In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: May 17, 2024

CINDY BARRIENTOS,	*	PUBLISHED
	*	
Petitioner,	*	No. 18-1899V
	*	
v.	*	Special Master Nora Beth Dorsey
	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Dismissal; Influenza ("Flu") Vaccine; Pre-Existing Condition; Significant Aggravation; Transverse Myelitis; Thoracic Myelopathy.
	*	
Respondent.	*	
	*	

Sylvia Chin-Caplan, Law Office of Sylvia Chin-Caplan, LLC, for Petitioner.
Felicia Langel, U.S. Department of Justice, Washington, DC, for Respondent.

DECISION[1]

On December 10, 2018, Cindy Barrientos ("Petitioner") filed a petition for compensation under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"), 42 U.S.C. § 300aa-10 et seq. (2018)[2] alleging that she suffered significant aggravation of her pre-existing transverse myelitis and thoracic myelopathy as a result of an influenza ("flu")

[1] Because this Decision contains a reasoned explanation for the action in this case, the undersigned is required to post it 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 Decision will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, the undersigned agrees that the identified material fits within this definition, the undersigned will redact such material from public access.

[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to -34 (2018). All citations in this Decision to individual sections of the Vaccine Act are to 42 U.S.C. § 300aa.

vaccination administered to her on December 11, 2015.[3] Petition at Preamble, ¶ 16 (ECF No. 1). Respondent filed his Rule 4(c) report on February 6, 2020, asserting that Petitioner had "not met her burden establishing entitlement to compensation under the terms of the Vaccine Act." Respondent's Report ("Resp. Rept.") at 1 (ECF No. 26).

After carefully analyzing and weighing the evidence presented in this case in accordance with the applicable legal standards, the undersigned finds that Petitioner has failed to provide preponderant evidence that her pre-existing condition was significantly aggravated by the flu vaccination she received on December 11, 2015. Thus, Petitioner has failed to satisfy her burden of proof under Loving v. Secretary of Health & Human Services, 86 Fed. Cl. 135, 142-44 (2009). Accordingly, the petition shall be dismissed.

I. ISSUES TO BE DECIDED

The parties stipulate that Petitioner was born on November 24, 1973, and that she received a flu vaccine on December 11, 2015. Joint Prehearing Submission ("Joint Submission"), filed Oct. 26, 2022, at 1 (ECF No. 93). They also stipulate that "prior to the administration of the flu vaccine on December 11, 2015, [Petitioner] suffered from a pre-existing thoracic myelopathy." Id. Further, they stipulate that after she received the flu vaccination on December 11, 2015, Petitioner's condition worsened as noted on her magnetic resonance imaging ("MRI") and by her treating physicians. Id. at 2. The parties stipulate that Petitioner suffered from her condition for more than six months and that she timely filed her petition within the applicable statute of limitations. Id.

The parties dispute the nature of Petitioner's condition before and after her flu vaccine. Joint Submission at 2. Petitioner "asserts that the flu vaccine that she received on December 11, 2015, resulted in a significant aggravation of her underlying [longitudinally extensive transverse myelitis]." Id. at 3. Respondent disputes that she suffered transverse myelitis and/or longitudinally extensive transverse myelitis "before or after the administration of the flu vaccine on December 11, 2015." Id. at 2. Respondent also disputes that the vaccine was the cause of her significantly worsened pre-existing condition. Id. Instead, Respondent attributes Petitioner's worsening to the "evolution of [her] pre-existing condition." Id.

[3] In the preamble of her petition, it was not clear that Petitioner's claim was significant aggravation, but in paragraph 16, she specifically asserted her claim based on significant aggravation. Petition at ¶ 16 (ECF No. 1). Additionally, in their joint submission, the parties specified that Petitioner was alleging significant aggravation. Joint Prehearing Submission ("Joint Submission"), filed Oct. 26, 2022, at 1-3 (ECF No. 93). The undersigned agrees that the facts presented raise a claim of significant aggravation.

II. PROCEDURAL HISTORY

Petitioner filed her petition and medical records on December 10 and December 12, 2018.[4] Petition; Petitioner's Exhibits ("Pet. Exs.") 1-9. On January 22, 2020, the case was reassigned to the undersigned. Notice of Reassignment dated Jan. 22, 2020 (ECF No. 23). Respondent filed his Rule 4(c) report arguing against compensation on February 6, 2020. Resp. Rept. at 1.

On September 9, 2020, Petitioner filed an expert report from Dr. Carlo Tornatore. Pet. Ex. 13. Respondent filed an expert report from Dr. Adil Javed on April 14, 2021. Resp. Ex. A. The undersigned held a Rule 5 conference on June 22, 2021. Rule 5 Order dated June 22, 2021 (ECF No. 47). The undersigned did not reach a preliminary opinion as to causation. Id. at 2. Given the complexity of the clinical course and MRI findings, a hearing was recommended. Id.

On September 30, 2022, Petitioner submitted her pre-hearing brief. Pet. Pre-Hearing Brief ("Br."), filed Sept. 30, 2022 (ECF No. 78). Respondent filed a pre-hearing brief on October 21, 2022. Resp. Pre-Hearing Br., filed Oct. 21, 2022 (ECF No. 86). An entitlement hearing was held November 16 and November 17, 2022. Order dated Nov. 17, 2022 (ECF No. 98); Transcript ("Tr.") 1, 148. Following the hearing, the undersigned requested the parties to file supplemental expert reports commenting on Petitioner's treating physician Dr. Elliot Frohman's diagnostic statement from 2019 and explaining how this statement affects their respective opinions as to diagnosis and causation. Order dated Nov. 17, 2022.

Respondent filed a supplemental expert report from Dr. Javed on December 16, 2022. Resp. Ex. C.[5] Instead of a supplemental expert report,[6] Petitioner responded to the Court's request by citing medical records submitting a brief. Pet. Status Rept., filed Mar. 6, 2022 (ECF No. 110); Pet. Br. Addressing the Court's Order ("Pet. Supplemental Br."), filed Mar. 13, 2023 (ECF No. 112). On June 5, 2023, Petitioner filed a post-hearing brief. Pet. Post-Hearing Br., filed June 5, 2023 (ECF No. 117). On August 7, 2023, Respondent filed a post-hearing brief. Resp. Post-Hearing Br., filed Aug. 7, 2023 (ECF No. 118).

The matter is now ripe for adjudication.

[4] Petitioner continued to file medical records throughout the course of litigation.

[5] Respondent labeled this as exhibit B however, Respondent already had an exhibit B and it is Dr. Javed's CV. Therefore, the undersigned will refer to Dr. Javed's supplemental expert report as exhibit C.

[6] In February 2023, Petitioner filed a status report indicating her expert was on medical leave until June/July 2023 and would be unable to draft a supplemental report. Pet. Status Rept., filed Feb. 2, 2023 (ECF No. 108).

III. MEDICAL TERMINOLOGY

A. Transverse Myelitis

Transverse myelitis "is a rare clinical syndrome in which an immune-mediated process causes neural injury to the spinal cord, resulting in varying degrees of weakness, sensory alterations[,] and autonomic dysfunction." Pet. Ex. 18 at 1.[7] The Transverse Myelitis Consortium Working Group proposed diagnostic criteria including "bilateral sensory, motor, or autonomic dysfunction attributable to the spinal cord, a clearly defined sensory level[,] and peaking of symptoms within [four] hours and 21 days." Id. "In addition, evidence of an inflamed spinal cord (e.g. cerebrospinal fluid (CSF) pleocytosis,[8] elevated [immunoglobulin G ("IgG")] index[9] or gadolinium enhancement[10] by [MRI] and exclusion of [] compressive etiology by neuroimaging should be observed." Id.

Petitioner's expert, Dr. Tornatore, opined that transverse myelitis is "a generic term for inflammation of the spinal cord." Tr. 11. Transverse myelitis does not indicate an etiology, "just that the [spinal] cord is inflamed." Tr. 127. It can be caused "by a host of different things," including medication toxicity, tumors, compression myelopathy, and autoimmune diseases. Tr. 11. Transverse myelitis is diagnosed by MRI findings which show "characteristic [] signal changes in the spinal cord suggesting inflammation or edema" of the spinal cord. Tr. 127, 163-64. MRI findings related to transverse myelitis include "spinal cord enlargement,

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[7] N. Agmon-Levin et al., Transverse Myelitis and Vaccines: A Multi-Analysis, 18 Lupus 1198 (2009).

[8] Pleocytosis is the "presence of a greater than normal number of cells in the [CSF]." Pleocytosis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=39556 (last visited Apr. 25, 2024).

[9] "Serum protein quantification is used to detect and monitor the course of [certain] diseases [including] autoimmune diseases." Kathleen Deska Pagana & Timothy J. Pagana, Blood Studies, in Mosby's Manual of Diagnostic and Laboratory Tests 10, 279 (6th ed. 2018). "Proteins within the blood are made up of albumin and globulin. Several types of globulin exists, one of which is gamma globulin. Antibodies are made up of gamma globulin protein and are called immunoglobulins. There are many classes of immunoglobulins (antibodies)." Id. at 280. IgG "constitutes approximately 75% of the serum immunoglobulins; therefore[,] it constitutes the majority of circulating blood antibodies." Id.

[10] "A contrast medium called gadolinium is a paramagnetic enhancement agent that crosses the blood-brain barrier. It is especially useful for distinguishing [certain] abnormalities." Miscellaneous Studies, in Mosby's Manual of Diagnostic and Laboratory Tests, supra note 9, at 1024, 1060.

intramedullary[11] increased T2 signal lesions,[12] and variable enhancement." Pet. Ex. 13 at 28 (quoting Pet. Ex. 39 at 1).[13] These abnormalities may be "diffuse and extend over several spinal levels." Id. (quoting Pet. Ex. 39 at 1). In addition to MRI, a lumbar puncture or "spinal tap" is "done to try to sort out what the source of [] inflammation" is by analysis of the CSF. Tr. 11-12.

Respondent's expert, Dr. Javed, opined that compression of the spinal cord can cause inflammation. Tr. 301. When the spinal cord is squeezed, "some of the fibers die, [and] some of the fibers [] inflame." Id. Inflammation can also occur in response to infection. Tr. 190, 245, 263. An infection can stimulate the immune response which causes transverse myelitis. Tr. 245.

In transverse myelitis, "[w]hen the inflammatory lesion extends across more than three vertebral segments longitudinally, it is commonly referred to as longitudinally extensive transverse myelitis." Pet. Ex. 13 at 27 (citing Pet. Ex. 16 at 2).[14]

B. Transverse Myelopathy

Transverse myelopathy is a "myelopathy[15] that extends across the spinal cord." Transverse Myelopathy, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=91256 (last visited Apr. 25, 2024).

According to Respondent's expert, Dr. Javed, compressive myelopathy occurs when there is "compression from [an] outside structure, the bone, the calcification, the disc material." Tr. 160. There is water inside and around and the center of the spinal cord. Id. As the spinal cord is compressed by the outside (disc material), it "squeezes," and "stifles the spinal cord, and the spinal cord's blood supply is also stifled, and the area where the blood supply is occurring

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[11] Intramedullary means "within the spinal cord." Intramedullary, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=25811 (last visited May 8, 2024).

[12] T1 and T2 are technical terms applied to different MRI methods used to generate magnetic resonance images." T1 and T2 Lesions, MS Austl., https://www.msaustralia.org.au/glossary/t1-t2-lesions/ (last visited May 8, 2024). Different methods are "used to detect different structures or chemical in the central nervous system. . . . A T2 MRI image provides information about disease burden or lesion load (the total amount of lesion area, both old and new)." Id.

[13] Rohit Bakshi & John C. Mazziotta, Acute Transverse Myelitis After Influenza Vaccination: Magnetic Resonance Imaging Findings, 6 J. Neuroimaging 248 (1996). This is also cited as Resp. Ex. A3.

[14] Wafa Akkad et al., Longitudinally Extensive Transverse Myelitis Following Vaccination with Nasal Attenuated Novel Influenza A(H1N1) Vaccine, 67 Archives Neurology 1018 (2010).

[15] Myelopathy is "any of various functional disturbances or pathologic changes in the spinal cord, often referring to nonspecific lesions in contrast to the inflammatory lesions of myelitis." Myelopathy, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=32732 (last visited Apr. 25, 2024).

starts to become ischemic and dies off." Tr. 160-61. For example, if you "squeeze a water bottle or a[] balloon, there is ballooning, congestion, upwards and downwards. . . . This is compressive myelopathy. And if it's not relieved then it gets worse and worse over time." Tr. 161.

Compressive myelopathy and spinal cord ischemia are related. Tr. 303. When the spinal cord is compressed, the "blood supply is [] stifled . . . [and] become[s] ischemic and dies off." Tr. 160-61. Compressive myelopathy "decrease[s] the [perfusion] of blood in that site." Tr. 304.

IV. FACTUAL SUMMARY

A. Summary of Medical Records[16]

1. Pre-Vaccination Records

Petitioner was born on November 24, 1973. Pet. Ex. 1 at 7. Petitioner received at least two other flu vaccinations prior to the one at issue: one on September 27, 2013, and another on October 2, 2014. Id. at 556. Prior to the vaccination at issue, Petitioner had an extensive and complicated medical history.

Relevant to the issues raised in this case, on March 24, 2014, Petitioner saw Kathryn Drake Hart, M.D., her primary care provider ("PCP") for thoracic back pain that had reportedly started in early February 2014. Pet. Ex. 1 at 227. She reported pain "all across the thoracic back area, [] tingling radiating down [her] rib cage down to hips and all the way down to the feet on both sides. [She] [a]lso ha[d] decreased sensation under both breasts at the lower rib cage all around the torso into the back." Id. Petitioner was assessed with thoracic back pain, lumbar radicular pain, hypertension, chest discomfort, and an abnormal urinalysis. Id. at 228-29. Dr. Hart ordered thoracic and lumbar spine x-rays, and prescribed meloxicam, tramadol, and cyclobenzaprine for Petitioner's back pain. Id.

Petitioner returned to Dr. Hart on April 1, 2014. Pet. Ex. 1 at 289. Petitioner reported that her back pain was improving, but "[s]he still [had] achiness in her thoracic back area, and tingling down her flanks, lateral thighs and down to her feet. Also with numbness in the same area intermittently," and she "frequently [felt] cold." Id. It was noted that Petitioner "relate[d] all of the symptoms to having had a Mirena placed," and which was removed "about a month ago." Id. Dr. Hart noted that Petitioner's thoracic back pain was a "somewhat puzzling presentation." Id. at 290. The X-rays showed mild arthritis but were otherwise normal. Id. Dr. Hart noted that she had a lengthy discussion with Petitioner and that Petitioner "[c]ould consider MRI given the paresthesias that she's had in her legs, but she [was] having steady improvement of her symptoms." Id. Dr. Hart suggested a follow-up visit in two to three weeks, and if her symptoms did not improve, they would "consider MRI and neurology referral." Id.

[16] Sections of this summary are largely taken from Respondent's Rule 4(c) report and Dr. Tornatore's initial expert report. See Resp. Rept. at 2-14; Pet. Ex. 13 at 2-27. Additional factual summaries are set forth in the parties' briefs. See Pet. Pre-Hearing Br. at 1-16; Resp. Pre-Hearing Br. at 3-10; Pet. Post-Hearing Br. at 1-19; Resp. Post-Hearing Br. at 3-11.

On April 9, 2014, Petitioner sent an e-mail to Dr. Hart, stating that she was having pain "on [her] left back side" and "some numbness in [her] mid[-]section and some in [the] outer part of [her] legs." Pet. Ex. 1 at 304. Petitioner wrote that she "[at] times . . . found [herself] unstable while starting to walk." Id. On April 10, 2014, Dr. Hart ordered an MRI of Petitioner's lumbar spine due to lumbar radicular pain, paresthesia of both legs, and weakness of both legs. Id. at 307. A note from April 16, 2014, indicated that Petitioner's insurance had "denied the MRI orders." Id. at 304.

Petitioner saw Dr. Hart again on April 21, 2014. Pet. Ex. 1 at 320. Regarding Petitioner's thoracic back pain, Dr. Hart noted it had been present for the last two months and that it had "[u]nusual presentation [and] puzzling symptoms." Id. at 321. Dr. Hart referred Petitioner to neurology, noting she had "[t]horacic back pain for [two] months, ha[d] paresthesia of flanks, legs, feet. Ha[d] occasional sensation of weakness in legs. Exam[ination] show[ed] decreased sensation in legs, strength OK. Insurance ha[d] declined MRIs." Id. at 319.

Two days later, on April 23, 2014, Petitioner saw neurologist Jeffrey Tramonte, M.D. Pet. Ex. 1 at 326. Dr. Tramonte summarized Petitioner's history as follows:

> [Petitioner] is a 40-year-old woman who works in the call center for Lone Star Circle of Care. . . . In early February 2014, she developed severe upper-mid thoracic back pain with tingling from about her bra line down to her toes. Since that time her pain and tingling have been constant. Symptoms are worse with heat exposure (Uhtoff phenomenon). In addition, she has urinary urgency and problems with initiating urination. She also complains of weakness in her legs, unsteadiness when standing and walking, and she complains of vaginal numbness and sexual dysfunction.

Id. at 334. Neurological examination was abnormal in that Petitioner's reflexes were "pathologically brisk" in both lower limbs and plantar response was "toe[s] upgoing bilaterally." Id. at 337. Sensation was "decreased to light touch to [] T4 spinal level bilaterally," and gait and balance were "notable for [] mild spastic paraparesis." Id. Dr. Tramonte assessed Petitioner with T4 thoracic myelopathy, with onset in early February 2014. Id. He also listed differential diagnoses including multiple sclerosis ("MS"), thoracic transverse myelitis, thoracic cord tumor, herniated thoracic disc, and infection. Id. He ordered an MRI of the thoracic spine, cervical spine, and brain "all with [gadolinium] per MS protocol." Id. Dr. Tramonte noted that he "[a]nticipate[d] this is most likely MS and discussed with patient." Id.

Petitioner underwent an MRI of the brain on April 29, 2014. Pet. Ex. 1 at 377. The MRI showed "[s]cattered punctate white matter foci, greater than expected for the patient's age." Id. It was noted that the finding was "nonspecific and may be seen with microangiopathic disease, vasculopathy, demyelination[,] and migraines." Id. An MRI of the cervical spine revealed "[s]pondylotic changes with multilevel cord contact; otherwise unremarkable evaluation of the cervical cord." Id. at 385. The MRI of Petitioner's thoracic spine was abnormal and showed a

"[p]artially calcified central/right paracentral ventral epidural mass at T5/T6.[17] Given its proximity to the disc space, this may represent a calcified extrusion; a meningioma may have a similar appearance. Associated myelopathic signal change [was] noted." Id. at 392. A surgical consultation was recommended due to the mass. Id.

On May 19, 2014, Petitioner saw neurologist Ekokobe Fonkem, D.O. Pet. Ex. 3 at 22. Dr. Fonkem noted that the differential diagnosis for Petitioner's T5-6 spinal mass was "meningioma versus schwannoma." Id. Dr. Fonkem advised Petitioner that she could have surgery or have serial monitoring with spine MRIs, and Petitioner stated she would like to see a spinal surgeon. Id.

On May 23, 2014, Petitioner saw David Garrett, Jr., M.D., a neurosurgeon. Pet. Ex. 3 at 28. Petitioner reported thoracic back pain since February 2014, and reported feeling "a band around her just under the area of her breasts" and "numb from this area to her feet." Id. Dr. Garrett noted that Petitioner had "markedly diminished proprioception in toes of both feet" and hyperreflexia in her knee and ankle joints. Id. at 31. Dr. Garrett noted that regardless of whether Petitioner's spinal mass was calcified disc or meningioma, "it need[ed] to be resected due to her myelopathy."[18] Id. at 32.

Petitioner underwent spinal surgery on June 27-28, 2014. Pet. Ex. 3 at 195-200. It was noted that the time spent in the operating room and difficulty of the case was increased "by at least 50%" due to Petitioner's "extreme obesity with a body mass index of 58." Id. at 200. The post-operative diagnosis indicated that Petitioner had suffered a calcified herniated thoracic disc at T5-T6. Id. at 195. During surgery, "the motor-evoked potential signal was lost from the right leg area. The left leg area remained intact with signal throughout." Id. at 200.

On June 29, 2014, the date after her surgery, Dr. Garrett noted that Petitioner had "some movement in her right foot, but no ability to move [her] right leg." Pet. Ex. 3 at 200.

A physical therapy ("PT") record from June 29, 2014 noted that Petitioner was on bed rest due to a CSF leak. Pet. Ex. 3 at 206. Her initial PT evaluation took place on July 1, 2014. Id. at 902. It was noted that Petitioner was still on bed rest, but she may be ready to begin the

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[17] The spinal cord is the "part of the central nervous system that is lodged in the vertebral canal. It extends from the foramen magnum, where it is continuous with the medulla oblongata, to the upper part of the lumbar region, ending between the twelfth thoracic and third lumbar vertebrae, often at or near the first and second lumbar vertebrae." Medulla Spinalis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=88447 (last visited Apr. 30, 2024). The spinal cord is referenced by its location within the spinal column, which is divided into parts, cervical, thoracic, lumbar, sacral, and coccygeal. Id. Abbreviations are used throughout this Decision. For example, T5/T6 or T5-T6 stand for the level of the spinal cord at the thoracic vertebral level 5 and 6.

[18] MRI of thoracic spine showed at T5/T6 a "central/right paracentral ventral epidural mass deforming the ventral cord occupying approximately 50% of the cross-sectional area of the canal." Pet. Ex. 3 at 31.

following day. Id. At PT on July 3, 2014, Petitioner reported that her right leg did "not work well." Id. at 901. It was also noted that Petitioner was beginning to mobilize but could not lift her right leg for transfers. Id. at 902. She began ambulating on July 5, 2014. Id. at 898. Petitioner decided that she wished to complete her PT at an outpatient facility, and she was discharged home on July 6, 2014. Id. at 146. She was instructed to follow-up with Dr. Garrett in two weeks for a post-operative wound check. Id.

On July 9, 2014, three days after discharge, Petitioner called reporting that she had "lost all balance and mobility again to the right leg since yesterday," stating it was "like before surgery." Pet. Ex. 3 at 7779. Petitioner reported that she could feel touch to her leg but she was unable to move her leg or wiggle her toes. Id. She was instructed to go to the emergency department ("ED") immediately. Id. Petitioner subsequently had an extensive inpatient admission, from July 9 to August 1, 2014. See generally Pet. Ex. 3. MRI of the thoracic spine done July 9, 2014 showed "postoperative changes of discectomy" at T5-T6. Id. at 2083. "Within the adjacent lung [was] a loculated rim-enhancing fluid collection representing an abscess." Id. There was "abnormal enhancement in the right paraspinal soft tissues at T5-T6 concerning for infection. Additionally, there [was] abnormal enhancement in the epidural space of the thoracic spine extending from T2-T3 inferiorly. There [was] a rim-enhancing fluid collection in the dorsal epidural space extending from T4-T11 representing an epidural abscess." Id. At T5-T6, there also appeared to be "a recurrent central and right paracentral disc herniation which results in moderate to severe spinal canal stenosis where there [was] significant cord compression and cord deformity without cord signal abnormality." Id. at 2083-84. The impression was

> 1. Extensive epidural abscess extending from T4-T11 which primarily involves the dorsal epidural space and results in multilevel mild and moderate spinal canal stenosis.
> 2. Large rim-enhancing loculated fluid collection in the right thoracic cavity representing an empyema.[19] There [was] extension of abnormal enhancement into the right paraspinal soft tissues at T5 and T6 likely representing infection.
> 3. Recurrent T5-T6 right paracentral disc herniation which results in moderate to severe spinal canal stenosis.

Id. at 2084.

[19] An empyema is an abscess ("a localized collection of pus within tissues, organs, or confined spaces") or pleural effusion ("the presence of fluid in the pleural space") containing pus. Empyema, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=16115 (last visited May 9, 2024); Abscess, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=185 (last visited May 9, 2024); Pleural Effusion, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=72555 (last visited May 9, 2024).

Petitioner underwent a number of surgical procedures and suffered significant post-operative complications, including but not limited to sepsis, epidural abscess[20] from T4-11, right lung loculated empyema, possible right paraspinal abscess at T5-6, paraplegia, and urinary incontinence. See generally Pet. Ex. 3. Petitioner's post-operative course was complicated by methicillin-resistant *Staphylococcus aureus* ("MRSA")[21] bacteremia, MRSA epidural abscess, right chest empyema with chest tube placement, atrial fibrillation, and persistent CSF leak requiring intubation and intensive care unit ("ICU") admission. Id. at 2021-29, 7865-66. Records note "she became paraplegic on August 26, 2014." Id. at 7850. On or about August 1, 2014, Petitioner was discharged to inpatient rehabilitation. See id. at 7865.

On August 26, 2014, Petitioner presented to the ED with worsening lower extremity weakness since August 25, 2014. See Pet. Ex. 3 at 7865. An MRI of the thoracic spine done that same day found

> [i]nterval development of cord expansion and associated increased intramedullary signal essentially throughout the thoracic spine without significant enhancement. This appearance is nonspecific and may [be] the result of venous congestion/edema, cord ischemia or potentially infectious myelitis. . . . Interval increase in T5 and T6 vertebra (to include the posterior elements) marrow signal and enhancement with further endplate destruction [] with now both anterior and posterior disc herniations compressing the spinal cord compatible with discitis osteomyelitis.[22]

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[20] An epidural abscess is "a collection of pus located between the dura mater and surrounding bone." Epidural Abscess, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=54350 (last visited Apr. 30, 2024). Dura mater is "the outermost, toughest, and most fibrous of the three membranes (meninges) covering the brain and spinal cord."). Dura Mater, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=15060 (last visited May 9, 2024).

[21] Staphylococcus aureus is "a species comprising the yellow-pigmented, coagulase-positive pathogenic forms of the genus; it causes serious suppurative infections and systemic disease, . . . and has developed resistance to nearly all classes of antibiotics." Staphylococcus Aureus, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=108211 (last visited May 8, 2024). Methicillin is "a semisynthetic penicillin, used intravenously or intramuscularly as an antibacterial in resistant staphylococcal infections." Methicillin Sodium, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=30916 (last visited May 9, 2024).

[22] Osteomyelitis is "inflammation of bone caused by infection, usually by a pyogenic organism, although any infectious agent may be involved. It may remain localized or may spread through the bone to involve the marrow, cortex, cancellous tissue, and periosteum." Osteomyelitis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=35862 (last visited May 9, 2024).

Id. at 7812. An MRI of the lumbar spine also done the same day found "[i]nterval development of loculated subdural fluid collection likely representing effusion anteriorly displacing the cauda equina[23] nerve roots throughout the lumbar spine." Id. There was a "drainable fluid collection to suggest epidural abscess." Id.

Also on August 26, a neurosurgical consultation noted that Petitioner had presented to the ED "this evening with complaint of bilateral lower extremity weakness." Pet. Ex. 3 at 7803. Petitioner had "minimal movement of her bilateral [lower extremities] with intact minimal ankle plantarflexion/dorsiflexion on the left. She [was] unable to perform this against resistance. [Petitioner] ha[d] very subtle movement of her right lower extremity." Id. at 7805. The MRI findings were reviewed and it was noted that since the prior examination, there had been "interval development of [c]entral increased cord signal and expansion extending from the C7-T1 level to the T11-T12 level without discrete associated intramedullary enhancement." Id. at 7806. "Discitis osteomyelitis ha[d] progressed centered at T5-T6 with associated endplate destruction, marrow signal abnormality and both anterior and posterior disc herniation [was] compressing the spinal cord at the T5-T6 level," and "[a]bnormal signal enhancement," involving the right more than the left, at "T5-T6 neural foramina." Id. The impression was

> 1. Interval development of cord expansion and associated increased intramedullary signal essentially throughout the thoracic spine without significant enhancement. This appearance is nonspecific and may the result of venous congestion/edema, cord ischemia[,] or potentially infectious myelitis.
> 2. Overall interval decrease in size of the epidural abscess seen on 7/9/2014 exam[ination] with residual signal abnormality and enhancement most compatible with phlegmon within the predominantly posterior epidural space.
> 3. . . .
> 4. Interval increase in T5 and T6 vertebra (to include the posterior elements) marrow signal and enhancement with further endplate destruction [] with now both anterior and posterior disc herniations compressing the spinal cord compatible with discitis osteomyelitis.

Id. at 7806-07.

On September 9, 2014, Petitioner was seen by Sangeetha Ranganath, M.D., an infectious disease specialist. Pet. Ex. 3 at 7865. Dr. Ranganath noted that Petitioner had suffered disseminated MRSA infection, MRSA bacteremia, and that her CSF cultures were positive for MRSA. Id. at 7865. Dr. Ranganath noted,[24]

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[23] Cauda equina is "the collection of spinal roots that descend from the lower part of the spinal cord and are located within the lumbar cistern of the caudal dural sac." Cauda Equina, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=63754 (last visited May 9, 2024).

[24] For a complete discussion of Petitioner's post-operative course, see Pet. Ex. 3 at 7865-66 (an extensive note authored by Dr. Ranganath).

> 1. Spinal epidural abscess due to MRSA, thoracic, [status post] decompression on 7/10/14
> 2. T5-6 discitis/osteomyelitis, interval progression on the imaging but improving clinical markers
> 3. Right lung empyema, [status post] decortication on 7/10, with pleural fluid [culture] positive for MRSA
> 4. MRSA bacteremia in July 2014 due to above all
> 5. Bilateral lower ext[remity] weakness
> 6. Bowel/bladder incontinence due to above
> 7. Morbid obesity.

Id. at 7870. Petitioner was able to move her left foot toes but not her right foot. Id. at 7866. She remained incontinent. Id. On September 16, 2014, Petitioner requested that she be discharged to home. Id. at 7884.

On September 26, 2014, Petitioner saw Dr. Ranganath in follow-up. Pet. Ex. 3 at 7914. At this time Petitioner had "sensations in her left foot and [was] able to move [her] left foot but [she was] still not able to move her right foot." Id. She had urinary retention and required a catheter. Id.

On September 29, 2014, computed tomography ("CT") scan of Petitioner's chest continued to show "osteomyelitis discitis of T5 and T6 and central stenosis and compression of the spinal cord." Pet. Ex. 1 at 483.

At an October 2, 2014 follow-up visit with Dr. Ranganath, Petitioner had sensations in her left foot but was still unable to move her right foot. Pet. Ex 3 at 7928. She was told to follow up in one week for labs and a CT chest scan. Id. at 7931. A flu shot was administered.[25] Id. She had a urology follow-up the same day. Id. at 7943. It was noted she was in a wheelchair and did "not walk at all" and had "numbness from her waist down." Id. She could not "feel to void or defecate." Id.

On October 14, 2014, at a neurosurgical follow-up examination, assessment was "[p]araplegia due to poorly controlled osteomyelitis, though the epidural abscess component has improved." Pet. Ex. at 7981. Dr. Garrett discussed with Petitioner that

> [s]he could experience further improvement in neurologic function, as the infection is non-detected. . . . Return of function is favored as her spinal cord shows more congestion rather than compression. [Dr. Garret] fe[lt] that the risk of further CSF-cutaneous fusion makes it unlikely that she would benefit from further decompressive surgery. She has risk of instability with further surgery []. .

[25] Petitioner is not alleging any injury related to this flu shot.

> . . Her pattern of spinal cord injury [was] a Brown-Sequard[26] type, which carries a more favorable prognosis.

Id.

An MRI of the thoracic spine done October 21, 2014 demonstrated a "[d]estructive process centered in the T5/T6 disc space with associated endplate erosion and patchy marrow edema involving the adjacent vertebral bodies [were] similar to the prior exam[ination]." Pet. Ex. 1 at 535. Associated mild anterolisthesis was unchanged. Id. "Residual T5/T6 endplate ridging [was] again noted, contacting the ventral cord. Epidural and paraspinal enhancement at this level [was] similar representing residual phlegmon and/or granulation. Intramedullary T2 prolongation with associated mild cord expansion [had] improved from the prior exam[ination], extending to the T9/T10 level, previously to T11/T12." Id. No discrete peripheral enhancing fluid collection was seen within the paraspinal or epidural soft tissues and the previously noted loculated right pleural fluid collection was no longer seen. Id. The impression was "[s]imilar configuration of spondylo-discitis at T5/T6. No discrete drainable fluid collection [was] seen. Improved intramedullary T2 prolongation as described." Id.

Petitioner saw her PCP, Dr. Hart, in follow-up on November 3, 2014. Pet. Ex. 1 at 562. Dr. Hart noted that Petitioner had back surgery with multiple complications. Id. Petitioner reported that she was discharged from rehabilitation "[ab]out a month ago." Id. She reported that she initially had no movement or sensation in her lower extremities and she was currently unable to stand or walk, but she was beginning to be able to pull to stand with assistance. Id.

Petitioner had outpatient PT evaluation on March 24, 2015. Pet. Ex. 1 at 604. It was noted that Petitioner had paraplegia following a spinal cord injury, bilateral leg weakness, spasticity, and difficulty walking. Id. Petitioner reported that she was ambulatory after her surgery in June 2014; however, she had complications after surgery, and had subsequent surgery on July 9, 2014. Id. at 605. She had been wheelchair bound since that time. Id. She had about six weeks of inpatient rehabilitation. Id. Assessment showed that Petitioner had "[right] [lower extremity] extensor spasticity and [left] [lower extremity] flexor spasticity, . . . limiting her full knee [range of motion] bilaterally." Id. at 608. Petitioner was "unable to ambulate even short household distances." Id. Petitioner also had weakness and poor endurance limiting her ability to walk. Id. at 611. She required assistance transferring out of the wheelchair and with bed mobility due to upper extremity and core weakness. Id. at 608, 619.

On March 26, 2015, Petitioner's assessments included "[s]pasticity." Pet. Ex. 1 at 628. Petitioner stated she had good days and bad days, when she was "less confident in use of her legs." Id. When standing "fully erect[,] the spasticity ma[de] her knees buckle." Id. The therapist noted "spasticity in her left [lower extremity] decreas[ed] [her] ability to tolerate standing for more than [two] minutes." Id. at 629.

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[26] Brown-Sequard is "a syndrome due to damage of one half of the spinal cord, resulting in ipsilateral paralysis and loss of discriminatory and joint sensation, and contralateral loss of pain and temperature sensation." Brown-Séquard Syndrome, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=110337 (last visited May 9, 2024).

Petitioner underwent urodynamic testing on June 1, 2015 "to characterize her neurogenic bladder, urinary leakage, and incomplete bladder emptying." Pet. Ex. 3 at 8178. Petitioner was currently "catheterizing [three] to [four] times per day and leaking between catheterization[s]." Id.

On July 24, 2015, Petitioner was using a wheelchair and a rolling walker. Pet. Ex. 1 at 955. It was noted she had become independent with many of her activities of daily living and had begun to walk unassisted with her walker. Id. Petitioner was going to continue therapy independently at her home, and the therapist noted that Petitioner would be reevaluated in November 2015. Id.

Petitioner saw her PCP on September 17, 2015. Pet. Ex. 1 at 1032. She reported that she was followed closely by her neurosurgeon, infectious disease specialist, physical therapist, and urologist. Id. Petitioner reported "very slow but steady improvement in her strength and walking ability." Id. Dr. Hart noted that Petitioner was still wheelchair-bound at this time. Id. at 1033. Petitioner had follow-up visits with her neurosurgeon on October 7, 2015, and her physical therapist on November 5 and December 2, 2015, and she was discharged from PT on December 10, 2015. Id. at 1078, 1104; Pet. Ex. 3 at 8215.

2. Vaccination and Post-Vaccination Records

On December 11, 2015, Petitioner saw her PCP for a follow-up visit. Pet. Ex. 1 at 1126. Petitioner reported very slow but steady improvement, but she reported that over "the last several weeks," her "legs [were] getting tight and stiffening at night" and she was having "noticeable spasms." Id. Dr. Hart referred Petitioner to Dr. Tramonte for an evaluation of muscle spasms of her lower legs. Id. at 1129, 1180. Petitioner received a flu vaccination at this visit. Id. at 1129; Pet. Ex. 9 at 9.

On December 18, 2015, seven days after the flu vaccine, Petitioner had a PT appointment, reporting that her left extremity "spasticity [was] worse today than previous[ly]." Pet. Ex. 1 at 1148. Petitioner reported she had "been unable to stand all day due to her 'good leg' being unreliable and not tolerating weight bearing." Id.

Petitioner reported similar problems at PT on December 30, 2015. Pet. Ex. 1 at 1170. Petitioner reported "continued inability to rely on [left lower extremity] for weight bearing" and "overall increase in spasticity making it difficult to transfer as independently as she had [] before." Id. She reported her husband had been helping her with stretching. Id. During the session, Petitioner was able to transfer, stand, and pivot, whereas previous session she was unable to. Id. at 1171. "Significant increase in extensor spasm tone" over the last two sessions was noted. Id.

On December 30, 2015, Petitioner saw neurologist Dr. Tramonte due to worsening weakness and spasticity. Pet. Ex. 1 at 1181. Dr. Tramonte noted that Petitioner had "worsening left leg weakness, spasticity, and sensory loss since the middle of December 2015." Id. Dr. Tramonte noted that Petitioner had "chronic urinary incontinence and has not noticed any recent

change to bowel or bladder control" and "chronic back pain that [was] unchanged." Id. MRIs of the thoracic and lumbar spine were ordered to further evaluate her condition. Id. at 1185. In his order for MRIs, Dr. Tramonte noted that the reason for the imaging was "worsening left lower extremity weakness. Eval[uate] for new compression." Id. at 1179. Dr. Tramonte's assessment was

> T4 thoracic myelopathy due to calcified dis[c] herniation, [status-post] decompression, followed by a complicated course of infection with empyema, abscess[,] and compression of the conus medullaris and cauda equina in 2014. Her chief complaint at this time is worsening weakness, spasticity, and sensory loss in the left leg since the middle of Dec[ember] 2015. This could be the natural evolution of thoracic cord compression with resolving infection/compression of the conus and cauda equina versus a new compressive lesion. . . . If there are no new lesions/compression [seen on MRI], then treatment will include referral for aquatic therapy and Baclofen for spasticity.

Id. at 1185.

An MRI of the thoracic spine was performed on January 8, 2016. Pet. Ex. 1 at 1211. It revealed "[i]nterval development of expansile T2 prolongation/edema within the thoracic cord extending between T1/T2 and T10. Associated intramedullary syringohydromyelia[27] [was] noted at T6 and T7 without enhancement." Id. It was noted that the findings were "nonspecific and may be secondary to altered CSF flow dynamics, transverse myelitis[,] and cord ischemia." Id.

On January 13, 2016, Petitioner saw neurosurgeon Dr. Garrett. Pet. Ex. 3 at 8249. Dr. Garrett's impression was "[t]horacic transverse myelitis due to either prior compression of spinal cord, meningitis, or even as autoimmune phenomenon from flu vaccine." Id. at 8250. He further noted that surgical intervention was not warranted and that "[t]his process may be inflammatory and [could] resolve with time, possibly." Id. He advised Petitioner to have another MRI in six months and to discontinue her antibiotics because her osteomyelitis appeared to have resolved. Id.

Petitioner returned to Dr. Tramonte for follow-up on January 20, 2016. Pet. Ex. 1 at 1250. Dr. Tramonte noted Petitioner's MRIs "showed worsening pre-existing signal abnormality

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[27] Syringohydromyelia or syringomyelia "a slowly progressive syndrome of cavitation in the central segments of the spinal cord, generally in the cervical region, but sometimes extending . . . down into the thoracic region; it may be of developmental origin, arise secondary to tumor, trauma, infarction, or hemorrhage, or be of unknown cause." Syringomyelia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=48639 (last visited May 8, 2024). "It results in neurologic deficits, usually segmental muscular weakness and atrophy with a dissociated sensory loss (loss of pain and temperature sensation, with preservation of the sense of touch), and thoracic scoliosis is often present." Id. It is sometimes referenced as a "syrinx." Id. The undersigned uses syringohydromyelia and syringomyelia interchangeably throughout this Decision.

in the thoracic spinal cord from T1-T10, a change that initially was present after developing a T4-T11 epidural abscess, along with new syringomyelia at T6/7. [He] suspect[ed] the changes [were] ischemic in nature." Id. at 1254.

Petitioner had a follow-up MRI of her thoracic spine on July 1, 2016. Pet. Ex. 3 at 8321. The MRI revealed the following:

> 1. Interval progression of abnormal signal and expansion of the mid thoracic cord with abnormal signal extending caudally through the T11 level. This has progressed since the prior study at which time the abnormal signal extended through the midportion of T10. These findings are consistent with mild progression of long segment myelomalacia.[28]
> 2. Focal kyphosis[29] at T5-T6 with chronic anterior wedging of T6. T5-T6 interbody fusion.
> 3. Moderate C5-C6 spinal canal stenosis due to posterior projecting osteophytes.
> 4. The cystic myelomalacia at T6 has slightly decreased in size when compared to the prior exam[ination].
> 5. Moderate multilevel spondylosis in the lower thoracic spine consisting of facet hypertrophy and ligamentum flavum thickening.
> 6. No abnormal enhancement in the thoracic spine.

Id.

Dr. Garrett next saw Petitioner on July 12, 2016. Pet. Ex. 3 at 8348. He noted that Petitioner "received a flu shot about [six] weeks prior to her onset of progressive loss of [lower extremity] strength." Id. His impression was that Petitioner was suffering a "[t]horacic myelopathy." Id. at 8349. He noted that the "recent regression in lower extremity strength correlates with the prolonged T2 signal within the spinal cord but cannot tell if it is new ischemia or myelitis. Process undergoing is unclear to me, but no surgical intervention is warranted at this point. Her disc area operated on is now fused. Kyphosis should not progress." Id.

On July 25, 2016, Petitioner saw Dr. Tramonte who noted that the new MRI "findings are thought to represent expected progression of long segment myelomalacia. [He] suspect[ed] the initial changes were ischemic in nature." Pet. Ex. 1 at 1308.

On November 15, 2016, Petitioner returned to Dr. Garrett (neurosurgery). Pet. Ex. 3 at 8373. His assessment was "[t]horacic myelopathy - recent regression in lower extremity strength correlates with the prolonged T2 signal within the spinal cord but cannot tell if it is new ischemia

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[28] Myelomalacia is the "morbid softening of the spinal cord." Myelomalacia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=32720 (last visited May 9, 2024).

[29] Kyphosis is "an area of the vertebral column that is dorsally convex" or "abnormally increased dorsal convexity in the curvature of the thoracic vertebral column." Kyphosis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=27290 (last visited May 3, 2024).

or myelitis." Id. at 8374. He further noted that he was "not sure why her spinal cord developed inflammatory changes and she began to lose ability[,] primarily motor strength in her legs about one and a half years after her initial decompression surgery." Id. at 8375.

On February 1, 2017, Petitioner saw Dr. Tramonte in follow-up. Pet. Ex. 1 at 1359. Dr. Tramonte noted that Petitioner had a calcified mass at T5/6 that was surgically removed, and that her post-surgery health was "complicated by an epidural abscess, empyema, meningitis, with compression of the conus medullaris[30] and cauda equina." Id. Petitioner "also developed a CSF leak needing a thoracotomy for resection of the dis[c] as well as dural repair." Id. Dr. Tramonte noted that Petitioner's prior MRI in July 2016 showed a syringomyelia that had progressed from T6/7 in December 2015 to T11 in July 2016. Id. at 1366. He noted that "[t]he findings are thought to represent expected progression of long segment myelomalacia," and he suspected that "the initial changes were ischemic in nature." Id.

On March 10, 2017, Petitioner saw neurologist Darin Okuda, M.D., at University of Texas ("UT") Southwestern Medical Center MS clinic. Pet. Ex. 7 at 7. Dr. Okuda noted that Petitioner's "initial difficulties began in February 2014 when she experienced complaints of numbness and tingling." Id. He described her surgery and hospital course, noting that she was unable to walk at the time of discharge. Id. He further noted that Petitioner received a flu vaccine in December 2015, after which she reported being non-ambulatory. Id. Dr. Okuda diagnosed Petitioner with myelopathy, noting that "etiology [was] currently unclear based on the available data." Id. at 9. He elaborated: "Based on the existing data, it is unclear if an intramedullary process was present only after exposure to a vaccination or if the described changes within the thoracic spinal cord were related to her prior neurosurgical procedures (i.e., presence of an infection followed by an aberrant immune behavior)." Id. Dr. Okuda wanted to see more of Petitioner's medical records related to her neurosurgical procedures and post-surgery treatments, and to schedule a follow-up appointment. Id.

Petitioner returned to the UT Southwestern Medical Center MS Clinic on May 16, 2017. Pet. Ex. 7 at 26-27. Impression was

> [m]yelopathy (etiology currently unclear based on available data). . . . Based on the available information from Baylor Scott and White, it appears [that] an intramedullary process was present following her neurosurgical procedures with evolution over time. However, this cannot be confirmed without the corresponding [] images.

Id. at 27. Petitioner was asked to obtain additional images for further evaluation and to sign a waiver so that the MS clinic could request the images directly. Id. While the record contains notes of email and telephone exchanges, it does not appear that Dr. Okuda saw Petitioner in follow-up. See, e.g., id. at 38, 45-47, 51-52, 66, 70, 74, 79-80.

[30] The conus medullaris or conus is "the cone-shaped lower end of the spinal cord, at the level of the upper lumbar vertebrae." Conus Medullaris, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=66882 (last visited Apr. 25, 2024).

On October 18, 2017, Petitioner saw Elliot Frohman, M.D., at UT Health Austin. Pet. Ex. 10 at 35. Dr. Frohman noted that Petitioner's symptoms in February 2014 were "reminiscent of a sensory transverse myelitis." Id. at 40. He suspected that Petitioner "did in fact have transverse myelitis, although the pathoetiologic underpinning of this process remain enigmatic." Id. Dr. Frohman recommended a further work-up for the causes of her myelitis, ordered plasma exchange treatment, and stated that Rituxan treatment would be considered. Id. at 41.

After further testing, Petitioner saw Dr. Frohman in follow-up on November 1, 2017. Pet. Ex. 10 at 29. Dr. Frohman counseled Petitioner that he believed her syndrome in February 2014 was consistent with myelitis, but "the underlying etiology remain[ed] elusive," which he emphasized was "NOT [an] uncommon circumstance in a significant proportion of myelitis patients." Id. at 31. He recommended a number of treatments, which he stated may lead to some improvement in Petitioner's condition, even though the treatment was so remote from the initial episode of myelitis in February 2014. Id. at 31-32. Diagnosis was "idiopathic transverse myelitis." Id. at 32.

On March 7, 2018, Petitioner had a follow-up visit with Dr. Frohman. Pet. Ex. 10 at 15. Petitioner had completed some treatment, but Dr. Frohman stated it was "too early to tell whether there ha[d] been a meaningful exchange." Id. at 18.

Petitioner saw Dr. Frohman again on July 9, 2018. Pet. Ex. 10 at 11. She had been attending PT twice weekly and felt she was making great progress. Id. at 14. Dr. Frohman noted that he wanted to see her back "in the next three months to review her progress." Id. Her diagnosis remained idiopathic transverse myelitis. Id.

Petitioner returned to the UT Health Austin on October 4, 2018. Pet. Ex. 10 at 4. Her diagnosis remained "idiopathic transverse myelitis by evidence to date." Id. at 8.

MRI studies of the cervical, thoracic, and lumbar spine showed "extension of expansile cord signal abnormality" throughout the entire spinal cord from the C2 level in the cervical spine extending to the conus. Pet. Ex. 12 at 23. There was "[n]o definitive abnormal enhancement appreciated," however, evaluation was limited due to motion. Id. The studies also showed "[m]ultilevel cervical spondylosis and facet arthropathy with varying degrees of mild foraminal narrowing and mild to moderate spinal stenosis." Id. Petitioner had the same findings in her thoracic spine, most notably at T10-T11 and T11-T12. Id. In the lumbar spine, she also had "spondylosis and facet arthropathy with superimposed pronounced dorsal epidural fat (epidural lipomatosis) which significantly narrows the thecal sac throughout the mid to lower lumbar spine." Id.

In a 2019 visit with Dr. Frohman to develop a plan of care, Dr. Frohman stated that "given [Petitioner's] worsening and fluctuating symptoms, we will treat her as having [neuromyelitis optica ("NMO")[31] spectrum disorder ("NMOSD")]."[32] Pet. Ex. 12 at 88.

3. Detailed Review of Physical Therapy Records: March 2015 – December 2015

PT records show that in March 2015, prior to the vaccination at issue, Petitioner was paraplegic following a spinal cord injury, with bilateral leg weakness, spasticity, and difficult walking. Pet. Ex. 1 at 604, 608. She was unable to ambulate even short distances with a rolling walker. Id. Petitioner attended over 40 outpatient PT visits from March through July 2015. See id. at 614, 954. During that time, she developed the ability to stand, pivot, perform transfers, and ambulate using a rolling walker. Id. at 955. Detailed PT notes establish that Petitioner had good days when she was able to accomplish her goals of walking, and other days when she was unable to do so due to spasticity, tone, and fatigue. Id. at 628-29. For example, on May 15, 2015, Petitioner was unable to walk from the bed to the bathroom with her walker due to increased muscle tone and fatigue. Id. at 770-71. On May 26, 2015, she had difficulty ambulating due to tone. Id. at 782. Three days later, on May 29, 2015, Petitioner had difficulty standing upright without initiating more tone. Id. at 789.

In June 2015, Petitioner had increased spasticity resulting in difficulty with transfers and ambulation. Pet. Ex. 1 at 793. She also complained of increased low back pain. Id. at 821, 830. She was able to ambulate with her rolling walker, but for shorter distances. Id. at 830, 837, 866-67. On July 6, 2015, Petitioner complained that her left lower extremity felt like it was going to buckle, and she continued to have increased spasticity that limited the distance she could use her walker. Id. at 884. On July 24, 2015, Petitioner was discharged from PT. Id. at 954. Her discharge notes indicated that Petitioner required a rolling walker and that her maximum gait in one session was 28 feet. Id. at 955. She did not meet her goal of walking 50 feet with her rolling walker. Id. She tolerated treatment but was limited due to increased spasticity in her lower extremities. Id. at 945.

After being discharged from PT at the end of July 2015, Petitioner returned to outpatient therapy on November 5, 2015. Pet. Ex. 1 at 1073. At this time, she complained of difficulty ambulating distances greater than 25 feet intervals. Id. at 1074. She also had central upper back

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[31] Neuromyelitis optica is the "combined, but not usually clinically simultaneous, demyelination of the optic nerve and the spinal cord; it is marked by diminution of vision and possibly blindness, flaccid paralysis of the extremities, and sensory and genitourinary disturbances." Neuromyelitis Optica, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition? id=92610 (last visited May 3, 2024).

[32] The undersigned asked the parties' experts to address Dr. Frohman's reference in 2019 to NMOSD. Order dated Nov. 17, 2022. Respondent filed an expert report by Dr. Javed addressing the question. Resp. Ex. C. Petitioner filed a brief addressing the Court's order. Pet. Supplemental Br. Both agreed that Petitioner did not meet the requirements for the diagnosis of NMOSD. Therefore, the undersigned does not discuss Dr. Frohman's diagnosis of NMOSD.

pain, aching, and burning, and bilateral lower extremity parasthesias. Id. When she walked, she experienced pain and her knees were described as spastic. Id. at 1076. On November 12, 2015, Petitioner had a "good day" and walked 40 to 45 feet with her rolling walker. Id. at 1088. But at the next visit, she reported a "bad day," when she was unable to walk at all. Id. at 1098. On December 2, 2015, she had significant fatigue, resulting in her knee buckling with ambulation. Id. at 1105-06. On December 3, 2015, she had difficulty walking with increased tone and lack of control of her right lower extremity. Id. at 1110. At the visit on December 10, 2015, she reported being unable to walk after her last therapy session. Id. at 1110-20.

Post-vaccination, on December 18, 2015, Petitioner had left lower extremity spasticity making it difficult to stand because her "good leg" was unreliable. Pet. Ex. 1 at 1148. On December 29, 2015, her increased spasticity made it difficult to transfer independently. Id. at 1170. She had a significant increase is extensor spasm and tone over two therapy sessions, and the therapist notified Petitioner's physician of the change in her condition. Id. at 1171.

B. Petitioner's Declaration[33]

Petitioner wrote that prior to receiving the flu vaccine at issue, she developed a wound infection from spinal surgery in July 2014. Pet. Ex. 33 at ¶ 1. She required readmission for surgery to clear out the infection and became paralyzed from the waist down. Id. Her neurosurgeon, Dr. Garrett, told her she had a 70-80% chance of walking again. Id.

After intensive therapy and rehabilitation, Petitioner regained "considerable strength" prior to the fu vaccine on December 11, 2015. Pet. Ex. 33 at ¶ 2. She was able to stand; take some steps with a walker; and pivot to sit on chairs, couches, the shower chair, the toilet, and into the car. Id. at ¶¶ 2, 7. She could also transfer from her wheelchair, pivot onto the workout table in PT, walk from one end of the parallel bars to the other, and ride a stationary bike for 10 minutes. Id. at ¶ 2. She had regained the ability to walk 50 feet with her walker. Id. She stated that for the six months prior to the flu vaccine, she did not use a Hoyer lift, or personal lift, to get in and out of bed, chairs, the shower, or the toilet. Id.

At a PCP appointment on December 11, 2015, Dr. Hart told Petitioner she needed the flu vaccine because "she was confined to a wheelchair and could not afford to get ill." Pet. Ex. 33 at ¶ 3. "Within days" of receiving the flu vaccine, Petitioner's family noticed Petitioner was losing control of her legs and "they were becoming weaker[] and would buckle causing [her] to lose [her] balance and fall." Id. She lost the ability to stand, use the walker, and pivot. Id. at ¶¶ 3, 7. She now required a Hoyer lift to get her in and out of bed, her wheelchair, and the toilet. Id. at ¶ 7. She had to use a sliding board to get in and out of the car because she could no longer stand and take the few steps needed to pivot into the car. Id. Petitioner's therapist recommended she contact her neurologist, Dr. Tramonte. Id. at ¶ 3.

Dr. Tramonte scheduled an MRI that was performed January 8, 2016. Pet. Ex. 33 at ¶ 3. Thereafter, Dr. Tramonte's office advised Petitioner to go back to see Dr. Garrett about having

[33] The exhibit is labeled as an "affidavit" however, it is not notarized and therefore the undersigned will refer to it as a declaration.

another surgery on her back. Id. at ¶ 4. Upon contacting Dr. Garrett, Petitioner was told the MRIs "showed fluid near the site of [her] previous operation." Id. Dr. Garrett told her he would not operate on her again because it would hurt more than it would help her. Id. He diagnosed her with TM and told Petitioner that the symptoms she was describing to him were "similar to Swine Flu. [She] told Dr. Garrett that [she] had received the flu shot earlier in December." Id.

Petitioner continued with PT twice per week. Pet. Ex. 33 at ¶ 5. After a few weeks, Petitioner's physical therapist noticed "very little improvement." Id. Petitioner's left leg, which had been her stronger leg before the flu vaccine, "was now weak and [she] could not stand on it. [Her] right leg, the weaker leg before the flu vaccine, would lift up and bend when [she] attempted to use it to stand." Id. at ¶¶ 5, 12. Petitioner had little feeling in her left leg besides numbness. Id. at ¶ 12. She could not stand without using a standing frame and soft locks to prevent her knees from buckling. Id. Petitioner later found out muscle spasms were the reason she could not stand. Id. at ¶¶ 5, 12. After the flu vaccine she also developed more spasms in her right leg. Id. at ¶ 13. "The spasms cause[d] [her] right foot to lift when [she] attempt[ed] to place it on the ground to stand and walk." Id. She could no longer lift her "right ankle automatically." Id. at ¶ 14. Petitioner then began outpatient PT where she used a sliding board to get on and off the table and the therapist stretched her legs to help with muscle spasms. Id. at ¶ 6. She did not notice much improvement. Id.

In addition to leg muscle spasms, Petitioner also suffered bladder spasms after the flu vaccine. Pet. Ex. 33 at ¶ 8. "While [she] required intermittent urinary catheterization every [four to six] hours, before the flu shot, catheterization every [six] hours kept me dry. After the flu shot, the bladder spasms would cause [her] to leak urine." Id. at ¶ 10. She averred she did not have bladder spasms or urinary leakage prior to the flu vaccine. Id. Petitioner's "bowels have also been affected by the flu shot." Id. at ¶ 11. "Before the flu shot, [she] was beginning to sense when a bowel movement was going to occur. After the flu shot, [she] lost that sensation." Id. Since she is now paraplegic, she cannot move or bend her legs for personal hygiene such as dressing below the waist, toileting, catharizing, or showering below the waist. Id. ¶¶ 8, 11, 15.

Because Petitioner cannot move below the waist, she has developed pressure sores. Pet. Ex. 33 at ¶ 19. The first pressure sore was on her left ankle in August 2020. Id. By 2021, Petitioner had developed pressure sores in her left buttock and right hip. Id. at ¶¶ 22-23. Petitioner required extensive treatment of her multiple pressure wounds including cleaning and dressing the wounds, use of VeraFlo Vacuum, an ED visit with IV antibiotics for infection, use of Hyperbaric oxygen chamber, and a flap surgery. Id. at ¶¶ 22-28. In addition to pressure sores, Petitioner is at higher risk of developing infections and kidneys stones because she is wheelchair bound. Id. at ¶¶ 21-22. In 2021, Petitioner had a stent inserted for kidney stones. Id. at ¶ 22.

When Petitioner started seeing Dr. Frohman at UT Austin MS Clinic, he ordered plasmapheresis for treatment of her transverse myelitis at UT Southwestern Dallas. Pet. Ex. 33 at ¶ 9. She also received Rituxan. Id. With these treatments, Petitioner averred she regained some sensation in her lower legs and could stand in a standing frame but was not able to move her legs on her own. Id. She stopped the Rituxan when she developed pressure sores. Id. Dr. Frohman also referred her for Botox injections to treat her muscle spasms, but she reported Botox did not make much of a difference in her condition. Id. As of the date of her declaration

(May 31, 2022), she was taking Baclofen and Levetiracetam for her leg spasms which decreased the number of them but did not completely prevent them from occurring. Id. She stated she did not require medications for muscle spasms prior to the flu vaccine. Id.

C. Expert Reports

1. Petitioner's Expert, Dr. Carlo Tornatore[34]

a. Background and Qualifications

Dr. Tornatore is a board-certified neurologist. Pet. Ex. 13 at 2; Pet. Ex. 40 at 2. He received his M.D. from Georgetown University School of Medicine where he subsequently completed a neurology residency at Georgetown University Hospital. Pet. Ex. 40 at 2-3. Thereafter he completed a post-doctoral fellow at the National Institutes of Neurologic Disorders and Stroke at the National Institutes of Health. Id. at 3; Pet. Ex. 13 at 2. Since then, he has been on the faculty at Georgetown. Pet. Ex. 13 at 2; Tr. 6. He is currently a Professor and Chairman of the Neurology Department at Georgetown University Medical Center where he oversees "research endeavors of 15 laboratories, consisting of 19 Principal Investigators and over 90 post-doctoral fellows, doctoral candidates, graduate students[,] and lab technicians. These labs focus on various aspects of the neuroscience, including neuroplasticity, neuroimaging, cognitive neuroscience[,] and language disorders." Pet. Ex. 13 at 1. He is also the Neurologist-in-Chief and Chairman of Medstar Georgetown University Hospital. Id.; Tr. 7. Additionally, Dr. Tornatore is the Director of the MS Center at Georgetown where sees and treats patients with MS and other neuroimmune system disorders including transverse myelitis. Pet. Ex. 13 at 2; Tr. 7-8. Dr. Tornatore testified he has about 400 patients with transverse myelitis that exclude MS. Tr. 8. Dr. Tornatore has authored or co-authored numerous publications. Pet. Ex. 40 at 8-19.

b. Opinion

i. Loving Factor One: What Was Petitioner's Condition Prior to Administration of the Vaccine?

Dr. Tornatore opined that Petitioner had a "very complicated course." Pet. Ex. 13 at 26. As a brief overview, in March 2014, Petitioner developed spinal cord-related neurologic symptoms caused by a mass compressing the T5/T6 level of the spinal cord. Id. She had significant stenosis in her thoracic cord due to degenerative disc disease. Tr. 10. The stenosis caused edema (swelling) and inflammation of the spinal cord seen on MRI. Pet. Ex. 13 at 26. She had decompressive surgery June 27, 2014. Id. Post-operatively, Petitioner developed a "very profound post-operative infection around her spinal cord" along with a thoracic abscess that required a second operative procedure on July 10, 2014. Tr. 10. As a result of the infection, Dr. Tornatore opined that about six weeks after her second surgery, Petitioner developed longitudinally extensive transverse myelitis, shown on the MRI August 26, 2014. Pet. Ex. 13 at 26. Two months later, on October 21, 2014, an MRI was done and showed improvement. Id.

[34] Dr. Tornatore submitted one expert report and testified at the hearing. Pet. Ex. 13; Tr. 3, 150.

After providing an overview, Dr. Tornatore reviewed Petitioner's clinical course in more detail and provided his opinions of significant MRI findings. See Pet. Ex. 13 at 2-25; Tr. 12-18. Petitioner's MRI on April 29, 2014 showed a partially calcified paracentral ventral epidural mass (calcified extrusion or meningioma) at T5 and T6. Tr. 12. Petitioner had surgery on June 27, 2014. Tr. 14-15. Post-operatively, she had a very complex clinical course. Id. She had a pleural effusion (fluid on the lungs), was hospitalized in ICU, and was discharged from the hospital on July 6, 2014, only to return on July 9, with significant weakness of the right lower extremity and bladder incontinence. Id. Her MRI on July 9, 2014, showed an extensive epidural abscess from T4 to T11 (above the nipples to below the belly button level). Tr. 16. Additionally, there was a "large loculated fluid collection in the thoracic cavity" described as an empyema or "collection of pus in the chest wall." Tr. 16-17. Thus, the MRI done July 9, 2014, showed pus in and around the spine and thoracic area. Id. Petitioner also had a "recurrent [] paracentral disc herniation [] result[ing] in moderate to severe spinal cord stenosis." Tr. 17. She also had an abscess from the top of her right lung at the level of her collar bone down to the level of T11/T12, below her belly button level, described by Dr. Tornatore as a "huge infection in the chest." Tr. 18.

Relative to the spinal cord, the July 2014 MRI showed focal inflammation of the spinal column bones and epidural space at T2 to T3. Tr. 18. Next to the dura, or covering of the spinal cord, was a collection of pus indicating there was a bacterial infection from T4, above the nipple line, and downward to below the level of the belly button. Tr. 18-19. The covering of the spinal cord, called the dura, showed enhancement on MRI "all up and down the spinal cord." Tr. 19. Dr. Tornatore explained that during the surgery, the dura was nicked, causing leakage of the spinal fluid and creating a pathway for bacteria to get to the spinal cord. Tr. 20. Analysis of the spinal fluid showed "[a]bsolutely astounding" results. Tr. 21. Petitioner's white blood cell count was elevated to 56,000 indicating "profound inflammation in the spinal fluid." Id. According to Dr. Tornatore, this indicated that Petitioner had "inflammatory myelitis, no question." Tr. 22.

The next MRI, done August 26, 2014, showed worsening with "interval development of central cord signal and expansion from C7 to T11," and it was very evident that Petitioner had inflammation of the spinal cord. Tr. 23 (quoting Pet. Ex. 3 at 7812). This inflammation was probably a result of venous congestion, cord ischemia (due to lack of blood flow), or infection. Tr. 24-25. Although Petitioner was still on antibiotics, it is very difficult to treat infection in the soft tissues. Tr. 26. Ongoing infections take months and months to treat, and during this time, it is not surprising for a patient to have "ups and downs" during their clinical course. Tr. 24.

By September 9, 2014, the infectious disease specialist noted that Petitioner had "disseminated MRSA." Pet. Ex. 3 at 7865. MRI done October 20, 2014 showed a destructive process at T5 and T6 with patchy bone marrow edema, and significant inflammatory changes in the spinal cord, and Petitioner's spine at this level was in a bent-over position. Tr. 30. At this point, Petitioner had a "really chronic issue in her spinal cord." Tr. 32. Dr. Tornatore opined that Petitioner's condition put her at risk for subsequent inflammatory events. Id. Petitioner remained on antibiotics. Id. From November 2014 to April 2015, Petitioner made slow improvement. Tr. 33-37. By April 2015, Petitioner still had significant weakness in her lower extremities, but was able to walk 30 feet with a walker, and she was beginning to feel her feet. She had foot drop in her right lower extremity. Tr. 36.

Moving forward to October 7, 2015, Dr. Tornatore opined that Petitioner could walk with a walker, that she was beginning to feel her legs, and she was still on antibiotics. Tr. 38-39. However, Dr. Tornatore opined that there was something continuing to happen in her spinal cord because Petitioner was not able to walk normally. Id. The records show that Petitioner had a "good day" on November 12, 2015, evidenced by her ability to walk 40 to 45 feet with her walker. Tr. 40 (quoting Pet. Ex. 1 at 1888). But she had difficulty placing her right foot with sitting, standing, or walking. Id. She also complained that day of stiffness in her legs, although she had less tightness or spasticity. Id.

On December 3, 2015, Petitioner had more stiffness and increased muscle tone in both legs, right greater than left, and fatigue. Tr. 42 (citing Pet. Ex. 1 at 1110). Dr. Tornatore opined that Petitioner was experiencing "more spasticity at this point." Tr. 43. Additionally, the note on December 3 raised the question of whether something else was going on in Petitioner's spinal cord or suggested that whatever was happening was "getting a little more prominent." Id. Petitioner remained on the antibiotic doxycycline. Id.

At her PT visit on December 11, 2015, Petitioner reported that she was "unable to walk" the day after her last session. Tr. 44 (quoting Pet. Ex. 1 at 1119). Dr. Tornatore said "there's a little something here" and he again questioned whether "something else [was] going on." Tr. 45. At her appointment with Dr. Hart on December 11, 2015, the records stated that in the "last several weeks," Petitioner's legs were getting tight and she had stiffening at night. Tr. 46 (quoting Pet. Ex. 1 at 1126). She was also having noticeable spasms. Id. Dr. Hart referred Petitioner to her to neurologist. Id. Dr. Tornatore opined there was "some change" in Petitioner's tone in her lower extremities. Tr. 47. Petitioner received her flu vaccination at this visit. Id.

ii. Loving Factor Two: What Is Petitioner's Current Condition (or Her Condition Following the Vaccination, If Also Pertinent)?

According to Dr. Tornatore, shortly after receiving a flu vaccination on December 11, 2014, Petitioner had a significant aggravation of her transverse myelitis with worsening neurologic symptoms and recurrent edema and inflammation of the thoracic spinal cord, demonstrated by her January 8, 2016 MRI. Pet. Ex. 13 at 26. Dr. Tornatore opined that Petitioner's diagnosis was transverse myelitis, initially caused by infection, and subsequently aggravated by the flu vaccination. Id.; Tr. 10.

On December 18, 2015, Petitioner reported that her spasticity was worse and she was unable to stand. Tr. 47-48 (citing Pet. Ex. 1 at 1148). This was also noted at her PT visit on December 30, 2015, when Petitioner's increased spasticity made it difficult for her to bear weight. Tr. 48 (citing Pet. Ex. 1 at 1170). Petitioner saw her neurologist, Dr. Tramonte, on December 30, and he noted that she had worsening weakness since mid-December. Tr. 50 (citing Pet. Ex. 1 at 1181). Petitioner did not have changes to her bladder or bowel, and she had no increase in her chronic pain. Tr. 51 (citing Pet. Ex. 1 at 1181).

Petitioner had an MRI on January 8, 2016, 15 months after her last one. Tr. 52. Dr. Tornatore opined that it showed edema of the spinal cord from T1 to T10 with an intramedullary syringohydromyelia at T6/T7 (an abnormal collection of fluid in the central canal of the spinal cord). Tr. 53 (citing Pet. Ex. 1 at 1211). According to Dr. Tornatore, this finding indicated persistent inflammation. Tr. 54. He agreed that Petitioner initially had inflammation of the spinal cord due to compression, and then there was more ischemia, lack of blood flow, and death of tissue, "related back to . . . inflammation and irritation at that site." Tr. 56. However, he also agreed that the MRI findings could be due to cord ischemia and altered CSF flow dynamics. Tr. 56-57.[35]

Petitioner saw her neurosurgeon, Dr. Garrett, on January 13, 2016. Pet. Ex. 3 at 8249. Dr. Tornatore interpreted Dr. Garrett's notes from that visit to mean that Dr. Garrett agreed that the MRI findings were "due to spinal cord compression that antedated the abscess and edema . . . but [Dr. Garrett] acknowledge[d] that the MRI could be [] an autoimmune phenomenon, and he[] invoke[ed] the flu vaccine." Tr. 58 (citing Pet. Ex. 3 at 8249). Although Dr. Garrett noted that Petitioner could have an autoimmune condition, Dr. Tornatore agreed that Dr. Garrett did not treat Petitioner for transverse myelitis. Tr. 58.

On January 20, 2016, Petitioner returned to see her neurologist, Dr. Tramonte. Pet. Ex. 1 at 1250. Dr. Tornatore interpreted the note from that visit to mean that Dr. Tramonte thought the changes in the MRI were "ischemic in nature," caused by a "lack of blood flow in that area of the spinal cord." Tr. 62 (citing Pet. Ex. 1 at 1254). Although Dr. Tornatore disagreed with Dr. Tramonte's opinions in this regard, he agreed that there could be a "slow[ly] diminish[ing] decrease in blood flow to the spinal cord" caused by swelling. Tr. 62-63. And he agreed that there was "probably some lack of blood flow contributing" to Petitioner's condition. Tr. 63.

The next MRI was done July 1, 2016, and again it was markedly abnormal. Tr. 64 (citing Pet. Ex. 3 at 8320). Dr. Tornatore observed that Dr. Tramonte now interpreted the results as transverse myelitis. Tr. 65. However, Dr. Tornatore agreed that Petitioner was not treated for transverse myelitis. Id.

Dr. Tornatore was questioned about Petitioner's visits to Dr. Frohman, a neuroimmunologist. See Tr. 72. In a note dated November 11, 2017, Dr. Frohman attributed Petitioner's initial syndrome, beginning in 2014, to be consistent with inflammation and myelitis of the spinal cord, but he concluded that the "underlying etiology remain[ed] elusive." Tr. 75 (quoting Pet. Ex. 10 at 31). At Petitioner's next visit with Dr. Frohman on October 4, 2018, Dr. Frohman noted that Petitioner's chief complaint was "idiopathic transverse myelitis." Tr. 76 (quoting Pet. Ex. 12 at 92).

At the hearing, after reviewing Petitioner's 2018 MRI of the spine, Dr. Tornatore supplemented his opinions. He opined that Petitioner's 2018 MRI, as compared to the prior MRI done in January 2016, showed Petitioner had an "inflammatory process" extending from C1 (with chronic compression at T6 and across the T5/T6 disc space and kyphosis at T5/T6) with an "increased extensive abnormal expansile cord signal seen throughout the thoracic

[35] For a more complete discussion of the January 8, 2016 MRI, see Tr. 52-57.

cord, extending . . . all the way to the bottom of the spinal cord." Tr. 283-284 (citing Pet. Ex. 12 at 140). Dr. Tornatore characterized Petitioner's 2018 MRI as the "worst" of all of Petitioner's MRI studies, showing an "area of increased signal going from C1 all the way down to the very bottom. It's the whole spinal cord now." Tr. 285.

After review of all of Petitioner's MRI studies, including the one done in 2018, Dr. Tornatore opined that in 2014, Petitioner had transverse myelitis due to active inflammation. Tr. 288-89. He then addressed the question of why Petitioner was worse in 2018, even though at that time she was being treated with immunosuppressants. Tr. 285-87. Dr. Tornatore agreed that given the findings on the 2018, it was time to consider other etiologies for Petitioner's condition, because her "inflammatory myelitis" had been ongoing for four years, along with scarring (glial formation). Tr. 291-92. He also agreed that based on the 2018 MRI study, that Petitioner's illness was not monophasic, but progressive. Tr. 292-93.

Dr. Tornatore opined that the onset of Petitioner's myelitis was July 2014. Tr. 293. He opined that one and one-half year later, she had the vaccination at issue, and according to Dr. Tornatore, Petitioner relapsed and never recovered. Tr. 293-94. Dr. Tornatore agreed there was "canal stenosis" and that the stenosis was "contributing, you know, to some of this." Tr. 296. He also agreed that the stenosis was present "at the very beginning, before she had the surgery." Id.

Ultimately, Dr. Tornatore agreed with Respondent's expert Dr. Javed, stating "we can't distinguish what is [caused by] canal stenosis versus what is inflammatory disease of the cord." Tr. 296-97. He further agreed that the vaccine did not cause the "subsequent progression that [Petitioner] experienced." Tr. 298. He opined that the vaccine "had one moment in time where it made things worse, causing exacerbation, and then the natural history of her myelitis [] just continued on from that point . . . forward." Id.

iii. Loving Factor Three: Does Petitioner's Current Condition (or Condition After Vaccination) Constitute a "Significant Aggravation" of Her Condition Prior to Vaccination?

As stated above, Dr. Tornatore opined that the vaccine "had one moment in time where it made things worse, causing exacerbation, and then the natural history of [Petitioner's] myelitis [] just continued on from that point . . . forward." Tr. 298.

When asked to quantify Petitioner's alleged vaccine-related injury, given her complex clinical history, his acknowledgement that canal stenosis was a contributing factor present before surgery, and that it contributed to the subsequent progression of disease, Dr. Tornatore

used the Kurtzke extended disability status ("EDS") score[36] to compare Petitioner's condition before and after vaccination. Tr. 299. Before her vaccination on December 11, 2015, Petitioner, on her best day, could walk with a walker, and thus, Dr. Tornatore assigned her an EDS score of 6.5. Tr. 300. After vaccination, she was wheelchair-bound, which placed her at a score of 7.0 or 7.5. Id. He concluded that Petitioner's vaccination worsened her disability by roughly 20%. Id.

iv. Loving Factor Four/Althen Prong One: Medical Theory of Causation

Dr. Tornatore explained that acute transverse myelitis "has been reported following certain vaccinations, including, specifically, the seasonal [flu] vaccination." Pet. Ex. 13 at 27. He referenced the National Institute of Neurological Disorders and Stroke ("NINDS") fact sheet on transverse myelitis which stated that the condition can be a "post-vaccinal" event. Tr. 78 (citing Pet. Ex. 38 at 2).[37] He also cited a paper about acute disseminated encephalomyelitis ("ADEM") by Huynh et al.,[38] which stated that ADEM ("of which transverse myelitis is part of") has been associated with vaccines, including the flu vaccine. Tr. 79-80 (citing Pet. Ex. 20 at 1).

He cited mechanisms proposed by Huynh et al., including molecular mimicry[39] and the "inflammatory cascade process." Tr. 81 (citing Pet. Ex. 20 at 6). Dr. Tornatore described a "first hit" that occurs with an antecedent infection and molecular mimicry, followed by a "second infection with an unrelated infection result[ing] in sufficient reactivation of the primed autoreactive T-cells to eventually end in demyelination of the central nervous system," constituting the "second hit." Id. With molecular mimicry, Dr. Tornatore explained that there is exposure to an antigen and "the immune system recognizes the antigen and processes it, but then because the antigen has homology to self-protein, the immune response is directed not only to

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[36] The Kurtzke EDS scale "was developed by Dr. John Kurtzke in the 1950s to measure the disability status of people with MS." Kurtzke Expanded Disability Status Scale, Multiple Sclerosis Ctrs. of Excellence, U.S. Dep't of Veterans Affs., https://www.va.gov/MS/Professionals/diagnosis/Kurtzke_Expanded_Disability_Status_Scale.asp (last updated Mar. 18, 2021). Dr. Tornatore testified that the Kurtzke EDS scale is used for other inflammatory disorders and is relevant here. Tr. 299.

[37] Transverse Myelitis Fact Sheet, NINDS, https://www.ninds.nih.gov/health-information/disorders/transverse-myelitis (last reviewed July 25, 2022).

[38] William Huynh et al., Post-Vaccination Encephalomyelitis: Literature Review and Illustrative Case, 15 J. Clinical Neuroscience 1315 (2008).

[39] For further discussion of molecular mimicry and transverse myelitis, see Pet. Ex. 13 at 30; Pet. Ex. 36 (Kai W. Wucherpfenning & Jack L. Strominger, Molecular Mimicry in T Cell-Mediated Autoimmunity: Viral Peptides Activate Human T Cell Clones Specific for Myelin Basic Protein, 80 Cell 695 (1995)) (discussing molecular mimicry, viral pathogens, and reactivity to myelin basic protein).

the protein that was presented, the antigen, but also to the host protein which was already there." Tr. 83. The vaccine is "misdirected at those antigens in the host . . . in the spinal cord." Id.

The paper by Huynh et al. does not relate to transverse myelitis, but instead is about post-vaccination ADEM. Pet. Ex. 20 at 1. The authors state that ADEM is "an inflammatory demyelinating disease of the central nervous system that is usually considered a monophasic disease." Id. ADEM has been associated with several vaccines, including the flu vaccine. Id. The article discusses the presumptive mechanisms of ADEM, including molecular mimicry and the immune-inflammatory model. Id. at 6. The immune-inflammatory model "combines the concept of molecular mimicry with the inflammatory cascade process." Id. "A 'first hit' is experienced after an antecedent infection with a virus that expresses determinants allowing molecular mimicry. This need not be clinically eventful or significant." Id. "A second infection with an unrelated virus results in sufficient reactivation of the primed autoreactive T cells to eventuate in demyelination of the [central nervous system]. This constitutes the 'second hit.'" Id. The authors also noted that some patients "have a certain underlying genetic predisposition [and] may be more prone to developing ADEM post-vaccination." Id.

Central to Dr. Tornatore's opinions as to causation is the concept of "fertile field." Tr. 83; Pet. Ex. 13 at 30. He explained that this mechanism presumes there is "already tissue injury." Tr. 83. According to Dr. Tornatore, in the fertile field model, "activation of an immune response by an immunogen with sequence homology to self-proteins may prime autoreactive T-cells in the host, but might lack the momentum to initiate an adverse autoimmune reaction independently." Pet. Ex. 13 at 30. "However, a later immune stimulation or coincidental immune stimulation event by a vaccine or other immunogen, even one without cross-reactive antigens or sequence similarity, could initiate an autoimmune reaction in a susceptible host that could lead to inflammatory demyelination." Id. He explained that the combination of molecular mimicry, fertile field, and genetic predisposition all play a role. Tr. 83.

In support of his opinions about "fertile field," Petitioner filed a paper by Fujinami et al.[40] Pet. Ex. 13 at 30 (citing Pet. Ex. 25). They wrote that "[i]nfection of the host and the interactions between the immune response and virus set the stage for a 'fertile field' where the host and/or target organ is 'primed' for subsequent immunopathology." Pet. Ex. 25 at 1. Three potential mechanisms are discussed: molecular mimicry, bystander activation, and persistent viral infection. Id. The authors discuss how autoimmunity could develop in the context of a viral infection. Id. at 2. "Persistent viral infections can lead to immune-mediated injury due to the constant presence of viral antigen driving the immune response." Id. An example given of a persistent viral antigen was Theiler's encephalomyelitis in mice that causes a central nervous system infection. Id. After the mice recover from an acute infection in the neurons, they

[40] Robert S. Fujinami et al., Molecular Mimicry, Bystander Activation, or Viral Persistence: Infections and Autoimmune Disease, 19 Clinical Microbiology Revs. 80 (2006).

develop persistent infection in glial cells[41] and the infection can be detected throughout the lifetime of the animal. Id. The authors suggested that while a person may be "repeatedly exposed to a potential immunogen without any untoward consequences . . . , if the person had a viral infection at the time of exposure, infection would alter the immunological environment in which the antigen was encountered, leading to a profound immune response." Id. at 4.

Fujinami et al. identified MS, myocarditis, and diabetes as three illnesses where the fertile field model is being studied in animal models. Pet. Ex. 25 at 5. One question is whether a viral infection "having mimicry with self [central nervous system] proteins could prime for autoimmune disease later in life." Id. The first step is a "priming infection (setting up the fertile field) [that] increases the number of autoreactive T cells but not sufficiently to cause disease." Id. at 6. The next step is that a "critical number or mass of autoreactive T cells must be generated in order for [the illness] to develop." Id. The authors suggested bystander activation and molecular mimicry as possible ways to explain how exacerbations or secondary events occur. Id. at 5-6. The authors concluded that "[v]iruses have been shown to be one of the environmental factors that are capable of precipitating autoimmune disease by a variety of possible mechanisms discussed" and noted the challenges in obtaining evidence to develop application of the fertile field model, including, for example, the need to obtain the "total infectious history of each individual" in order to assess whether they contribute to autoimmune diseases. Id. at 12.

In addition to molecular mimicry, the immune-inflammatory model, and the fertile field model, Dr. Tornatore opined that another mechanism, bystander activation, can contribute to the development of transverse myelitis in the context of the facts and circumstances relevant here. Tr. 85-86; Pet. Ex. 13 at 29. He defined bystander activation as a "nonspecific antiviral response to inflammation" resulting in T-cells releasing cytokines and chemokines that cause tissue damage. Tr. 86. In support of bystander activation, Dr. Tornatore cited an article by Smatti et al.[42] about viruses and autoimmunity. Tr. 84 (citing Pet. Ex. 37 at 2 fig.1). The authors used "bystander activation" to describe a mechanism "whereby a non-specific and over-reactive antiviral immune response creates a localized pro-inflammatory environment along with the release of self-antigens from the damaged tissue. These self-antigens are subsequently taken up and presented by antigen presenting cells [] to stimulate the previously non-responsive, yet autoreactive T cells in the vicinity triggering autoimmunity." Pet. Ex. 37 at 2. The authors discussed flu viruses and noted that viral replication of these viruses have been reported in the respiratory tract, pancreas, and other internal organs. Id. at 4. One of the studies referenced by the authors reported an association between the flu virus and ADEM. Id. at 8 tbl.1. However, vaccines were not discussed in Smatti et al. and neither was transverse myelitis.

[41] Glial cells are "the cells of the supportive tissue of the central nervous system." Neuroglia Cells, Dorland's Online Med. Dictionary, https://www.dorlandsonline.com/dorland/definition?id=64287 (last visited May 9, 2024).

[42] Maria K. Smatti et al., Viruses and Autoimmunity: A Review on the Potential Interaction and Molecular Mechanisms, 11 Viruses 762 (2019).

Lastly, Dr. Tornatore discussed epitope spreading, "whereby invading antigens accelerate an ongoing autoimmune process by local activation of antigen presenting cells and over processing of antigens." Pet. Ex. 13 at 29; see also Tr. 86. Dr. Tornatore explained that in the backdrop of a viral infection, there is antigen presentation. Tr. 86. Due to disruption of tissue, "there are more epitopes . . . released from the tissue." Id. He likened this process to the fertile field, "where you have not only epitopes, but you also have a lot of inflammation." Id. According to Dr. Tornatore, all these mechanisms "feed on one another . . . especially given [the context of] a previous underling inflammatory event in the spinal cord and then a subsequent one with the [flu] vaccination." Tr. 86-87. The Smatti et al. paper also discussed epitope spreading, describing it as a mechanism "in which a viral infection triggers the release of more self-antigens and the de novo activation of autoreactive cells, which consequently spread to target additional self-epitopes." Pet. Ex. 37 at 2.

Specific to molecular mimicry, Dr. Tornatore described hemagglutinin, "an important viral protein of [flu] A" and the "current [flu] A virus [in] vaccines." Tr. 87. He explained that it is "an antigen present on [flu] A" in the flu vaccine. Id. He cited a paper by Li et al.[43] which showed that an antigen in the flu vaccine reacts against brain tissue and parts of the nervous system. Tr. 88 (citing Pet. Ex. 35). Dr. Tornatore stated that Li et al. identified "two broadly cross-reactive epitopes on hemagglutinin A." Tr. 88. The "antibodies against these epitopes react with hemoglobin and numerous types of important normal tissues and organs." Id. One of the antibodies discussed in Li et al. "reacted against neuronal cells." Tr. 89 (citing Pet. Ex. 35 at 5 tbl.II). Immunoreactivity testing showed that the antibodies "attached to [] brain tissue . . . [and] parts of the nervous system." Id. Dr. Tornatore concluded that Li et al. supported the mechanism of molecular mimicry because they identified "the antigen, [] the antibody, the immune system . . . and the fact that it will attach to [] part of the nervous system, causing injury." Id.

Dr. Tornatore also cited Agmon-Levin et al. which discussed transverse myelitis and vaccines. Pet. Ex. 18. The authors described the mechanisms of molecular mimicry and epitope spreading, as well at the mechanisms associated with post-infectious transverse myelitis. Id. at 4. Regarding vaccines, they also discussed the role of adjuvants. Id. at 5. They noted that "[t]he association of [transverse myelitis] with so many different vaccines alludes to the idea that a common denominator might be responsible, such as a common adjuvant."[44] Id. In Agmon-Levin et al., the authors conducted a literature search identifying 37 cases[45] of transverse myelitis following vaccinations. Id. at 2, 3 tbl.1. Two of the cases occurred after the flu vaccine. Id. The authors concluded, "[t]he rarity of post-[flu] vaccination neurological complications reported in recent years makes it impossible to establish a definite causal relation." Id. at 4.

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[43] Yuan Li et al., Identification and Characterization of Epitopes from Influenza A Virus Hemagglutinin that Induce Broadly Cross-Reactive Antibodies, 41 Int'l J. Molecular Med. 1673 (2018).

[44] There is no evidence to suggest that Petitioner's flu vaccine contained an adjuvant.

[45] Forty-three cases were found but six were excluded due to lack of clinical or demographic information. Pet. Ex. 18 at 2.

In addition to citing the papers discussed above, Dr. Tornatore referenced case reports of transverse myelitis after the H1N1 vaccination. Pet. Ex. 13 at 28. The first case report (published in 2011) described a patient with a cervical lesion at C6 and C7 that had "mild expansion of the cord and enhancement." Id. (quoting Pet. Ex. 17 at 1).[46] The patient was previously healthy. Pet. Ex. 17 at 1. About one month after vaccination, he developed fever, followed two days later by decreased sensation on his abdomen and urinary urgency. Id. Neurology examination showed brisk reflexes and sensory impairment of the right leg. Id. CSF showed mild pleocytosis and was positive for oligoclonal bands. Id. Cervical MRI revealed a lesion at the C6/C7 level with gadolinium enhancement, consistent with transverse myelitis. Id. Steroid treatment for five days resulted in almost complete recovery. Id. The authors specifically noted that there was "no spinal cord compression, other neurological disease, . . . or progression over [four] weeks." Id. at 2. Regarding vaccine causation, the authors suggested that the adjuvant in the vaccine[47] may have played a role in an immune-mediated process. Id.

The second case was reported by Akkad et al. (published in 2010) and described a patient with longitudinally extensive transverse myelitis in association with receiving an attenuated virus nasal[48] H1N1 vaccine.[49] Pet. Ex. 16 at 1. Akkad et al. described a patient who developed symptoms of transverse myelitis four days after vaccination. Id. MRI showed a non-enhancing lesion extending from the cervical spine thought the entire thoracic spinal cord (longitudinally extensive transverse myelitis). Id. No oligoclonal bands were present. Id. at 2. After steroids and plasmapheresis, the patient had significant improvement. Id.

Nakamura et al.[50] also reported a case of a patient who developed transverse myelitis (C6 to T3) seven days after the flu vaccine. Pet. Ex. 19 at 1-2. Treatment with gamma globulin afforded a partial recovery. Id. at 2-3. Bakshi and Mazziotta also described a case of acute transverse myelitis with onset four weeks after receipt of a flu vaccination. Pet. Ex. 39 at 1. MRI showed spinal cord enlargement from the C3 level to the upper thoracic levels. Id. at 1. No enhancement was seen. Id. at 3. No antecedent infectious or alternative causes were noted. Id.

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[46] Isabelle Korn-Lubetzki et al., H1N1 Vaccine-Related Acute Transverse Myelitis, 13 Isr. Med. Ass'n J. 249 (2011).

[47] Again, there is no evidence to suggest that Petitioner's flu vaccine contained an adjuvant.

[48] Petitioner here did not receive a nasal vaccine.

[49] Ambrose et al. noted that the patient reported in the Akkad et al. paper received treatment for mycoplasma pneumonia 20 days before the onset of her transverse myelitis, and therefore, it was also a potential cause. Pet. Ex. 15 at 1 (Christopher S. Ambrose, A Case Report of Transverse Myelitis Following Influenza Vaccination, 68 Archives Neurology 1085 (2011)).

[50] Naoko Nakamura et al., Neurologic Complications Associated with Influenza Vaccination: Two Adult Cases, 42 Internal Med. 191 (2003).

Other articles filed by Petitioner have been reviewed, but they are not discussed because the substance was covered by other articles, or they are less relevant. See, e.g., Pet. Ex. 21 (case report describing encephalitis, not transverse myelitis);[51] Pet. Ex. 22 (an older article discussing vaccine-induced autoimmunity);[52] Pet. Ex. 23 (describing Guillain-Barré Syndrome ("GBS"), a peripheral neuropathy, not transverse myelitis, a central nervous system disorder).[53]

v. Loving Factor Five/Althen Prong Two: Logical Sequence of Cause and Effect

Dr. Tornatore opined that Petitioner initially developed transverse myelitis in July 2014, when she was readmitted to the hospital after surgery. Tr. 128. Her MRI on July 9, 2014 was, in his opinion, consistent with transverse myelitis. Id. The immune response to infection (caused by the surgery and its complications) led to the transverse myelitis that occurred in July 2014. Id. In summary, he opined that Petitioner's initial trigger of transverse myelitis was post-operative infection. Tr. 127.

In December 2015, after the flu vaccination, Dr. Tornatore opined that Petitioner's transverse myelitis worsened. Tr. 129. He attributed the worsening to the flu vaccination via the mechanisms described above. Id. However, when asked to do so, he advanced two non-vaccine related possibilities to explain Petitioner's worsening in December 2015. The first was that he acknowledged that Petitioner had "good days and bad days." Id. Thus, the worsening in her clinical condition could reflect the ups and downs that occur with the disease. See Tr. 24.

The second possibility was that something re-triggered Petitioner's transverse myelitis even before she received her flu vaccination. Tr. 129. Dr. Tornatore stated there was no explanation as to what the trigger was since Petitioner did not have a fever or new infection. Id. He suggested that Petitioner may have had a deep seeded infection and so although she was on an antibiotic, it was possible that the treatment was "really not adequate," and "maybe that's what triggered the December changes prior to the vaccine." Tr. 131. If Petitioner still had a lingering infection, he suggested that it was "most likely the discitis, the bone infection, because that's the hardest to get rid of. If there was any bacteria that had lingered . . . the immune system would . . . fight that off . . . lead[ing] to [] inflammation all over again in the spinal cord." Tr. 132. He further explained that "any infection will stimulate the immune system . . . and then it's going to overshoot because it's already had this happen one time and then it's going to cause the myelitis to develop as a spillover. It's a fertile field effect." Tr. 132-33. He explained that it is the "[s]ame concept as if the antigens came in from the vaccine." Id.

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[51] Isabelle Van Ussel et al., Encephalitis Related to a H1N1 Vaccination: Case Report and Review of the Literature, 124 Clinical Neurology & Neurosurgery 8 (2014).

[52] Arnon Dov Cohen & Yehuda Shoenfeld, Vaccine-Related Autoimmunity, 9 J. Autoimmunity 699 (1996).

[53] Reinhard Kiefer et al., Immune-Mediated Neuropathies, in 88 Advances in Neurology, Neuromuscular Disorders 11 (Rahman Pourmand & Yadollah Harati eds., 2001).

Dr. Tornatore generally agreed with the interpretation of Petitioner's MRI studies provided by the treating radiologists and her physicians. Tr. 133-35. He noted that she had inflammation, congestion, and edema. Tr. 134. The MRIs showed that Petitioner had destruction of the discs and bone itself, which caused her to be "tilted," which could cause changes in her ambulation. Tr. 135. While he agreed that destruction of bone can lead to instability and inflammation, he believed that in Petitioner's case, it was the effect of the vaccine because of "the tempo of her change in December." Tr. 138. Regarding Petitioner's spasms, spasticity, and muscle tightness, Dr. Tornatore agreed that these problems can be caused by "anything that hits the spinal cord." Tr. 136. And on cross-examination, Dr. Tornatore acknowledged that Petitioner had compressive myelopathy and that from the onset she had severe stenosis that was never resolved. Tr. 115.

vi. Loving Factor Six/Althen Prong Three: Proximate Temporal Relationship

Dr. Tornatore opined that the initial onset of Petitioner's transverse myelitis was in the post-operative period when she was readmitted to the hospital on July 9, 2014. Tr. 128. He based this opinion on the MRI done July 9, 2014, which he believed is consistent with transverse myelitis. Id.

Generally, Dr. Tornatore testified that the time frame for development of transverse myelitis after vaccination is one to 63 days. Tr. 90. He opined that the mechanisms of molecular mimicry and fertile field "happen pretty quickly." Id. He testified that Petitioner's significant aggravation of transverse myelitis occurred within a week of vaccination, although "there was even a hint that there was something maybe that happened just before the vaccination." Tr. 91. However, he believed that the vaccine "really triggered everything." Id.

2. Respondent's Expert, Dr. Adil Javed[54]

a. Background and Qualifications

Dr. Javed is a board-certified neurologist. Resp. Ex. A at 1; Resp. Ex. B at 3. He received his M.D. from Southern Illinois University School of Medicine. Resp. Ex. B at 1. Thereafter he completed a neurology residency at Yale University School of Medicine and a neuroimmunology fellowship at the University of Chicago. Id. He is currently an Associate Professor in the Department of Neurology at the University of Chicago School of Medicine. Id.; Resp. Ex. A at 1. He also leads a "multi-disciplinary autoimmune clinic" where he diagnoses patients with transverse myelitis, MS, NMO, and other autoimmune diseases. Resp. Ex. A at 1; Tr. 153, 155-57. Dr. Javed has authored or co-authored multiple publications. Resp. Ex. B at 4-6. His research focuses on "evaluating MS lesions and brain/spine atrophy using advanced MRI techniques." Resp. Ex. A at 1; Tr. 153-54, 156.

b. Opinion

[54] Dr. Javed submitted two expert reports and testified at the hearing. Resp. Exs. A, C; Tr. 150.

i. Loving Factor One: What Was Petitioner's Condition Prior to Administration of the Vaccine?

Prior to vaccination, Dr. Javed's opinion was that Petitioner had compressive myelopathy. Resp. Ex. A at 3; Tr. 159-60. In reviewing Petitioner's medical records, he cautioned that the terminology was confusing. Tr. 210. He agreed that Petitioner had inflammation or myelitis, but it was caused by compressive myelopathy. Id. He explained that "compressive myelitis can be confused . . . with either [an] infectious or autoimmune . . . myelitis." Tr. 210. To distinguish the cause, Dr. Javed explained that a spinal tap to analyze CSF for the presence of inflammation is required. Tr. 209-10. When Petitioner's records stated "transverse myelitis" as a chief complaint, or otherwise referenced transverse myelitis, he stated that conclusions as to the cause could not be reached absent a proper "vet[ting]" or workup. Tr. 210-12.

Dr. Javed provided the following opinions relevant to Petitioner's condition before vaccination. Petitioner's relevant clinical course began prior to June 2014, when her pre-operative MRIs showed that she had a moderate central canal stenosis at the T5 level and mild central canal stenosis at T6. Resp. Ex. A at 3; Tr. 159-60. Dr. Javed explained that Petitioner had a disc or calcification pressing into the ventral (back) side of the spinal cord, compressing it by 50%. Tr. 161. This resulted in a 6.5 mm "spinal cord area being pressed significantly to almost [] half that." Id. He added the ventral part of the spinal cord supplies power and strength "to the legs, control of bladder, [and] sensation to the legs." Id.

Surgery to remove the disc or calcification pressing the spinal cord was not successful. Tr. 160-62. Instead of being relieved by surgery, Petitioner's compressive myelopathy was "worsened by surgery," and then worsened even more by her post-operative infections. Id. Dr. Javed opined that the appearance of compressive myelopathy looked like longitudinally extensive transverse myelitis, but the cause was compression. Tr. 162 (citing Resp. Ex. A1).[55] Moving forward to October 2014, Petitioner's MRI showed intramedullary T2 prolongation with mild cord enhancement to T9/T10 and T11/T12. Tr. 172; Pet. Ex. 1 at 535.

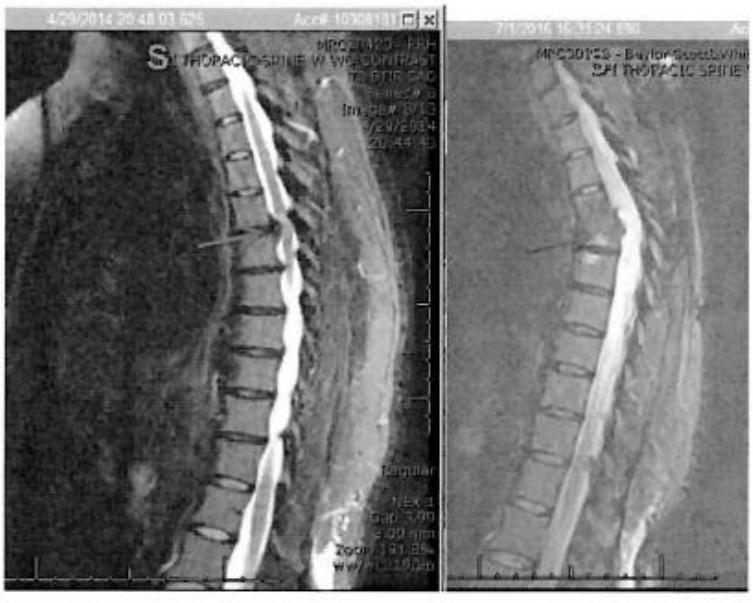
During Petitioner's post-operative course, Dr. Javed opined that there were dynamic changes in her clinical condition due to her infections and treatment for the infections. Tr. 173. In other words, Petitioner's clinical course was not linear, but dynamic. Tr. 174. However, moving forward to December 2015, Petitioner experienced an increase in muscle tone and spasticity evidencing a progression of her compressive myelopathy. Tr. 174-75. This continuous and worsening compression caused more damage to nerve fibers and more venous congestion. Id.

[55] Eoin P. Flanagan et al., Teaching Neuro*Images*: "Pancake-Like" Gadolinium Enhancement Suggests Compressive Myelopathy Due to Spondylosis, 80 Neurology e229 (2013)

ii. Loving Factor Two: What Is Petitioner's Current Condition (or Her Condition Following the Vaccination, If Also Pertinent)?

After vaccination, Dr. Javed opined that Petitioner continued to have transverse myelitis secondary to a mechanical obstruction caused by her herniated disc. Tr. 263. As stated earlier, the surgery was not successful and did not relieve the obstruction. Tr. 160-62. Then she had a post-operative infection that caused inflammation and transverse myelitis. Tr. 263-65. The infection was treated with antibiotics. See Pet. Ex. 1 at 562. Although there was no inflammation or evidence of infection on the MRI done January 8, 2016, Petitioner had "ongoing symptoms of compressive myelopathy which mimic[ked] . . . infection. So it is very hard to tease [] out" what was caused by infection and what was caused by compression. Tr. 266. Regardless, Dr. Javed testified that the MRI done in January 2016 did not show active infection. Tr. 264. Dr. Javed opined that the MRI findings and Petitioner's clinical course were the result of progressive compressive myelopathy not vaccination. Tr. 200.

To illustrate his opinions, Dr. Javed reviewed two images from Petitioner's MRIs: one taken before surgery on April 29, 2014, and the other taken after vaccination on July 1, 2016. Resp. Ex. A at 13.



Id. The arrow on the left image shows "severe compression of the spine from the surrounding tissue" on the MRI dated April 29, 2014, prior to vaccination. Id. The MRI done July 1, 2016 again shows the "severe bony defect" (arrow on the right image) and "more diffuse white signal change . . . due to altered CSF flow." Id. Dr. Javed opined these findings were not due to inflammatory or "vaccine induced transverse myelitis," but the result of severe compression. Id.

Dr. Javed next discussed the MRI done January 8, 2016 (done after Petitioner's flu vaccination on December 11, 2015). Tr. 186 (citing Pet. Ex. 1 at 1211). He opined it showed worsening myelopathy due to compression. Id. Clinically, this MRI is consistent with

"[p]aralysis of the legs, weakness, spasticity, urine retention," and all the other symptoms that Petitioner was experiencing. Id. The MRI report stated the finding was "nonspecific and may be secondary to altered CSF flow dynamics, transverse myelitis [,] and cord ischemia. Correlation with clinical history [was] recommend." Pet. Ex. 1 at 1211. Dr. Javed opined that the most likely cause of the findings was "stenosis and venous congestion." Tr. 187.

When asked to differentiate between symptoms caused by infection/inflammatory myelitis from those caused by compressive myelopathy, Dr. Javed explained that was not possible because "[t]hey're the same. They present the same way." Tr. 266. Thus, it is not possible to tease out signs and symptoms caused of transverse myelitis caused by infection and inflammation from ongoing symptoms of compressive myelopathy. Id.

Moving forward in Petitioner's clinical course, Dr. Javed offered his interpretation of Petitioner's 2018 MRI. Tr. 304-05. He observed that the stenosis or "kink" in Petitioner's spinal cord at T5/T6 was worse, causing kyphosis. Tr. 305. There was dilation or swelling of the spinal cord above and below the lesion (stenosis), and the worsening observed on the MRI was probable due to that finding. Tr. 305-06. Dr. Javed further opined that Petitioner's spinal stenosis (and compressive myelopathy) caused the syrinx and further extension of disease. Tr. 306. He explained that transverse myelitis is a monophasic illness, but the 2018 MRI shows progressive worsening, over four to five years after initial onset. Id. Dr. Javed opined that this progression is not consistent with transverse myelitis, but it is consistent with worsening due to progressive compressive myelopathy. Tr. 306-07.

iii. Loving Factor Three: Does Petitioner's Current Condition (or Condition After Vaccination) Constitute a "Significant Aggravation" of Her Condition Prior to Vaccination?

The parties stipulated that after Petitioner received the flu vaccine on December 11, 2015, that her condition worsened as noted on her MRI imaging and by her treating physicians. Joint Submission at 1-2. While the parties agreed that Petitioner's worsened, they disagreed on the nature or cause of the worsening. Id. at 2. As described above, Dr. Javed opined that Petitioner's worsening after vaccination was caused by progressive compression myelopathy and not an autoimmune reaction to the flu vaccine. Tr. 306-07.

iv. Loving Factor Four/Althen Prong One: Medical Theory of Causation

Dr. Javed opined that the weight of the evidence does not support a finding that the flu vaccine can cause transverse myelitis. Tr. 189. He cited the 2012 Institute of Medicine ("IOM") report, which stated that the "epidemiologic evidence is insufficient or absent to assess an association between [the] [flu] vaccine and transverse myelitis." Resp. Ex. A6 at 3.[56] The IOM

[56] Inst. of Med., Influenza Vaccine, in Adverse Effects of Vaccines: Evidence and Causality 293, 309 (Kathleen Stratton et al. eds., 2012).

committee reviewed six papers that reported "the development of transverse myelitis after administration of a[] [flu] vaccine" and they concluded that aside from a temporal association, there was no other evidence of causality. Id. at 3-4. The committee also observed that the natural flu infection is "rarely" associated with transverse myelitis. Id. at 3. They concluded that while "[a]utoantibodies, T cells, and molecular mimicry may contribute to the symptoms of transverse myelitis," the papers "did not provide evidence linking these mechanisms to [the] [flu] vaccine." Id. Thus, they concluded that any association "based on knowledge about the natural infection" was "weak." Id. Dr. Javed further noted that the flu vaccine at issue here was an inactivated vaccine, not a live viral vaccine, and he inferred that the causal association was lacking due to that fact. Tr. 190.

Next, Dr. Javed opined that here, there is no evidence to support the hypothetical mechanism of a fertile field due to the lack of MRI findings showing autoimmune or infectious inflammation after vaccination. This opinion is discussed more below Loving factor five/Althen prong two.

Regarding Petitioner's theory of molecular mimicry, Dr. Javed opined that to invoke this mechanism "as a likely cause of autoimmune disease, several criteria need to be met."[57] Resp. Ex. A at 16. These include "1) proof that flu antigens are similar to human myelin antigens; 2) proof antibodies or T cells generated against the flu antigens cross react with human myelin antigens; [and] 3) a reliable scientific [link] in the real word that exposure to flu antigen causes human disease."[58] Id. at 16-17. Because there is insufficient evidence to meet these three criteria, Dr. Javed concluded that the flu vaccine does not cause "inflammatory, longitudinal, transverse myelitis." Id. at 17; see also Tr. 193-94.

Dr. Tornatore also discussed the theory of epitope spreading, "whereby invading antigens accelerate an ongoing autoimmune process by local activation of antigen presenting cells and over processing of antigens." Pet. Ex. 13 at 29. In response, Dr. Javed opined that there was no evidence of an ongoing autoimmune response prior to vaccination. Resp. Ex. A at 17. Dr. Javed also disagreed that there was support for the bystander activation theory since there was no evidence that Petitioner had "auto-reactive immune cells prior" to her vaccination. Id.

Dr. Javed discussed the medical literature cited by Dr. Tornatore. See Resp. Ex. A at 13, 15; Tr. 195-96, 238-42. He disagreed that Mealy et al.[59] provided support that vaccinations can

[57] The undersigned does not apply the criteria discussed by Dr. Javed. Instead, the undersigned uses the Loving/Althen criteria and the legal standards set forth below in the legal standards and analysis sections.

[58] During the hearing, Dr. Javed referenced the IOM in support of this criteria; however, he did not cite the portion of the text that contains this criteria. Tr. 244. Again, the undersigned does not adopt this criteria or use it in her analysis as it is not consistent with Loving/Althen.

[59] Maureen A. Mealy et al., Vaccines and the Association with Relapses in Patients with Neuromyelitis Optica Spectrum Disorder, 23 Multiple Sclerosis & Related Disorders 78 (2018).

cause relapses in patients with transverse myelitis because the patients in that paper had NMOSD, which Petitioner did not have. Id. (citing Pet. Ex. 41). The Van Ussel et al. paper reviewed 22 cases of central nervous system demyelinating illnesses associated with either the H1N1 flu vaccine and/or natural infection. Pet. Ex. 21 at 1; see Tr. 238. Five patients had transverse myelitis following vaccination and one after infection. Pet. Ex. 21 at 4 tbl.1. Although Dr. Javed acknowledged the case reports, he compared them to thousands of patients who get natural infections and vaccinations and who do not experience transverse myelitis. Tr. 239, 241. Further, he noted that vaccines are recommended to those with transverse myelitis. Id. Discussing Agmon-Levin et al., Dr. Javed opined that transverse myelitis is rare, and that rare cases can be temporally associated with vaccination, but he disagreed that a temporal association alone could establish cause and effect. Tr. 240-42 (citing Pet. Ex. 18).

Dr. Javed also reviewed the transverse myelitis fact sheet, published by NINDS, which stated that transverse myelitis can be a "post-infectious autoimmune phenomenon." Pet. Ex. 38 at 3. He noted that the statement contrasted with the 2012 IOM findings relative to vaccines and transverse myelitis. Tr. 245. He acknowledged that autoimmune disease causation is multifactorial, and may include "genetic, hormonal, endocrine, infectious, and environmental factors." Tr. 251.

When asked what evidence he would need before he could find that transverse myelitis was caused by a vaccine, Dr. Javed acknowledged that he had "very high standards."[60] Tr. 242. He stated that case reports were insufficient, particularly in this case, because Petitioner here had "ongoing pathology," with very different circumstances than those described in the case reports. Tr. 243. Ultimately, Dr. Javed reiterated that in order to find causation, he would want to see evidence meeting the three-pronged criteria to assess vaccine causation, as discussed earlier. Tr. 242-44.

v. Loving Factor Five/Althen Prong Two: Logical Sequence of Cause and Effect

Dr. Javed opined that the flu vaccine was not the cause of Petitioner's worsening condition after vaccination. Tr. 159. "Inherent in [Petitioner's] claim is first the acknowledgement that she had [] ongoing thoracic myelopathy from another cause unrelated to the vaccine, i.e., spinal cord compression." Resp. Ex. A at 2. Dr. Javed explained that Petitioner had an ongoing myelopathy from spinal cord compression caused by a mechanical obstruction due to a herniated disc. Id.; Tr. 263. She underwent surgery and had a post-operative infection. Tr. 263. Dr. Javed agreed that Petitioner's post-operative infection did cause inflammation correctly identified as transverse myelitis. Id. Petitioner's infection was treated with antibiotics, and it was cleared up by the time of her flu vaccine on December 11, 2015. Tr. 264. Dr. Javed testified that there was no evidence of inflammation/myelitis seen in Petitioner's MRI done January 8, 2016. Id. There was no evidence of T2 prolongation representing inflammation, no enhancement, and no evidence of osteomyelitis or discitis. Tr. 178, 264. Although enhancement on MRI is not always present, it is often present, and if she had infection, Dr. Javed would have

[60] Again, Dr. Javed's standards and criteria are not used by the undersigned. Dr. Javed's expert medical opinions are evaluated using the applicable legal framework.

expected to see enhancement. Tr. 264. Thus, while he agreed that Petitioner had a post-operative infection that caused transverse myelitis, he strongly disagreed that there was evidence of transverse myelitis after her vaccination, specifically on the MRI done post-vaccination (January 8, 2016). Id.

In reviewing the Petitioner's records, Dr. Javed noted confusion with the use of the word inflammation. Tr. 301-02. He explained that there is inflammation caused by compression as well as that due to an autoimmune cause. Id. The word myelitis does not distinguish between these causes. Tr. 302. He explained, "[w]hen you squeeze the [spinal] cord, some of the fibers die [and] some of the fibers [] inflame." Tr. 301.

Dr. Javed explained that Petitioner's treating physicians did not perform a lumbar puncture after vaccination to determine whether there was inflammation in the CSF. Tr. 192. He suggested that a lumbar puncture was not done because Petitioner's physicians did not see inflammation suggestive of transverse myelitis on her MRI, and so they did not think the procedure was warranted. Id. Moreover, Petitioner's MRI showed no diffuse enhancement to indicate inflammation consistent with transverse myelitis. Tr. 198-99.

In support of his opinion that if Petitioner's treating physicians had suspected acute transverse myelitis after her flu vaccination, they would have ordered diagnostic testing and treatment with steroids, Dr. Javed filed a case report by Bakshi and Mazziotta. Resp. Ex. A at 13-14; Pet. Ex. 39. There, the MRI showed enlarged spinal cord from C3 to the upper thoracic cord and CSF showed elevated inflammatory markers. Pet. Ex. 39 at 1. The patient was treated with intravenous steroids which led to "a marked recovery by [two] weeks." Id.

According to Dr. Javed, Petitioner was able to walk after her initial back surgery because the surgery may have relieved some of the pressure on her spinal cord. Tr. 202-03. Additionally, she received pain medications, therapy, and other treatment. Id. Although she may have had some improvement, Dr. Javed opined that she "never really [had] progressive improvement" after surgery. Tr. 203. Her course was complicated by infection and an abscess which caused inflammation. Id. After treatment (with antibiotics), the inflammation subsided, and as a result there may have been some improvement. Tr. 202-03.

Further, Dr. Javed observed that the physician who interpreted Petitioner's post-vaccination MRI on January 8, 2016, noted "[i]nterval development of expansile T2 prolongation/edema within the thoracic cord extending between T1/T2 and T10. Associated intramedullary syringohydromyelia [was] noted at T6 and T7 without enhancement. Finding [was] nonspecific and may be secondary to altered CSF flow dynamics, transverse myelitis and cord ischemia." Resp. Ex. A at 2 (quoting Pet. Ex. 1 at 1211). Based on this report, Dr. Javed opined that the radiologist mentioned three possible causes for the findings.[61] Id. at 3. It was Dr. Javed's opinion that the "progression of [Petitioner's] symptoms and changes in the MRI are related to the progressive of her pre-existing compressive myelopathy." Id.

[61] Two of these included altered CSF flow dynamics and cord ischemia (which Dr. Javed opined was caused by compression). Tr. 227-28.

On cross-examination at the hearing, Dr. Javed was questioned about many of Petitioner's medical records. When questioned about PT records from December 2015, documenting changes in Petitioner's condition, Dr. Javed agreed that Petitioner had complaints, but he opined that her symptoms were fluctuating because her "underlying process . . . was worsening." Tr. 216.

During this period of questioning, Dr. Javed was asked about Petitioner's treating neurologist, Dr. Tramonte, and his records dated January 20, 2016, when he saw Petitioner for "worsening weakness, sensory loss, and spasticity . . . since the middle of December 2015."[62] Tr. 218; see Pet. Ex. 2 at 864. Dr. Javed noted that Dr. Tramonte recounted Petitioner's "history of thoracic spondylitic myelopathy." Tr. 218 (quoting Pet. Ex. 2 at 863). Dr. Javed noted that in December 2015, Dr. Tramonte concluded that Petitioner had "T4 thoracic myelopathy due to calcified dis[c] herniation." Tr. 219 (quoting Pet. Ex. 2 at 864).

Dr. Javed was also questioned about Dr. Garrett's records from January 2016 and specifically about the assessment which stated, "thoracic transverse myelitis due to prior compression of spinal cord, meningitis, or even as autoimmune phenomenon from flu vaccine." Tr. 224; see Pet. Ex. 3 at 8250. Dr. Javed explained that an autoimmune reaction from the flu vaccine was only one of the possibilities mentioned by Dr. Garrett. Tr. 224-26. Dr. Javed testified that in July 2016, Dr. Garrett also documented an assessment stating that Petitioner's worsening "correlate[d] with a prolonged T2 signal within the spinal cord but [could not] tell if [it was] a new ischemia or myelitis." Tr. 227 (quoting Pet. Ex. 3 at 8349). Dr. Javed opined that the reference to ischemia meant lack of oxygen due to worsening compression. Tr. 227-28.

Dr. Frohman's medical records documented a history taken in November 2017, which state that Petitioner had a "slow but steady improvement" after surgery until December 2015. Pet. Ex. 10 at 37. When questioned about the note, Dr. Javed explained that it was written one-and-one-half years after the events at issue. Tr. 204-05. Dr. Javed also agreed that Petitioner did have improvement after surgery, although he characterized this period as Petitioner having "good days and bad days." Tr. 204. As her infection improved, she probably "had slow, steady improvement." Tr. 205. However, Petitioner's progressive compressive myelopathy was not

[62] Dr. Tramonte's records from this visit on January 20, 2016, include his assessment that Petitioner had a "T4 thoracic myelopathy due to calcified dis[c] herniation, [status post] decompression, followed by a complicated course of infection with empyema, abscess and compression of the conus medullaris and cauda equina in 2014." Pet. Ex. 2 at 867. Her chief complaint at the time of her December 30, 2015 visit with Dr. Tramonte "was worsening weakness, spasticity, and sensory loss in the left leg since the middle of Dec[ember] 2015. Her MRIs showed worsening pre-existing signal abnormality in the thoracic spinal cord from T1-T10, a change that initially was present after developing a T4-T11 epidural abscess, along with new syringomyelia at T6/7." Id. Dr. Tramonte suspected "the changes were ischemic in nature" and "referred her for aqua therapy, and scheduled repeat MRIs . . . in [six] months." Id. It does not appear that Petitioner had the repeat MRI six months later. Dr. Tramonte's referral order for aqua therapy stated "[e]val[uate] and treat weakness in legs due to thoracic myelopathy." Id. at 862.

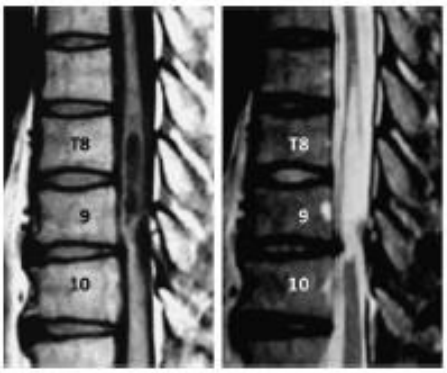
being treated. Id. And Dr. Javed again emphasized that a lumbar puncture was not performed and there was no workup to diagnose transverse myelitis. Tr. 209.

Regarding the references in the medical records to Petitioner's allergies and the flu vaccine being identified as an allergy, Dr. Javed disagreed that this was evidence of vaccine causation. Tr. 212, 230-31 (referencing Dr. Frohman's and Dr. Tramonte's records). Dr. Javed explained that allergies are usually charted by nursing staff, not physicians. Tr. 213. Additionally, with regard to the records which state that Petitioner developed TM after a flu vaccine, Dr. Javed also opined that these records were not evidence of vaccine causation. Tr. 221-23 (citing Pet. Ex. 2 at 864); see also Pet. Ex. 1 at 1293 ("[T]here was consideration that the changes could be vaccine relate myelitis from the flu shot.").

While some of Petitioner's treating physicians questioned the role of Petitioner's flu shot, Dr. Javed opined that they did not conclude that it was the cause of her illness, or that it caused an aggravation of her condition. Tr. 227-31; Resp. Ex. A at 10-11. Dr. Javed further explained that Dr. Garrett questioned whether Petitioner's symptoms were due to cord compression, although he concluded that surgical intervention was not warranted. Tr. 232-33, 236-38.

Dr. Javed also disagreed with Dr. Frohman's diagnosis of transverse myelitis. Tr. 197-98. He emphasized, however, that Dr. Frohman did not attribute Petitioner's transverse myelitis to vaccination. Pet. Ex. 10 at 40. Dr. Frohman wrote that he "suspect[ed] that [Petitioner] did in fact have transverse myelitis, although the pathoetiological underpinnings of this process remain enigmatic." Id. Based on this note, Dr. Javed opined that Dr. Frohman did not attribute the cause of Petitioner's condition to the flu vaccine. Tr. 198-99.

Another fact important to Dr. Javed's opinion that Petitioner did not have post-vaccination transverse myelitis was that her MRI on January 8, 2016 showed an intermedullary syringohydromyelia at T6 and T7. Resp. Ex. A at 13. He defined syringohydromyelia as a "syrinx, a fluid filled cavity in the spinal cord from chronic and slow blockage of CSF fluid caused by subarachnoid space [] flow blockage, i.e., from spinal stenosis." Id. He asserted that "[a]cute transverse myelitis does not cause syringohydromyelia/syrinx." Id. The following image shows the syrinx at T8-9:



Resp. Ex. A4 at 2 fig.1.[63]

In support of his opinion, Dr. Javed cited Arai et al., which reported a case of spinal stenosis that caused an intramedullary syringohydromyelia. Resp. Ex. A at 14 (citing Resp. Ex. A4 at 1). The authors of the paper explained that "[l]ong-standing blockade of [CSF] circulation is considered to be one of the main causes of syringomyelia associated with degenerative diseases of the cervical spine." Resp. Ex. A4 at 1. In the case reported, the patient had a disc herniation at T9 and T10, and the syringomyelia occurred in that part of the spine. Id. The authors discussed CSF flow dynamics and the improvement which can be achieved by decompression of the spinal cord compression. Id. at 2-3. They concluded that although rare, "long-standing compression of the spinal cord due to spinal spondylosis could . . . be one of the main causes of syringomyelia." Id. at 3.

In further support of his opinion that Petitioner suffered from compressive myelopathy and not post-vaccination transverse myelitis, Dr. Javed cited several other articles. See Resp. Ex. A at 10-13. A paper by Kelley et al.[64] described five patients with compressive myelopathy that mimicked transverse myelitis. Tr. 184 (citing Resp. Ex. A2). All five patients were suspected to have transverse myelitis but were ultimately diagnosed with spinal stenosis. Resp. Ex. A2 at 1. The authors explained that "[c]ompressive myelopathy is occasionally associated with [] T2 signal changes on [MRI]. When this occurs, the radiologic appearance can result in misdiagnosis of transverse myelitis." Id. Specific to the facts here, the authors stated that in the case of longitudinally extensive transverse myelitis appearance on MRI, there are other causes that are important to recognize, including "in the setting of myelopathy . . . symptomatic spinal stenosis." Id. at 2. All five of the patients with "compressive myelopathy experienced progressive neurological dysfunction over months, as reflected in the mean duration from onset to symptom nadir or interventions, whereas patients with transverse myelitis characteristically reach[ed] a nadir of deficit within [two] weeks." Id.

Flanagan et al. described a 41-year old male with progressive hand weakness and paraparesis. Resp. Ex. A1 at 1. The patient deteriorated in spite of steroid treatment. Id. One year later, he underwent cervical decompression and his clinical condition stabilized. Id. The authors explained that MRI enhancement "due to focal disruption of the blood brain barrier at the point of maximal stenosis strongly suggest[ed] cervical stenosis as the cause of myelopathy." Id.

Regarding Petitioner's theory that there was a "fertile field" which allowed for an immune response to cause transverse myelitis, Dr. Javed disagreed that there was evidence to support a fertile field mechanism. Tr. 191-92. He explained that the MRI done in January 2016 did not show evidence of infection, discitis, enhancement, or meningitis. Tr. 175-77. The MRI did show a lot of bony destruction. Tr. 178. Dr. Javed opined that the MRI showed worsening compressive myelopathy. Tr. 177, 186. The pressure on Petitioner's spine looked worse. Id.

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[63] Atsushi Arai et al., Syringomyelia Due to Thoracic Spinal Stenosis with Ossified Ligamentum Flavum, 51 Neurologia medico-chirurgica 157 (2011).

[64] Brendan J. Kelley et al., Compressive Myelopathy Mimicking Transverse Myelitis, 16 Neurologist 120 (2010).

According to Dr. Javed, Petitioner's compressive myelopathy got worse because her spinal cord nerves were damaged, causing more venous congestion, and worsening of her condition. Tr. 175.

In addition to the lack of findings to support a "fertile field" for the development of an autoimmune reaction, Dr. Javed also explained that none of Petitioner's treating physicians treated her for transverse myelitis after her vaccination. See Tr. 167, 198-99, 231, 305. If Petitioner's treating doctors thought she had autoimmune transverse myelitis, Dr. Javed asserted that she would have received diagnostic testing (a lumbar puncture and CSF analysis) to confirm the diagnosis. Tr. 163-64, 171, 207, 209. And if the diagnosis of transverse myelitis was confirmed by the presence of inflammation in the CSF, treatment would have been initiated. Tr. 266-71. But neither Petitioner's neurosurgeon (Dr. Garrett) nor her neurologist (Dr. Tramonte) performed diagnostic testing or treated her for transverse myelitis. Tr. 164, 271.

Dr. Javed disagreed with Dr. Tornatore that treatment for transverse myelitis, including IVIG and plasma exchange, were contraindicated due to Petitioner's condition or clinical course. Tr. 303. Even in patients with high risk of developing complications, like clotting, Dr. Javed opined that the risk of clots was very low. Id.

Lastly, Dr. Javed opined that it was "extremely unlikely" that the vaccine caused autoimmune inflammation of the spinal cord that continued for a period of four or five years (as evidenced by Petitioner's MRI in 2018). Tr. 306. He emphasized that transverse myelitis is a monophasic illness, not a progressive one. Id. Thus, he opined that it is more probable that Petitioner's worsening was caused by compressive myelopathy. Tr. 307.

vi. Loving Factor Six/Althen Prong Three: Proximate Temporal Relationship

Dr. Javed agreed that given Petitioner received the vaccination at issue on December 11, 2015, and approximately one week later complained of weakness, there was an appropriate time period for onset of an autoimmune reaction (transverse myelitis). Tr. 261. However, he believed that Petitioner was "already declining prior to vaccination." Resp. Ex. A at 16. "Given that [Petitioner] had very low reserve in the spinal cord to begin with (i.e., added injuries from spinal cord damage from stenosis + injury from abscess + injury from discitis), a slowly developing process may have a sharp inflection point in terms of clinical decline." Id. He opined "[v]accination in this case would be [] a mere temporal association (coincidental) and not a causal association." Id.

V. LEGAL FRAMEWORK

A. Standard of Adjudication—Factual Issues

A petitioner must prove, by a preponderance of the evidence, the factual circumstances surrounding her claim. § 13(a)(1)(A). To resolve factual issues, the special master must weigh the evidence presented, which may include contemporaneous medical records and testimony. See Burns v. Sec'y of Health & Hum. Servs., 3 F.3d 415, 417 (Fed. Cir. 1993) (explaining that a

special master must decide what weight to give evidence including oral testimony and contemporaneous medical records). Contemporaneous medical records, "in general, warrant consideration as trustworthy evidence." Cucuras v. Sec'y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). But see Kirby v. Sec'y of Health & Hum. Servs., 997 F.3d 1378, 1382 (Fed. Cir. 2021) (rejecting the presumption that "medical records are accurate and complete as to all the patient's physical conditions"); Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 538 (2011) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance." (quoting Murphy v. Sec'y of Health & Hum. Servs., 23 Cl. Ct. 726, 733 (1991), aff'd per curiam, 968 F.2d 1226 (Fed. Cir. 1992))), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 503 F. App'x 952 (Fed. Cir. 2013).

There are situations in which compelling testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. Campbell v. Sec'y of Health & Hum. Servs., 69 Fed. Cl. 775, 779 (2006) ("[L]ike any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking."); Lowrie v. Sec'y of Health & Hum. Servs., No. 03-1585V, 2005 WL 6117475, at *19 (Fed. Cl. Spec. Mstr. Dec. 12, 2005) ("[W]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent." (quoting Murphy, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. Andreu v. Sec'y of Health & Hum. Servs., 569 F.3d 1367, 1379 (Fed. Cir. 2009); Bradley v. Sec'y of Health & Hum. Servs., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

Despite the weight afforded to medical records, special masters are not bound rigidly by those records in determining onset of a petitioner's symptoms. Valenzuela v. Sec'y of Health & Hum. Servs., No. 90-1002V, 1991 WL 182241, at *3 (Fed. Cl. Spec. Mstr. Aug. 30, 1991); see also Eng v. Sec'y of Health & Hum. Servs., No. 90-1754V, 1994 WL 67704, at *3 (Fed. Cl. Spec. Mstr. Feb. 18, 1994) ("[Section 13(b)(2)] must be construed so as to give effect also to § 13(b)(1) which directs the special master or court to consider the medical records (reports, diagnosis, conclusions, medical judgment, test reports, etc.), but does not require the special master or court to be bound by them." (emphasis omitted)).

B. Standards for Adjudication—Causation

The Vaccine Act was established to compensate vaccine-related injuries and deaths. § 10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" Rooks v. Sec'y of Health & Hum. Servs., 35 Fed. Cl. 1, 7 (1996) (quoting H.R. Rep. No. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

Petitioner's burden of proof is by a preponderance of the evidence. § 13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1315,

1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. Bunting v. Sec'y of Health & Hum. Servs., 931 F.2d 867, 873 (Fed. Cir. 1991). Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano v. Sec'y of Health & Hum. Servs., 440 F.3d 1317, 1325 (Fed. Cir. 2006). Instead, Petitioner may satisfy her burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

In particular, Petitioner must prove that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface v. Sec'y of Health & Hum. Servs., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); see also Pafford v. Sec'y of Health & Hum. Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). The received vaccine, however, need not be the predominant cause of the injury. Shyface, 165 F.3d at 1351. A petitioner who satisfies this burden is entitled to compensation unless Respondent can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors unrelated to the administration of the vaccine." § 13(a)(1)(B). However, if a petitioner fails to establish a prima facie case, the burden does not shift. Bradley, 991 F.2d at 1575.

"Regardless of whether the burden ever shifts to the [R]espondent, the special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner has established a prima facie case." Flores v. Sec'y of Health & Hum. Servs., 115 Fed. Cl. 157, 162-63 aff'd, 586 F. App'x 588 (Fed. Cir. 2014); see also Stone v. Sec'y of Health & Hum. Servs., 676 F.3d 1373, 1379 (Fed. Cir. 2012) ("[E]vidence of other possible sources of injury can be relevant not only to the 'factors unrelated' defense, but also to whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question."); de Bazan v. Sec'y of Health & Hum. Servs., 539 F.3d 1347, 1353 (Fed. Cir. 2008) ("The government, like any defendant, is permitted to offer evidence to demonstrate the inadequacy of the [P]etitioner's evidence on a requisite element of the [P]etitioner's case-in-chief."); Pafford, 451 F.3d at 1358-59 ("[T]he presence of multiple potential causative agents makes it difficult to attribute 'but for' causation to the vaccination. . . . [T]he Special Master properly introduced the presence of the other unrelated contemporaneous events as just as likely to have been the triggering event as the vaccinations.").

To receive compensation through the Program, Petitioner must prove either (1) that she suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that she received, or (2) that she suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); Capizzano, 440 F.3d at 1319-20. Because Petitioner does not allege she suffered a Table Injury, she must prove a vaccine she received caused her injury. To do so, Petitioner must establish, by preponderant evidence: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." Althen v. Sec'y of Health & Hum. Servs., 418 F.3d 1274, 1278 (Fed. Cir. 2005).

The causation theory must relate to the injury alleged. Petitioner must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the

explanation need only be "legally probable, not medically or scientifically certain." Knudsen v. Sec'y of Health & Hum. Servs., 35 F.3d 543, 543, 548-49 (Fed. Cir. 1994). Petitioner cannot establish entitlement to compensation based solely on his assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). In determining whether Petitioner is entitled to compensation, the special master shall consider all materials in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 13(b)(1)(A). The undersigned must weigh the submitted evidence and the testimony of the parties' proffered experts and rule in Petitioner's favor when the evidence weighs in her favor. See Moberly, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence."); Althen, 418 F.3d at 1280 (noting that "close calls" are resolved in Petitioner's favor).

Testimony that merely expresses the possibility—not the probability—is insufficient, by itself, to substantiate a claim that such an injury occurred. See Waterman v. Sec'y of Health & Hum. Servs., 123 Fed. Cl. 564, 573-74 (2015) (denying Petitioner's motion for review and noting that a possible causal link was not sufficient to meet the preponderance standard). The Federal Circuit has made clear that the mere possibility of a link between a vaccination and a petitioner's injury is not sufficient to satisfy the preponderance standard. Moberly, 592 F.3d at 1322 (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and the injury" does not equate to proof of causation by a preponderance of the evidence); Boatmon v. Sec'y of Health & Hum. Servs., 941 F.3d 1351, 1359-60 (Fed. Cir. 2019). While certainty is by no means required, a possible mechanism does not rise to the level of preponderance. Moberly, 592 F.3d at 1322; see also de Bazan, 539 F.3d at 1351.

C. Standards for Adjudication—Significant Aggravation

The elements of an off-Table significant aggravation case are set forth in Loving. See Loving, 86 Fed. Cl. at 142-44; see also W.C. v. Sec'y of Health & Hum. Servs., 704 F.3d 1352, 1357 (Fed. Cir. 2013) (holding that "the Loving case provides the correct framework for evaluating off-table significant aggravation claims"). The Loving court combined the Althen test, which defines off-Table causation cases, with a test from Whitecotton. Whitecotton v. Sec'y of Health & Hum. Servs., 17 F.3d 374 (Fed. Cir. 1994), rev'd sub nom., Shalala v. Whitecotton, 514 U.S. 268 (1995) (concerning on-Table significant aggravation cases). The resultant test has six components, which are:

> (1) the person's condition prior to administration of the vaccine, (2) the person's current condition (or the condition following the vaccination if that is also pertinent), (3) whether the person's current condition constitutes a 'significant aggravation' of the person's condition prior to vaccination, (4) a medical theory causally connecting such a significant worsened condition to the vaccination, (5) a logical sequence of cause and effect showing that the vaccination was the reason for the significant aggravation, and (6) a showing of a proximate temporal relationship between the vaccination and the significant aggravation.

Loving, 86 Fed. Cl. at 144.

The statute defines "significant aggravation" as "any change for the worse in a pre-existing condition which results in markedly greater disability, pain, or illness accompanied by substantial deterioration in health." § 33(4).

VI. ANALYSIS

A. Significant Aggravation

1. Loving Factor One: What Was Petitioner's Condition Prior to Administration of the Vaccine?

The first step in the Loving test is to determine Petitioner's condition prior to the administration of her flu vaccine on December 11, 2015. The parties agree and stipulate that prior to receiving the flu vaccine at issue on December 11, 2015, Petitioner "suffered from a pre-existing thoracic myelopathy." Joint Submission at 1. Petitioner's medical records establish, and both experts generally agree, that in March/April 2014 Petitioner developed spinal cord-related neurologic symptoms caused by a herniated disc/mass compressing the spinal cord at T5 and T6. Both experts agree that Petitioner had significant stenosis in her thoracic spinal cord. Dr. Javed opines that the disc was compressing the spinal cord by about 50%. The stenosis caused edema (swelling) and inflammation of the spinal cord seen on MRI. Petitioner underwent decompressive surgery on June 27, 2014.

The medical records show, and the experts agreed, that post-operatively, Petitioner developed a very profound post-operative infection around her spinal cord along with an epidural and thoracic abscess. This infection required a second operative procedure and lengthy treatment with antibiotics. Dr. Tornatore opined that about six weeks after her second surgery, Petitioner developed longitudinally extensive transverse myelitis, as seen on her MRI August 26, 2014. Ultimately, Dr. Javed agreed that due to the infection, Petitioner developed transverse myelitis. Two months later, on October 21, 2014, an MRI showed improvement. Both experts acknowledged that Petitioner improved some after surgery and antibiotics.

Both experts agreed that the initial back surgery did not result in decompression of the stenosis in Petitioner's spinal cord. Both experts agreed that there was a component of compressive myelopathy causing Petitioner's condition. Thus, the undersigned finds that prior to her vaccination, Petitioner had thoracic myelopathy with compression and inflammatory/infectious myelitis; that Petitioner had a spinal cord injury and paraplegia; and that she had a limited ability to walk with a rolling walker, although she had difficulty with use of her right foot and problems with spasticity and tone that limited her ability to stand, pivot, and walk.

2. Loving Factor Two: What Is Petitioner's Current Condition (or Her Condition Following the Vaccination, If Also Pertinent)?

The second part of the Loving test is to discuss "the person's current condition (or condition following the vaccination if that is also pertinent)." Loving, 86 Fed. Cl. at 144. Here, Petitioner's condition following vaccination through 2018 is most pertinent.

To the extent that Petitioner's affidavit or histories noted in the medical records are inconsistent with and contradicted by the physicians' objective physical examinations or diagnostic testing, the undersigned defers to the contemporaneous physician findings as the most reliable source of information. See Cucuras, 993 F.2d at 1528 (noting that "the Supreme Court counsels that oral testimony in conflict with contemporaneous documentary evidence deserves little weight"); Doe/70 v. Sec'y of Health & Hum. Servs., 95 Fed. Cl. 598, 608 (2010); Stevens v. Sec'y of Health & Hum. Servs., No. 90-221V, 1990 WL 608693, at *3 (Cl. Ct. Spec. Mstr. Dec. 21, 1990) (noting that "clear, cogent, and consistent testimony can overcome such missing or contradictory medical records"); Vergara v. Sec'y of Health & Hum. Servs., No. 08-882V, 2014 WL 2795491, at *4 (Fed. Cl. Spec. Mstr. May 15, 2014) ("Special Masters frequently accord more weight to contemporaneously-recorded medical symptoms than those recorded in later medical histories, affidavits, or trial testimony.").

Petitioner received her flu vaccine on December 11, 2015. On December 18, 2015, Petitioner reported that her spasticity was worse and she was unable to stand. This was also noted at her PT visit on December 30, 2015, when Petitioner's increased spasticity made it difficult for her to bear weight. Based on the records filed, Petitioner has not been able to ambulate, even for short distances, since December 2015.

Subsequent MRI studies, done in 2016 and 2018, show progressive worsening. On January 8, 2016, the MRI showed expansile prolongation/edema and intramedullary syringohydromyelia within the thoracic spinal cord that "may be secondary to altered CSF flow dynamics, transverse myelitis[,] and cord ischemia." Pet. Ex. 1 at 1211. A July 1, 2016 MRI showed "progression of abnormal signal and expansion" of the thoracic cord. Pet. Ex. 3 at 8321. On November 9, 2018, the MRI showed "extension of expansile cord signal abnormality" and multilevel cervical spondylosis with "varying degrees of mild foraminal narrowing and mild to moderate spinal stenosis." Pet. Ex. 12 at 23. The undersigned finds that since vaccination, Petitioner has experienced a progressive worsening of her clinical course and MRI findings.

3. Loving Factor Three: Does Petitioner's Current Condition (or Condition After Vaccination) Constitute a "Significant Aggravation" of Her Condition Prior to Vaccination?

The next factor of the Loving test is to determine whether there is a "significant aggravation" of Petitioner's condition by comparing her condition before vaccination to her condition after vaccination. The statute defines "significant aggravation" as "any change for the worse in a pre-existing condition which results in markedly greater disability, pain, or illness accompanied by substantial deterioration in health." § 33(4). Using this definition, the

undersigned finds that, based on all of the facts and circumstances here, Petitioner did have a significant aggravation of her underlying condition.

The parties stipulated that Petitioner's condition worsened post-vaccination. Joint Submission at 2. The undersigned agrees that prior to her vaccination on December 11, 2015, Petitioner had a limited ability to ambulate with a rolling walker and that after vaccination she no longer could do so.

As explained by Dr. Tornatore, Petitioner's condition worsened after her vaccination. Prior to December 11, 2015, Petitioner could ambulate short distances with a rolling walker. Beginning mid-December 2015, approximately December 18, she developed increasing spasticity, particularly in her left leg. This increase in spasticity limited her ability to stand, pivot, and ambulate. She was unable to walk with her walker. The increase in spasticity was concerning enough that it was reported to her physician.

Based on the parties' joint submission, the PT records, Petitioner's affidavit, and the opinions of Dr. Tornatore, the undersigned finds that the Petitioner developed a worsening of her condition in mid-December 2015, which constituted a significant aggravation of her condition as compared to her condition before vaccination on December 11, 2015.

4. Loving Factor Four/Althen Prong One: Medical Theory of Causation

The fourth Loving factor has its origins in Althen prong one, and Petitioner must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. Andreu, 569 F.3d at 1379; Pafford, 451 F.3d at 1355-56. Petitioner's theory of causation need not be medically or scientifically certain, but it must be informed by a "sound and reliable" medical or scientific explanation. Boatmon, 941 F.3d at 1359; see also Knudsen, 35 F.3d at 548; Veryzer v. Sec'y of Health & Hum. Servs., 98 Fed. Cl. 214, 223 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both "relevant" and "reliable"). If Petitioner relies upon a medical opinion to support her theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. See Broekelschen v. Sec'y of Health & Hum. Servs., 618 F.3d 1339 at 1347 (Fed. Cir. 2010) ("The special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories."); Perreira v. Sec'y of Health & Hum. Servs., 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) (stating that an "expert opinion is no better than the soundness of the reasons supporting it" (citing Fehrs v. United States, 620 F.2d 255, 265 (Ct. Cl. 1980))).

This case is different than any other transverse myelitis case that the undersigned has adjudicated. Dr. Tornatore offered opinions about several mechanisms by which the flu vaccine can cause transverse myelitis. However, in this case, the starting point is a spinal cord that is already injured due to compression from spinal stenosis, longstanding inflammation from that compression and associated ischemia, and injury from inflammation/infectious processes (transverse myelitis) secondary to a severe post-operative infection. The facts and circumstances here are therefore novel. Neither party filed literature or case reports that explain how a flu

vaccine can cause more injury to a spinal cord that is already significantly damaged by pre-existing compressive myelopathy and transverse myelitis.

The undersigned's findings in this case primarily turn on Loving factor five/Althen prong two, whether there is a logical sequence of cause and effect showing that the vaccination significantly aggravated Petitioner's already injured spinal cord. However, the undersigned also finds that Petitioner's "fertile field" model of causation does not provide a sound and reliable explanation of how the vaccine can aggravate transverse myelitis.

According to Dr. Tornatore, under the fertile field model, "activation of an immune response by an immunogen with sequence homology to self-proteins may prime autoreactive T-cells in the host, but might lack the momentum to initiate an adverse autoimmune reaction independently." Pet. Ex. 13 at 30. "However, a later immune stimulation or coincidental immune stimulation event by a vaccine or other immunogen, even one without cross-reactive antigens or sequence similarity, could initiate an autoimmune reaction in a susceptible host that could lead to inflammatory demyelination." Id.

In support of the fertile field model, Dr. Tornatore relied on a paper by Fujinami et al. The authors discuss how autoimmunity could develop in the context of a persistent viral infection. "Persistent viral infections can lead to immune-mediated injury due to the constant presence of viral antigen driving the immune response." Pet. Ex. 25 at 2. An example of a persistent viral antigen occurs in Theiler's encephalomyelitis in mice which causes a central nervous system infection. After the mice recover from an acute infection in the neurons, they develop persistent infection in glial cells, and the infection can be detected throughout the lifetime of the animal.

Fujinami et al. identified MS, myocarditis, and diabetes as three illnesses where the fertile field model is being studied in animal models. There is a "priming infection (setting up the fertile field) [that] increases the number of autoreactive T cells but not sufficiently to cause disease." Pet. Ex. 25 at 6. Fujinami et al. described the presence of a lifelong viral infection that leads to a continuing persistent viral antigen being present in the body without causing overt illness.

However, Fujinami et al. did not describe an acute episode of a bacterial infection (such as MRSA), which has been treated with antibiotics, as a lifelong infection that develops a persistent infection in the glial cells of the spinal cord. And Petitioner did not provide any evidence that a MRSA infection could act in the same way as a persistent viral infection described by Fujinami et al. in neurons or glial cells.

Second, transverse myelitis is not identified as a disease that may be explained by the "fertile field" model. The three diseases described by Fujinami et al. are MS, diabetes, and myositis. While MS involves the central nervous system (like transverse myelitis) it is not a monophasic disease like transverse myelitis, but is instead a progressive disease. Petitioner did not file any evidence to support the application of the "fertile field" model as playing a role in the cause of transverse myelitis.

For the above reasons, the undersigned finds that Petitioner has not offered preponderant evidence of a sound and reliable mechanism to explain how the flu vaccine can cause significant aggravation of a pre-existing spinal cord injury.

5. Loving Factor Five/Althen Prong Two: Logical Sequence of Cause and Effect

Under Loving factor five and Althen prong two, Petitioner must prove by a preponderance of the evidence that there is a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." Capizzano, 440 F.3d at 1324 (quoting Althen, 418 F.3d at 1278). "Petitioner must show that the vaccine was the 'but for' cause of the harm . . . or in other words, that the vaccine was the 'reason for the injury.'" Pafford, 451 F.3d at 1356 (internal citations omitted).

In evaluating whether this prong is satisfied, the opinions and views of the vaccinee's treating physicians are entitled to some weight. Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326 ("[M]edical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" (quoting Althen, 418 F.3d at 1280)). Medical records are generally viewed as trustworthy evidence, since they are created contemporaneously with the treatment of the vaccinee. Cucuras, 993 F.2d at 1528. Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano, 440 F.3d at 1325. Instead, Petitioner may satisfy her burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

Regarding the fifth Loving factor/second Althen prong, the undersigned finds that Petitioner has failed to show by preponderant evidence that the flu vaccination significantly aggravated her pre-existing spinal cord condition. Instead, the undersigned agrees with Respondent that Petitioner's worsening reflects an evolution or progression of her pre-existing conditions. There are several reasons for this finding.

First, Petitioner's clinical course began with compressive myelopathy caused by spinal stenosis and disc herniation. In February 2014, Petitioner had thoracic pain, numbness, and tingling, and she developed occasional weakness in her legs. Dr. Tramonte diagnosed her with T4 thoracic myelopathy. In April 2014, her thoracic MRI was abnormal, showing a partially calcified mass at T5/T6, which, as explained by Dr. Javed, compressed her spinal cord by 50%. Petitioner was referred to a neurosurgeon, Dr. Garrett, who recommended resection of the mass due to Petitioner's compressive myelopathy symptoms. Dr. Tornatore agreed that Petitioner had compression myelopathy prior to surgery.

Prior to surgery, Petitioner was diagnosed with spinal cord myelopathy by her neurologist and neurosurgeon, and her MRI showed thoracic spinal cord compression. Both experts agreed that Petitioner had compressive myelopathy. Therefore, the undersigned finds that consistent with the medical records, treating physicians, diagnostic images, and experts, that in 2014, prior

to vaccination, Petitioner had compressive myelopathy due to compression of her spinal cord at T5/T6.

In June 2014, Petitioner had spinal surgery. During surgery, the motor evoked potential signal from the right leg area was lost. Post-operatively, Dr. Garrett observed that Petitioner could not move her right leg. CT scan on June 27 showed Petitioner continued to have spinal stenosis at T5 and T6. On July 9, she was unable to walk. MRI showed an extensive epidural abscess from T4 down to T11, which "result[ed] in multilevel mild and moderate spinal canal stenosis." Pet. Ex. 3 at 2079-84. Petitioner also still had T5/T6 disc herniation with "moderate to severe spinal stenosis." Id. Thus, the evidence supports a finding that Petitioner continued to have spinal stenosis and compressive myelopathy. In addition to compressive myelopathy, MRI showed dural enhancement with epidural abscess. According to Dr. Tornatore, in July 2014, Petitioner also had "inflammatory myelitis" as evidenced not only by her MRI, but also her very elevated white blood cell count in her CSF. Therefore, the undersigned finds that in July 2014, Petitioner had compressive myelopathy and an infectious/inflammatory myelitis.

Petitioner was taken back to surgery, and her post-operative course was complicated by MRSA bacteremia, MRSA epidural abscess, right chest empyema with chest tube required for drainage, and persistent CSF leak. Petitioner was very ill and required intubation and ICU admission. She was discharged to a rehabilitation facility on August 1, 2014, but readmitted to the hospital on August 26, 2014 with sudden bilateral lower extremity weakness and the inability to move either of her legs. MRI of the thoracic cord showed increased intramedullary signal extending throughout her entire thoracic spine. The appearance was described as nonspecific, but "may be the result of venous congestion/edema, cord ischemia or potential infectious myelitis." Pet. Ex. 3 at 7812. At T5 and T6, there were "anterior and posterior disc herniations compressing the spinal cord compatible with discitis osteomyelitis." Id. In other words, Petitioner continued to have compression of the spinal cord which was now complicated by infection/inflammation. The undersigned finds that Petitioner had an infectious/inflammatory process or transverse myelitis in addition to her compressive myelopathy as of August 2014.

Petitioner was treated with antibiotics for an extended period of time for MRSA. Neurosurgical examination on October 14, 2014, revealed "paraplegia due to poorly controlled osteomyelitis." Pet. Ex. 3 at 7981. MRI on October 21, 2014 showed improvement of the osteomyelitis infection, but there continued to be "a destructive process" at the T5/T6 disc space "with associated endplate erosion and patchy marrow edema involving the adjacent vertebral bodies." Pet. Ex. 1 at 535. There also continued to be intramedullary T2 prolongation with associated cord expansion, but slightly improved (only to T9/T10 from T11/T12). The lumbar spine was also abnormal. Based on the medical records, MRI reports, and expert opinions, the undersigned finds that Petitioner had an extensive spinal cord injury with paraplegia, spinal stenosis, progressive and ongoing compressive myelopathy, and was worsened by a severe post-operative epidural infection with discitis and osteomyelitis and an infectious/inflammatory process, or transverse myelitis.

Although Petitioner underwent extensive PT from March to July 2015, and again in November and December 2015, she did not recover from her spinal cord injury. She was able to ambulate short distances with a rolling walker but was limited due to spasticity and weakness of

her lower extremities. PT records are replete with references to the significant challenges Petitioner experienced during therapy. In March 2015, Petitioner had lower extremity spasticity and weakness that limited her ability to transfer or ambulate, even for short distances. Standing fully erect caused her knees to buckle. These problems persisted throughout her therapy, although she persevered until she was able to walk with the rolling walker for short distances. She had back pain and increased spasticity in June and July 2015. In July 2015, she complained that her left knee felt like it was going to buckle, and she had increased spasticity. Petitioner took a break from PT from the end of July 2015 until November 2015. In November 2015, she reported thoracic spine pain, aching and burning, and bilateral lower extremity paresthesias. She continued to have spasticity. There were days she was unable to ambulate using her rolling walker. She described good and bad days. These problems continued into December 2015.

On December 3, 2015, she had difficulty walking due to increased tone and lack of control. One week later, on December 10 (one day before the flu vaccination at issue), she reported being unable to walk after the prior PT session. On December 18, 2015, she had worsening left extremity spasticity and was unable to stand. The following week, on December 29, 2015, she was noted to have an overall increase in spasticity making it difficult to transfer as independently. However, on that date, Petitioner was able to transfer, stand, and pivot, which she had not been able to do the prior session. Overall, she had a significant increase in tone. Petitioner was seen by Dr. Tramonte on December 30, and he noted that Petitioner's "worsening weakness, spasticity, and sensory loss . . . could be the natural evolution of thoracic cord compression with resolving infection/compression." Pet. Ex. 1 at 1251.

MRI on January 8, 2016, showed "interval development of expansile T2 prolongation/edema" of the thoracic cord from T1/T2 to T10 with "associated intramedullary syringohydromyelia at T6 andT7 without enhancement." Pet. Ex. 1 at 121. The syringomyelia was at the site of the previous spinal cord compression. There was no cord enhancement.

Dr. Garrett's assessment was "thoracic transverse myelitis due to either prior compression of spinal cord, meningitis, or even as autoimmune phenomenon from the flu vaccine. . . . Process [] [was] unclear to [him], but no surgical intervention [was] warranted. This process may be inflammatory and can resolve with time, possibly." Pet. Ex. 3 at 8250. Notably, although Dr. Garrett considered an autoimmune reaction to the flu vaccine in his list of differential causes, he did not order or perform a diagnostic lumbar puncture with CSF analysis to determine whether Petitioner had evidence of inflammation diagnostic of transverse myelitis. Moreover, he did not order treatment for transverse myelitis (steroids or immune mediating treatment).

After Dr. Tramonte reviewed Petitioner's January 8, 2016 MRI, and noted the "abnormal cord signal from T1 to T10" without enhancement, he concluded that he "suspect[ed] the changes [were] ischemic in nature." Pet. Ex. 1 at 1254.

In summary, after vaccination, neither Dr. Garrett nor Dr. Tramonte ordered a lumbar puncture to determine whether Petitioner had an aggravation of her myelitis or an inflammatory process. Neither ordered treatment for transverse myelitis. Dr. Tramonte suggested that

Petitioner's worsening could be a result of the natural evolution of her thoracic cord compression and concluded that it was ischemic in nature.

Dr. Tramonte had the benefit of evaluating Petitioner over her entire clinical course, from onset until post-vaccination; he conducted frequent evaluations and reviewed Petitioner's MRI studies over the course of her illness. Therefore, the undersigned finds the opinions of Dr. Tramonte to be persuasive. The opinions of Dr. Javed are consistent with those of Dr. Tramonte: that Petitioner's worsening was caused by the natural evolution of her underlying thoracic cord compression and that it was ischemic in nature. Since Dr. Javed's opinions are consistent with Petitioner's treating neurologist, the undersigned finds them to be more persuasive than those of Dr. Tornatore. Therefore, the undersigned finds that after vaccination, Petitioner experienced an evolution and progression of her thoracic cord compression. The worsening of her clinical condition and MRI findings were due to ischemia in the context of compressive myelopathy.

This finding is supported by the fact that in January 2016, Petitioner's MRI did not show enhancement of the dura or spinal cord. The only MRI that showed enhancement was done July 9, 2014, when Petitioner presented with very significant inflammation as evidenced by her extremely elevated white blood cell count in her CSF. While Dr. Javed explained that enhancement is not always evident on MRI in transverse myelitis, it usually is present.

Another fact that supports compressive myelopathy as the cause of Petitioner's worsening in December 2015/January 2016, is the presence of an intramedullary syringohydromyelia at T6 and T7. Dr. Javed explained that this abnormality develops due to subarachnoid space flow blockage of CSF, which occurs due to spinal stenosis. He cited Arai et al. which concluded that long-standing compression of the spinal cord "may be one of the main causes of syringomyelia." Resp. Ex. A4 at 1.

Further, Dr. Javed provided medical articles showing that the signs and symptoms of compressive myelopathy can mimic those of transverse myelitis, particular the MRI findings. Kelley et al. described five patients who were initially diagnosed with transverse myelitis due to MRI findings. One patient had longitudinally extensive involvement, like Petitioner here. All five of the patients described in Kelley et al. had progressive neurological dysfunction that did not improve until they had decompression of the spinal cord compression.

Lastly, the undersigned agrees with Dr. Javed that the evidence does not support a "fertile field" as contemplated by Dr. Torantore. The MRI done in January 2016 was not interpreted to show evidence of active or persistent infection. Further, in December 2015 and January 2016, Petitioner saw her PCP, neurosurgeon, and neurologist, and none of them documented any signs or symptoms consistent an active infection that would set the stage for the fertile field model. In fact, Dr. Garrett discontinued Petitioner's antibiotics after reviewing the MRI results and determining there was no need for her to continue taking them.

As described by Agmon-Levin et al., the proposed diagnostic criteria for transverse myelitis include evidence of an inflamed spinal cord (CSF pleocytosis), elevated IgG index, or MRI enhancement. See Pet. Ex. 18 at 1; see also Pet. Ex. 17 at 2 ("[M]ild pleocytosis[,] . . . positive oligoclonal bands[,] . . . and [] enhancement on MRI [] all point[] to an inflammatory

process."). Here, there is no evidence of any of these criteria. Petitioner did not have CSF drawn or analyzed and her MRI (in January 2016) did not show enhancement. Further, Agmon-Levin et al. provided that "exclusion of [] compressive etiology by neuroimaging should be observed." Pet. Ex. 18 at 1. Petitioner's MRI showed evidence of a long-standing compressive etiology. She had focal kyphosis at T5 and T6, along with the presence of the syringomyelia. Thus, Petitioner's MRI did not exclude evidence of a compressive pathology, and therefore, Petitioner did not meet the proposed criteria for the diagnosis of transverse myelitis.

Lastly, Petitioner's course was not monophasic. In transverse myelitis, the symptoms peak within "[four] hours and 21 days." Pet. Ex. 18 at 1. However, as demonstrated by Petitioner's 2018 MRI, her condition progressively worsened over time. Dr. Tornatore described Petitioner's 2018 MRI as the "worst" of her MRI studies, showing an increased signal abnormality from C2 to the bottom of her spinal cord. Tr. 285. He agreed that her illness was not monophasic but instead, that it was progressive. Moreover, Dr. Torantore agreed that the subsequent progression seen in the MRI of 2018 was not caused by vaccination.

Dr. Javed explained that Petitioner's 2018 showed worsening of her kyphosis at T5/T6 and expansion of the cord above and below the area. Based on the MRI results, he concluded that Petitioner did not have transverse myelitis, but that she had spinal stenosis and compressive myelopathy which had progressively worsened.

Regarding the medical records which state that Petitioner had an allergy to the flu vaccination, the undersigned does not find that those entries provide persuasive evidence of causation. Medical record documentation about allergies is often reported by patients, or documented by staff. See Tr. 213. An opinion by a treating physician that is not supported by a factual basis or other evidence is conclusory in nature. See Robertson v. Sec'y of Health & Hum. Servs., No. 18-554V, 2022 WL 17484980, at *17 (Fed. Cl. Spec. Mstr. Dec. 7, 2022) (finding treating physicians' statements of mere suspicion fall short of an opinion supporting vaccine causation); Cedillo v. Sec'y of Health & Hum. Servs., 617 F.3d 1328, 1347 (Fed. Cir. 2010) (concluding the special master did not err in affording little weight to the opinions of Petitioner's treating physicians where "none of the treating physicians concluded that the [] vaccine caused [Petitioner's] [condition]").

As for the records by subsequent treating physician Dr. Frohman beginning in late 2017, the undersigned does not find that they provide evidence that weighs in favor of a different result. Petitioner began seeing Dr. Frohman in October 2017, and he did not treat her before or after vaccination. After reviewing her history, Dr. Frohman suggested that Petitioner had transverse myelitis, but he did not opine that it was caused by the flu vaccination. Instead, he stated that the "pathoetiologic underpinning of this process remain enigmatic." Pet. Ex. 10 at 40; see also Pet. Ex. 10 at 31 (stating "the underlying etiology remains elusive"). Because Dr. Frohman did not have first-hand knowledge of Petitioner's clinical course (in contrast to Dr. Tramonte), and because he did not opine that Petitioner had transverse myelitis caused by the flu vaccine, the undersigned finds that the records of Dr. Frohman do not provide evidence as to vaccine causation.

In 2017, Petitioner also saw Dr. Okuda, another neurologist, who reviewed her history. His preliminary diagnosis was myelopathy. However, based on the information available to him, he did not reach any conclusions about the cause of Petitioner's myelopathy. At a second appointment, Dr. Okuda noted, "it appears an intramedullary process was present following her neurosurgical procedures with evolution over time." Pet. Ex. 7 at 27. He was unable to confirm this without reviewing Petitioner's MRI images, and it does not appear that he saw Petitioner after that visit. However, his preliminary opinion is consistent with Dr. Tramonte and Dr. Javed, that Petitioner had myelopathy, an intramedullary process, that evolved over time.

In conclusion, the weight of the evidence supports a finding that Petitioner had spinal stenosis and that she developed a post-operative infectious inflammatory myelitis while continuing to have a progressive compressive myelopathy. Her post-operative infection cleared, but her progressive compressive myelopathy did not improve. Her clinical course has evolved and worsened over time. There is not preponderant evidence that Petitioner's flu vaccination played a role in contributing to the worsening of her condition.

Therefore, the undersigned finds that Petitioner has failed to provide preponderant evidence of Loving factor five/Althen prong two that the flu vaccination significantly aggravated her pre-existing condition.

6. Loving Factor Six/Althen Prong Three: Proximate Temporal Relationship

The last element in the six-part Loving test has origins in Althen prong three. As stated in Loving, this element is "a showing of a proximate temporal relationship between vaccination and the significant aggravation." 86 Fed. Cl. at 144. Althen prong three requires Petitioner to establish a "proximate temporal relationship" between the vaccination and the injury alleged. Althen, 418 F.3d at 1281. A proximate temporal relationship has been equated to mean a "medically acceptable temporal relationship." Id. Petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disease's etiology, it is medically acceptable to infer causation-in-fact." de Bazan, 539 F.3d at 1352. The explanation for what is a medically acceptable time frame must also coincide with the theory of how the relevant vaccine can cause the injury alleged (under Althen prong one). Id.; Koehn v. Sec'y of Health & Hum. Servs., 773 F.3d 1239, 1243 (Fed. Cir. 2014); Shapiro, 101 Fed. Cl. at 542.

Based on the case law cited above, this factor/prong consists of two parts. Petitioner must first establish the time frame within which it is medically acceptable to infer causation. And secondly, she must show that the onset of the worsening or aggravation of her illness occurred during this time frame.

Petitioner received her flu vaccination on December 11, 2015. Dr. Tornatore opined that the Petitioner had a significant aggravation of her pre-existing transverse myelitis within one week of vaccination. However, when pressed, Dr. Tornatore agreed that there was "a hint" that something was happening before vaccination. Tr. 91.

Dr. Javed opined that there was an appropriate time period between vaccination and the onset of an autoimmune reaction, however, he disagreed that Petitioner had an autoimmune response to the flu vaccination. Further, he opined that Petitioner's condition was progressively worsening prior to vaccination.

The undersigned agrees that the records, especially the more detailed PT records show that Petitioner did not have a stable, improving course prior to vaccination. From 2014 to 2015, Petitioner made very slow progress despite her diligent efforts though therapy. While she was able to walk with a rolling walker, there were days where she could not ambulate due to spasticity, weakness, and fatigue. She had good days and bad days. She also had, as Dr. Tornatore observed, "a hint" that something was happening before vaccination. Tr. 91. She did have a worsening of her condition, but the worsening continued to progress, as evidenced by her 2018 MRI. Thus, while there was a temporal association between vaccination and her worsening, this worsening was not static. And the undersigned finds it was not caused by transverse myelitis, but by progressive compressive myelopathy.

Although the time frame on one week is an appropriate temporal association between a flu vaccination and the onset of an autoimmune response, the undersigned finds that Petitioner did not have an autoimmune response to her vaccination. Therefore, she has failed to prove an appropriate temporal association between vaccination and her significant aggravation of her underlying condition.

VII. CONCLUSION

Petitioner has suffered a devasting spinal cord injury. The undersigned extends her deepest sympathy. The undersigned's Decision, however, cannot be decided based upon sympathy, but rather on the evidence and law.

For all the reasons discussed above, the undersigned finds that Petitioner has failed to establish by preponderant evidence that the flu vaccination significantly aggravated her pre-existing condition. Therefore, Petitioner is not entitled to compensation and her petition must be dismissed.

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master