tr. 45-46.) Therefore, the fact that petitioner was prescribed anti-inflammatories is not compelling evidence that petitioner was suffering from an inflammatory polyradiculopathy. Additionally, Dr. Smith's specific reference to a likely inflammatory process is inextricably linked to his interpretation of petitioner's second EMG study as showing an improvement in demyelination. (*Id.* at 219-20.) Yet, both Dr. Steinman and Dr. Price disagree with Dr. Smith's finding of evidence of demyelination on EMG and clearly opined that petitioner did not ever suffer from demyelination. (Tr. 102-03, 110-11, 113, 116, 138, 163-64, 201, 209, 216-17, 232.) Therefore, these points do not lend any support to petitioner's argument that inflammatory polyradiculopathy is the most appropriate diagnosis in this case.

Ultimately, Dr. Steinman indicated that his preferred diagnosis of inflammatory polyradiculopathy is arrived at by a process of elimination. (Tr. 118.) He stressed that petitioner's EMG and nerve conduction study results and the antibody testing results reinforce his preferred diagnosis of inflammatory polyradiculopathy because these tests are negative for MG. (*Id.* at 111-12, 118-19.) However, as explained above, petitioner's antibody testing results and EMG findings do not rule out a diagnosis of MG. Moreover, Dr. Steinman agreed that inflammatory polyradiculopathy is an umbrella term for a variety of conditions, such as CIDP. (*Id.* at 115-16.) However, especially because both parties agree that petitioner does not suffer from CIDP, Dr. Steinman is unable to point to any specific diagnosis under that umbrella, instead relying broadly on the concept of inflammatory polyradiculopathy as a non-specific umbrella diagnosis with no real diagnostic criteria. Dr. Steinman has not pointed to any significant objective evidence in the medical records that affirmatively supports his argument that inflammatory polyradiculopathy is the most likely diagnosis in this case.

f. Petitioner is not persuasive in arguing that his clinical presentation is most consistent with inflammatory polyradiculopathy

Dr. Steinman testified that all of petitioner's presenting symptoms could be explained by a diagnosis of inflammatory polyradiculopathy. (Tr. 118.) Because petitioner's presenting symptoms were predominantly bulbar, Dr. Steiman explains that petitioner's inflammatory polyradiculopathy would involve the "nerve roots that come off the central nervous system and innervate the extraocular muscles and the swallowing muscles." (*Id.*) Dr. Price acknowledged that an individual can develop a cranial radiculopathy that causes symptoms similar to those experienced by petitioner, including dysphagia, double vision, weakness, and numbness. (*Id.* at 139.) However,

the fact that a polyradiculopathy implicating cranial nerves is possible, does not necessarily demonstrate that inflammatory polyradiculopathy is the best supported diagnosis in this case.

Dr. Price opined that with inflammatory polyradiculopathy, symptoms are relatively fixed and therefore likely would not fluctuate as the day progresses. (Tr. 139-40, 195.) As explained above, there are several notations within the medical records indicating that petitioner reported his symptoms worsened with fatigue and as the day progressed, as well as documentation of fatigable weakness with repetitive use observed on physical exam. Petitioner has not offered any explanation of how the fluctuating fatigability of petitioner's symptoms is consistent with inflammatory polyradiculopathy. While Dr. Steinman cited literature in which patients who were found to have an anti-contactin-1-associated neuropathy experienced a relapsing and remitting course of disease (Doppler et al., *supra*, at Ex. P24, Tab 6, p. 6), there is no evidence in that article that suggests those patients experienced fluctuations in their symptoms as the day progressed. Moreover, Dr. Price distinguished a relapsing and remitting course from the fluctuating fatigability within the course of a day that is frequently observed in patients with MG. (Tr. 135-36.) Therefore, petitioner has not explained how his fluctuating fatigability is consistent with a diagnosis of inflammatory polyradiculopathy.

To support his opinion that inflammatory polyradiculopathy is the most likely diagnosis in this case, Dr. Steinman placed significant emphasis on the Doppler et al. paper. (Ex. P24, pp. 6-7; Tr. 104-08, 110, 116, 120 (discussing Doppler et al., *supra*, at Ex. P24, Tab 6).) In Doppler et al., the authors tested the serum of patients diagnosed with either GBS or CIDP for autoantibodies against contaction-1. (Doppler et al., *supra*, at Ex. P24, Tab 6, pp. 1, 3; Tr. 234.) The authors noted that autoantibodies directed against nodal and paranodal proteins, such as contactin-1, had recently been detected in patients with GBS and CIDP. (Doppler et al., *supra*, at Ex. P24, Tab 6, p. 1.) High reactivity to contactin-1 by ELISA was observed in four of the patients studied, all of whom had CIDP. (*Id.* at 1, 3.) While all four patients had electrodiagnostic studies indicative of demyelination and therefore consistent with a diagnosis of CIDP, sural nerve biopsies were performed and semithin sections demonstrated axonal loss and degeneration of nerve fibers without typical features of demyelination, such as onion bulbs or numerous thinly myelinated fibers. (*Id.* at 4-5, 8.) Analysis of myelinated fibers in skin biopsies revealed disruption of paranodal architecture. (*Id.* at 1, 6, 8.) Accordingly, the authors state that these histopathological findings support the theory that damage to the paranodes, rather than the myelin sheath, causes impairment of nerve conduction in the anti-contactin-1-associated neuropathy subtype of CIDP. (*Id.* at 8.)

Dr. Steinman opined that petitioner's clinical presentation is most consistent with the clinical picture of the four patients discussed in Doppler et al., stating that the diagnosis of inflammatory polyradiculopathy in Doppler et al. is the diagnosis that fits best in this case. (Tr. 116, 120; Ex. P24, p. 6 (discussing Doppler et al., *supra*, at Ex. P24, Tab 6).) However, Dr. Steinman did not further explain or specifically elaborate on

how petitioner's clinical presentation was similar or consistent with the four patients in Doppler et al. Moreover, Dr. Price persuasively described how petitioner's clinical presentation does not resemble those four patients from a clinical or electrophysiological perspective. (Tr. 233-39; Ex. A, p. 13.)

In Doppler et al., the authors summarized the clinical picture of each of the four patients. (Doppler et al., *supra*, at Ex. P24, Tab 6, pp. 3-4.) All four patients were initially diagnosed with GBS due to their acute and rapidly progressive distal and proximal weakness, as well as their distal sensory symptoms. (Doppler et al., *supra*, at Ex. P24, Tab 6, p. 3; Tr 234-35.) However, the diagnosis for all four of the patients was modified to CIDP when their symptoms progressed after experiencing a short period of initial improvement. (Doppler et al., *supra*, at Ex. P24, Tab 6, p. 3.) Additionally, all four of the patients in Doppler et al. experienced initial improvement with IVIG treatment. (*Id.*) The nerve conduction studies of all four patients showed prolonged distal latencies, decreased nerve conduction velocity, and prolonged F-wave latencies, which worsened throughout the course of the disease. (Tr. 237-38; Doppler et al., *supra*, at Ex. P24, Tab 6, p. 4.) Total cerebrospinal fluid protein was elevated in all four patients. (Tr. 238; Doppler et al., *supra*, at Ex. P24, Tab 6, p. 4).

As explained by Dr. Price, petitioner's clinical presentation is distinguishable from the four patients in Doppler et al. (Tr. 233-39; Ex. A, p. 13.) There is no record evidence indicating that petitioner's treating providers diagnosed him with GBS, and both experts agree that petitioner does not suffer from CIDP. Dr. Price emphasized that, in contrast to the four patients in Doppler et al., the onset of petitioner's disease was not characterized by rapidly progressive distal and proximal weakness and distal sensory symptoms. (Tr. 235.) As explained above, petitioner's symptoms were predominantly bulbar with some proximal weakness. While he complained of sensory symptoms at his initial neurology encounter, Dr. Price highlighted that those symptoms were never objectively corroborated on physical exam. (Tr. 235.) Additionally, petitioner experienced initial improvement on Mestinon, not IVIG, and there is nothing in the Doppler et al. paper that suggests that treating providers for any of the four patients suspected MG as a potential diagnosis. Moreover, both parties agree that petitioner did not have elevated protein in his cerebrospinal fluid. (*Id.* at 117, 238.) There is also no evidence in the medical records indicating that petitioner was ever tested for contactin-1 antibodies or that his treating providers suspected that petitioner suffered from a contactin-1 mediated disease.

Most notably, both Dr. Steinman and Dr. Price agree that petitioner's nerve conduction studies were relatively normal and did not show any evidence of demyelination. (Tr. 111, 160-65, 237.) Therefore, petitioner's electrodiagnostic studies stand in stark contrast to the progressively worsening prolonged distal and F-wave latencies and decreased nerve conduction velocity observed in the four patients in Doppler et al. Importantly, the authors in Doppler et al. emphasized that anti-contactin-1-associated neuropathy is associated with a reduction of nerve conduction velocity. (Doppler et al., *supra*, Ex. P24, Tab 6, p. 8.) Based on their findings, the authors suggested that reduced nerve conduction velocities and increased distal latencies, as

observed in the four patients at issue, could be indicative of disruption to the paranodal architecture caused by autoantibodies against contactin-1. (*Id.*) Therefore, I find Dr. Price's testimony regarding the relevance of Doppler et al. persuasive. While Doppler et al. demonstrates that a contacted-1-mediated autoantibody disease is possible, it is not evidence that inflammatory polyradiculopathy is more likely than not the most appropriate diagnosis in petitioner's case.

In sum, petitioner has not offered preponderant evidence that his clinical presentation is most consistent with a diagnosis of inflammatory polyradiculopathy.

g. Petitioner is not persuasive in suggesting that petitioner suffers from both MG and inflammatory polyradiculopathy

In his prehearing briefing, petitioner states that petitioner's treating neurologist, Dr. Smith, "did not agree that he was experiencing MG but rather his diagnosis was that [petitioner] was experiencing polyradiculopathy." (ECF No. 70, p. 17.) He emphasizes that Dr. Smith documented that petitioner "does not meet criteria" for a diagnosis of MG, stressing that petitioner's EMG studies did not reveal evidence of decrement on repetitive nerve stimulation and that petitioner tested negative for any antibody markers for MG. (ECF No. 70, pp. 18-19 (emphasis omitted) (citing Ex. P28, pp. 1012, 1479; Ex. P26, pp. 126-27).) Additionally, throughout the hearing, Dr. Steinman repeatedly testified that petitioner does not suffer from MG. (*E.g.*, Tr. 46, 53-54, 95, 98,100, 104-05, 109, 120.)

However, in his post-hearing reply brief, petitioner stresses that Dr. Smith diagnosed him with both MG and polyradiculopathy. (ECF No. 85, p. 2.) He argues that finding MG is an appropriate diagnosis does not warrant a conclusion that inflammatory polyradiculopathy is not also an appropriate diagnosis in this case. (*Id.* at 2-3.) Petitioner emphasizes that both Dr. Steinman and Dr. Price agreed that an individual could suffer from both MG and inflammatory polyradiculopathy. (*Id.* at 3 (citing Tr. 119-20, 240).)

While both experts did agree that it is possible that an individual could have both MG and inflammatory polyradiculopathy, petitioner ignores the fact that Dr. Steinman explicitly testified that it is far more likely in this particular case that the petitioner suffers from one or the other. (Tr. 119-20.) Accordingly, given the undersigned's conclusion that petitioner more likely than not suffers from MG, Dr. Steinman has effectively agreed that his preferred diagnosis of inflammatory polyradiculopathy is unlikely. Even if the undersigned were to overlook Dr. Steinman's testimony on this point, the analysis above explains why petitioner likely does suffer MG and likely does *not* suffer an inflammatory polyradiculopathy. Therefore, petitioner's suggestion that he suffers from both MG and inflammatory polyradiculopathy is unpersuasive.

h. *Althen* Analysis is Unnecessary

Petitioner did not offer any causal theory or argument implicating his flu vaccine as a cause of his MG. In fact, petitioner ultimately released his claim that his flu vaccine caused MG. (ECF No. 77.) Accordingly, no *Althen* analysis is necessary in this case. *Lombardi*, 656 F.3d at 1352-53. However, even if petitioner had continued to pursue his MG claim, it is unlikely he would have succeeded in preponderantly showing that his flu vaccine caused his condition. Prior petitioners have been unsuccessful in seeking to provide theories of causation establishing that the flu vaccine can cause or significantly aggravate MG. *E.g.*, *Kelly v. Sec'y of Health & Human Servs.*, No. 16-1548V, 2023 WL 3274159 (Fed. Cl. Spec. Mstr. May 5, 2023) (undersigned); *Smilo v. Sec'y of Health & Human Servs.*, No. 18-1585V, 2023 WL 3918397 (Fed. Cl. Spec. Mstr. May 15, 2023) (Dorsey); *Demore v. Sec'y of Health & Human Servs.*, No. 20-1265V, 2024 WL 4542934 (Fed. Cl. Spec. Mstr. Sep. 26, 2024) (Moran), *aff'd*, 175 Fed. Cl. 756 (2025).

VI. Conclusion

There is no question that petitioner has suffered. He has my sympathy, and I do not question his sincerity in bringing this claim. However, for all the reasons discussed above, I find that petitioner has not met his burden of proof in this case. Therefore, pursuant to § 300aa-12(d)(3)(A) and Vaccine Rule 10, this decision concludes that petitioner is not entitled to an award of compensation. Absent a timely motion for review, the Clerk is directed to enter judgment dismissing this case for insufficient proof in accordance with Vaccine Rule 11(a).

IT IS SO ORDERED.

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