In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 17-547V

Filed: January 13, 2025

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MITCHELL VALENTINE,

Petitioner,

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,

Respondent.

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Joseph Vuckovich, Esq., Mctlaw, Washington, DC, for petitioner.
Eleanor Hanson, Esq., U.S. Department of Justice, Washington, DC, for respondent.

DECISION[1]

Roth, Special Master:

On April 18, 2017, a petition was filed on behalf of Mitchell Valentine ("Mr. Valentine" or "petitioner")—then a minor[2]—for compensation pursuant to the National Vaccine Injury Compensation Program.[3] Petitioner alleges that he received a Fluvirin influenza ("flu") vaccination on August 30, 2015 that caused him to develop transverse myelitis ("TM"). *See* Petition ("Pet."), ECF No. 1. Petitioner further alleged during the course of this matter that his TM caused him to develop lymphedema of the left leg. Respondent disputed petitioner's claim, contending that petitioner did not suffer from TM. The matter was argued through expert reports and legal memoranda.

[1] Because this Decision contains a reasoned explanation for the action taken in this case, it must be made publicly accessible and will be posted on the United States Court of Federal Claims' website, and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). This means the Decision will be available to anyone with access to the internet. In accordance with Vaccine Rule 18(b), the parties have 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. Any changes will appear in the document posted on the website.

[2] Once Mitchell Valentine reached the age of 18, the caption was amended to reflect him as the petitioner. ECF Nos. 71-72.

[3] National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755. Hereinafter, for ease of citation, all "§" references to the Vaccine Act will be to the pertinent subparagraph of 42 U.S.C. § 300aa (2018).

Following review of all the evidence and arguments presented, I find that petitioner has not provided preponderant evidence that the flu vaccine he received on August 30, 2015 caused him to suffer a compensable injury.

I. Procedural History

The petition was filed on April 18, 2017. Petition, ECF No. 1. Petitioner then filed medical records and affidavits. Petitioner's Exhibits ("Pet. Ex.") 1-12, ECF Nos. 7-8, 10, 13.

On December 22, 2017, respondent filed his Rule 4(c) Report, advising that the matter was not appropriate for compensation. ECF No. 17. Petitioner was ordered to file an expert report. Following several motions for extension of time and throughout the next three years, the parties exchanged expert reports and supporting literature. Pet. Ex. 14-53, ECF Nos. 18-22, 31, 34-35, 37-38, 45-47, 49, 58-64, 78-81; Respondent's Exhibits ("Resp. Ex.") A-J, ECF Nos. 39-43, 51-55, 66-70, 83-85.

Petitioner filed a status report on September 12, 2022, advising that the matter was appropriate for a ruling on entitlement and filed his Motion for Ruling on the Record ("Motion") on December 19, 2022. ECF No. 86; Motion, ECF No. 88. Respondent filed his Response to the Motion on May 11, 2023. Response, ECF No. 94. Petitioner filed a Reply on June 2, 2023. Reply, ECF No. 96.

Upon review of the record, issues with some of the evidence filed were found and the parties were ordered to correct the deficiencies. ECF Nos. 98, 101. Respondent refiled some of the medical literature relied on by his experts. Resp. Ex. F Tab 13; Resp. Ex. H Tabs 1-4; Resp. Ex. J Tabs 5-7. Petitioner also refiled medical literature. Pet. Ex. 39; Pet. Ex. 42.

I determined that the parties had a full and fair opportunity to present their cases and it was appropriate to resolve this issue without a hearing. The parties agreed. *See* Vaccine Rule 8(d); Vaccine Rule 3(b)(2); *Kreizenbeck v. Sec'y of Health & Human Servs*., 945 F.3d 1362, 1366 (Fed. Cir. 2020) (noting that "special masters must determine that the record is comprehensive and fully developed before ruling on the record."). Accordingly, this matter is now ripe for resolution.

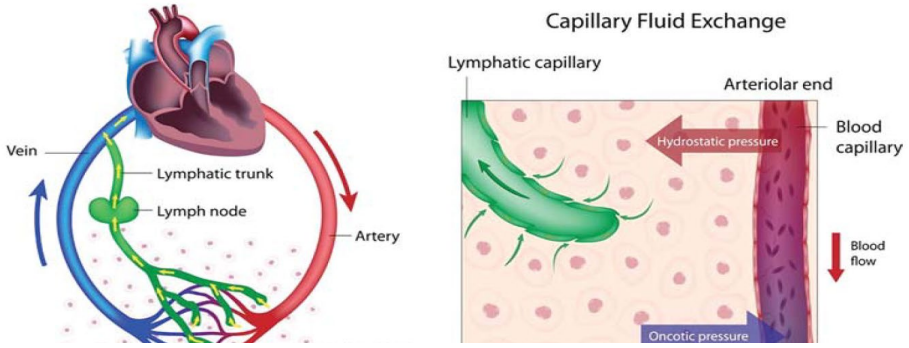
II. Medical Terminology

Transverse myelitis ("TM") is a rare acquired demyelinating disorder that presents with the sudden onset of neurological deficits due to spinal cord lesions. Annual incidence is between 1 and 8 per million with a significant proportion of cases preceded by infectious disease. Pet. Ex. 16 at 2.[4] TM is characterized by acute or subacute motor, sensory, and autonomic (bladder, bowel, and sexual) spinal cord dysfunction arising typically from an autoimmune phenomenon after infection or vaccination, with 15-30% of cases being characterized as idiopathic. Pet. Ex. 29 at 1.[5] The pathological hallmark of TM is the presence of a focal collection of lymphocytes and

[4] Roger Baxter et al., *Acute Demyelinating Events Following Vaccines: A Case-Centered Analysis*, 63 CLINICAL INFECTIOUS DISEASE 1456 (2016), filed as "Pet. Ex. 16".
[5] Elliot M. Frohman, M.D., Ph.D. & Dean M. Wingerchuk, M.D., *Transverse Myelitis*, 363 N. ENG. J. MED. 564

monocytes with varying degrees of demyelination, axonal injury, and astroglial and microglial activation within the spinal cord. *Id*. Symptoms of TM generally evolve over the course of hours to days and are usually bilateral but may be unilateral and asymmetric. *Id*. at 2. A well-defined truncal sensory level, below which the sensation of pain and temperature is changed or lost, distinguishes myelopathy from cerebral lesions and peripheral neuropathies. Urinary incontinence or retention, bowel incontinence or constipation, and sexual dysfunction are common but vary in severity in TM patients. The finding of one or more intrinsic cord lesions on MRI is characteristic of myelitis. *Id*. TM is a common manifestation of acquired demyelinating disease of the central nervous system. *Id*. at 4. "Normal MRI results should prompt a reconsideration of the diagnosis of myelopathy in favor of other disorders of the central or peripheral nervous system." *Id*.

The lymphatic system "is composed of lymphatic organs, such as lymph nodes, tonsils, thymus and the spleen", which are all connected by a network of lymphatic vessels that run parallel to the venous circulating system. Resp. Ex. F Tab 11 at 2.[6] The lymphatic system has three main functions: drainage of excess interstitial fluid, fat absorption, and immune surveillance. *Id*. The importance of the immune surveillance function is that circulating lymph transports various antigens and activated antigen-presenting cells into the lymph nodes to orchestrate the immune response. *Id*.



Resp. Ex. F Tab 11 at 2, Figure 1. This is a depiction of normal lymphatic circulation. The image on the left shows lymphatic drainage returning fluid to the bloodstream. The image on the right shows lymphatic capillaries collecting excess fluid from interstitial space, with about 90% of the fluid being filtered by the blood capillaries will be reabsorbed and returned to venous microcirculation. The remaining 10% of the fluid will be drained by the lymphatic capillaries. *Id*.

(2010), filed as "Pet. Ex. 29".

[6] Ayman A. Grada, MD & Tania J. Phillips, MD, *Lymphedema: Pathophysiology and Clinical Manifestations*, 77 J. American Academy of Dermatology 1009 (2017), filed as "Resp. Ex. F Tab 11".

Importantly, each limb has its own lymphatic system with a network of lymphatic vessels and lymph nodes that drain fluid from that specific area of the body.[7] Pet. Ex. 13 at 29-30 (Lymphedema in the leg is dysfunction of the lymphatic system in that leg. An artery delivers blood flow down the leg, it then goes into the smallest blood vessels where it drops of oxygen and nutrients and picks up waste that goes into the return vessel, like a French drain system. When there is excessive swelling in the limb it is a breakdown in that system.)

Lymphedema results from an imbalance between inflow and removal of interstitial fluid and protein or a malfunction of the lymphatic drainage system. Resp. Ex. F Tab 5 at 6.[8] It is a progressive disease defined as swelling of a part of the body caused by accumulation of protein-rich interstitial fluid secondary to a malformation, malfunction, obstruction, or impairment of the lymphatic drainage system. Pet. Ex. 44 at 1;[9] Resp. Ex. F Tab 5 at 1. Lower extremity lymphedema, which is usually associated with infection and malignancies, is much more common than upper extremity lymphedema, which is commonly associated with breast cancer. Resp. Ex. F Tab 11 at 3.[10]

Lymphedema is either primary (hereditary) or secondary (acquired). Resp. Ex. F Tab 11 at 3.[11] Primary lymphedema is divided into three categories: 1) congenital (at or shortly after birth), 2) lymphedema praecox (around puberty), and 3) lymphedema tarda (late in life). *Id*. at 3-4. Secondary lymphedema is much more common, and the result of damage caused by infection, trauma, surgery, or malignancy and malignancy-associated treatment. *Id*. at 3; Resp. Ex. F Tab 5 at 1, 6.[12]

The etiology of primary lymphedema is unknown. Resp. Ex. F Tab 5 at 5.[13] It develops in a delayed fashion over years or even decades. Not all patients who experience insult to the lymphatic system develop disease. Pet. Ex. 44 at 2.[14] "The natural history of primary lymphedema classically has been stated to be a slow, constant progression from a mild, painless swelling of an ankle to a huge, swollen extremity." Resp. Ex. F Tab 5 at 7.[15] Risk factors including radiation, infection, surgical dissection, and obesity have been identified, but are insufficient to reliably predict the development of lymphedema. Pet. Ex. 44 at 2.[16] Animal studies have provided insight into the cellular and molecular mechanisms of inflammation and

[7] For example, the fibular lymph node is a lymph node situated along the fibular artery. Fibular lymph node, *Dorland's Medical Dictionary Online*, https://www.dorlandsonline.com/dorland/definition?id=93278. Likewise, the distal deep inguinal lymph node is the most inferior of the deep inguinal lymph nodes, located just distal to the junction of the great saphenous and femoral veins. Distal deep inguinal lymph node, *Dorland's Medical Dictionary Online*, https://www.dorlandsonline.com/dorland/definition?id=93301. The mandibular lymph node is one of many facial lymph nodes located near the angle of the mandible. Mandibular lymph node, *Dorland's Medical Dictionary Online*, https://www.dorlandsonline.com/dorland/definition?id=93328.

[8] David M. Smeltzer, MD et al., *Primary Lymphedema in Children and Adolescents: A Follow-up Study and Review*, 76 PEDIATRICS 206 (1985), filed as "Resp. Ex. F Tab 5" and "Resp. Ex. G Tab 3".

[9] Catherine L. Ly et al., *Inflammatory Manifestations of Lymphedema*, 18 INT'L J. OF MOLECULAR SCIENCES 171 (2017), filed as "Pet. Ex. 44".

[10] Grada & Phillips, *supra* note 6.

[11] *Id*.

[12] Smeltzer et al., *supra* note 8.

[13] *Id*.

[14] Ly et al., *supra* note 9.

[15] Smeltzer et al., *supra* note 8.

[16] Ly et al., *supra* note 9.

changes in lymphedematous tissue. *Id*. Current evidence suggests that a variety of key players including T helper cells, T regulatory cells, macrophages, and dendritic cells play complex roles in the pathology of lymphedema by inflammatory response. *Id*. at 5-8. "The absence of lymphedema in some patients despite lymphatic injury and the delayed development in others indicate that secondary events are likely necessary to elicit these important pathologic interactions." *Id*. at 9. More studies are needed to understand how inflammatory changes regulate the development of lymphedema. *Id*.

Primary lymphedema is more frequent in children than secondary lymphedema with overall female predominance; however, males dominate congenital lymphedema while female onset is more frequently during adolescence with hormonal influence. Resp. Ex. J Tab 6 at 4.[17]

May-Thurner Syndrome is an anatomical variant where the right common iliac artery compresses the left common iliac vein and can result in iliofemoral deep venous thrombosis[18] ("DVT"). Pet. Ex. 33 at 1, 2;[19] Pet. Ex. 34 at 1.[20] This variant exists in 20% of the population and can exist without symptomatology. Pet. Ex. 33 at 1; Pet. Ex. 34 at 1. May-Thurner syndrome is observed in 50% of adult patients with descending iliofemoral DVT. Pet. Ex. 35 at 2.[21] It is very rare in children and usually associated with underlying sepsis, cancer, heart disease, immobilization, and medications such as oral contraception and asparaginase. *Id*. at 1-2.

III. Background

A. Petitioner's History Prior to the Subject Flu Vaccination

Petitioner had a normal childhood with uneventful childhood illnesses and seasonal allergies. He received all his vaccinations without event. *See generally* Pet. Ex. 3.

Petitioner received the subject seasonal flu vaccine at Walgreen's Pharmacy on August 30, 2015. Pet. Ex. 1; Pet. Ex. 3; Pet. Ex. 25 at 7-12 (noting in 2019 a "transverse myelitis-like reaction 3 weeks after flu shot 2012").

B. Petitioner's History Following the Subject Flu Vaccination

Twenty-two days later, on September 21, 2015, petitioner presented to the pediatrician reporting one day of left leg numbness. Pet. Ex. 3 at 5-6. He played soccer on Saturday and was stumbling on Sunday. *Id*. He had a stuffy nose but no fever. On examination, his right leg was

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[17] Caroline Colmant et al., *Pediatric Lymphedema: Study of 180 Patients Referred to a Tertiary Lymphedema Clinic*, 26 J. CUTANEOUS MEDICINE AND SURGERY 502 (2022), filed as "Resp. Ex. J Tab 6".

[18] Deep vein thrombosis is thrombosis of one or more deep veins, usually of the lower limb, characterized by swelling, warmth, and erythema. Deep vein thrombosis, *Dorland's Medical Dictionary Online*, https://www.dorlandsonline.com/dorland/definition?id=113602.

[19] Matthew Peters, MD et al., *May-Thurner Syndrome: A Not So Uncommon Cause of a Common Condition*, 25 PROC (BAYL UNIVERSITY MEDICAL CENTER) 231 (2012), filed as "Pet. Ex. 33".

[20] Melina R. Kibbe, MD et al., *Iliac Vein Compression in an Asymptomatic Patient Population*, 39 J. VASCULAR SURGERY 937 (2004), filed as "Pet. Ex. 34".

[21] Martin Olivieri, MD et al., *Ultrasound Assisted Endovascular Thrombolysis in Adolescents: 2 Case Reports*, 138 PEDIATRICS e20160022 (2016), filed as "Pet. Ex. 35".

normal, but his left leg had minimal strength. The pediatrician wrote "…suspect spinal lesion, ? Tumor vs. infection. Recent flu vaccine but [symptoms] do not fit GBS." *Id*. The pediatrician called a neurologist at Children's Hospital of Pittsburgh ("CHP") who agreed further evaluation was necessary. *Id*.

Petitioner was taken to CHP that day and admitted. His admission history included playing soccer on Saturday without issue and waking Sunday morning with tingling in his right lower extremity, numbness in his left lower extremity, and his left leg "not working" well. Symptoms continued throughout the day on Sunday with trouble walking because his knee would "buckle underneath" him. This morning he awoke with the same symptoms. Upon presentation he complained of shooting pain from his left calf to behind his knee that did not radiate up his leg. When he laid down, the weakness was more pronounced on the left. He had a flu vaccine three weeks ago and mild upper respiratory infection-like symptoms but no fever 2 weeks ago. He was otherwise healthy. Pet. Ex. 5 at 39.

Neurological exam revealed slightly decreased motor strength in the left lower extremity with normal strength in the right lower extremity. Pet. Ex. 5 at 40, 42. He had increased clonus[22] in the left ankle with upgoing toes on the left foot. *Id*. Although it was difficult to assess with consistency, he had diminished pinprick and light touch sensation in both feet. *Id*. at 41-42. His left leg was notably unstable when walking. *Id*. He had no bladder or bowel changes. *Id*. at 100. Primary concerns were cortical lesion, cord lesion, mass, or vascular malformation. *Id*. at 42.

Petitioner was hospitalized until September 25, 2015 and underwent extensive testing all of which was normal or negative. More specifically, MRI of the brain was normal. Pet. Ex. 5 at 24-25, 31, 139. MRI of the spine showed Schmorl's nodes[23] at T11-T12 and L1, disc narrowing and desiccation at T11-T12 and T12-L1 but was otherwise normal. *Id*. at 26, 137-38. EMG was normal with no evidence of large fiber peripheral neuropathy. *Id*. at 166-69. Cerebral spinal fluid ("CSF") studies were negative for Epstein Barr virus (EBV), cytomegalovirus (CMV), varicella virus (VZV), and enterovirus. White blood cell levels were within normal limits. *Id*. at 80, 150-154. "A presumptive diagnosis of early transverse myelitis was made based on his symptoms and his recent URI symptoms. He was treated with IV methylprednisolone [for] 5 days and was started on an oral prednisone taper…". *Id*. at 31. "His left leg weakness improved throughout admission but did not disappear completely" by the time of discharge. *Id*.

One month later, petitioner presented to his pediatrician for a yearly check-up and follow up for "transient myelitis." Pet. Ex. 3 at 3. His dad expressed concern that the flu vaccine was to blame. The pediatrician advised that it was an "unfortunate possibility that we may not know for sure what the true cause was." *Id*. She advised she would speak with petitioner's neurologist Dr. Alper about reporting the event to VAERS. *Id*. at 4. Petitioner's examination was normal on that date, and he was noted to be a well child with "[i]mproving transient myelitis." *Id*. at 3-4.

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[22] Clonus is a continuous rhythmic reflex tremor initiated by the spinal cord below an area of spinal cord injury, set in motion by reflex testing. Clonus, *Dorland's Medical Dictionary Online*, https://www.dorlandsonline.com/dorland/definition?id=10153&searchterm=clonus.

[23] Dr. McCormick described Schmorl's nodes as the lymph nodes that are located in the thoracic spine. It is defined as an irregular or hemispherical bone defect in the upper or lower margin of the body of the vertebra. Schmorl node, *Dorland's Medical Dictionary Online*, https://www.dorlandsonline.com/dorland/definition?id=93112.

Petitioner presented to Dr. Alper for a follow up visit on December 3, 2015. His parents reported that he was "completely fine" with no tingling, buckling, or strength complaints. He was playing soccer with no complaints. Pet. Ex. 5 at 422-23. Dr. Alper noted on examination that petitioner had some swelling of the left ankle with a few beats of clonus raising concern for arthritis. *Id*. at 422-24. Dr. Alper asked petitioner about the swelling and petitioner said the swelling may have been there since Monday after a family hunting trip. He also reported some heel pain after playing soccer in November. *Id*. at 423. He was sent to CHP for further evaluation of the ankle swelling. *Id*. at 424.

Petitioner presented that day to CHP for painless left ankle swelling. Pet. Ex. 5 at 44. Ten beats of clonus and asymmetric reflexes of the left leg were noted. Neurological consult was ordered. *Id*. Neurology exam showed no significant abnormalities and no further neurological testing needed. *Id*. at 45. X-ray showed some diffuse swelling of the left ankle and knee but no bone or joint abnormality. *Id*. at 105-07. A venous doppler conducted on the left leg was normal. *Id*. at 108.

Petitioner had no further medical visits until February 20, 2016 when he presented to CHP for left leg swelling. Pet. Ex. 5 at 10. The record documented that since he was last seen in December, he has had mild intermittent swelling around his left toes and ankle especially at the end of the day. Two weeks ago, his parents noticed increased swelling of his left leg. Two days ago, there was a marked increase of swelling involving his entire left leg up to the groin. His parents called Dr. Alper who recommended MRI of the spine. Petitioner reported left knee pain that day after playing in two soccer games. Petitioner reported the swelling was only of his left leg, never anywhere else and there was no numbness, weakness, tingling, or incontinence. *Id*. He was admitted with a differential of "obstructive malignancy including bone cancers vs lymphoma, testicular cancer, post infectious/viral/recurrence of transverse myelitis though w/out any overt neurological symptoms, autoimmune process, myxedema (though would expect more widespread)." *Id*. at 26. There was lesser concern for venous abnormality due to localization and lack of pitting edema. Ultrasounds done in December were normal. *Id*. The unilateral presentation made it less likely to be a systemic syndrome such as cardiac, renal, or hepatic abnormality. Systemic inflammatory arthritis was also being considered. *Id*.

Imaging studies for deep vein thrombosis were negative. Scrotal ultrasound was normal. Pet. Ex. 5 at 10, 13-15. An MRI of the pelvis showed left iliac vein compression by the right iliac artery. *Id*. at 10, 15. MRIs of the thoracic and lumbar spine were normal/negative with no evidence of transverse myelitis. *Id*. at 10, 127-28. Petitioner had 5/5 strength in all extremities, grossly intact sensation, and normal reflexes in the lower extremities with normal gait and balance throughout his admission and discharge. *Id*. at 10-11. The discharge diagnosis was swelling of the lower extremity. He was to follow up at the vascular clinic and wear compression stockings. *Id*. at 10-11, 13.

Petitioner presented to Dr. McEnaney at the UPMC Vein Center on February 26, 2016. Pet. Ex. 6 at 2. Examination revealed normal lower extremity motor strength and reflexes, but 1+ pitting edema of the left lower extremity. *Id*. at 3-4. Dr. McEnaney's impression was venous compression from the iliac artery (May Thurner syndrome) v. primary lymphedema.

Conservative treatment with compression therapy was recommended. He advised petitioner to remain active and continue playing soccer to assist in drainage. *Id*. at 4. A discussion about doing a lymphoscintigraphy[24] and "venogram to transduce a pressure across the segment of the vein in order to confirm diagnosis" was had in the event there was no improvement. *Id*.

On March 9, 2016, petitioner presented to the pediatrician with left knee pain since February 20, 2016. He thought it was from playing in multiple soccer games, but he now had increased leg swelling. Pet. Ex. 3 at 2. Examination of the knee was normal. The pediatrician questioned if the pain may be due to mechanical injury and made a referral to a sports medicine specialist/orthopedic. *Id*.

Petitioner presented to the orthopedist the next day and reported knee pain since playing in a soccer tournament on February 28, 2016.[25] Pet. Ex. 4 at 2. He reported being fine in the first game but had increasing pain in the second game then had to stop playing due to knee discomfort. He went to the ER for lower leg swelling and was discharged with compression stockings from thigh to toes. *Id*. at 2-3. His history included "left lower extremity swelling…initially began in September of 2015" with numbness and weakness diagnosed as TM and treated with steroids and PT. *Id*. at 3. Petitioner had diffuse swelling from left thigh to toes, including his left knee, though he had full range of motion of his left hip and knee. The impression was left "patellofemoral syndrome and Sinding-Larsen-Johansson syndrome[[26]], status post injury on February 20, 2016." *Id*. PT for stretching and strengthening was recommended along with a knee brace. He could participate in sports as tolerated. *Id*. He attended nine physical therapy sessions through April 2016 for juvenile osteochondrosis of the patella. *See generally* Pet. Ex. 7.

At his April 1, 2016 physical therapy visit, he was noted to be a 12 year old boy with a history of "myelitis (LLE numbness, tingling and weakness) 9/2015, increased LLE (lymphedema) 12/2015" who was seen in the neuroimmunology clinic. He had improving lymphedema and felt his outpatient physical therapy was helping. Pet. Ex. 10 at 6. Examination was within normal limits, but ankle clonus was still present. A physical therapist that specialized in lymphedema was suggested. *Id*. at 6-7.

That same day, petitioner returned to Dr. Alper for follow up. His neurological examination was normal, with subtle 1-2 clonus in the left foot. Pet. Ex. 10 at 14-15. Dr. Alper noted that she had treated petitioner since his hospitalization in September of 2015 and that the lower leg swelling/edema is possibly lymphedema without explanation and no underlying diagnosis. *Id*. at 15. She noted the possibility that his mild edema started in September, expressed initially as numbness; however, this would not explain the few beats of clonus or right-sided complaints at that time. The September examination showed most symptoms to be left-sided with left leg weakness, more proximal with knee buckling, so there could have been some soft

[24] Lymphoscintigraphy has been described as the "ideal imaging technique to evaluate lymphatic flow and confirm the diagnosis of lymphedema." Resp. Ex. J Tab 6 at 4.

[25] All other records refer to February 20, 2016 as the date his pain began, so this likely a typographical error.

[26] Sinding-Larsen-Johansson syndrome, also called Larsen disease, is traction apophysitis in the lower pole of the patella, an overuse injury seen most often in children between the ages of 10 and 16, characterized by pain and tenderness, often with inflammation. Larsen disease, *Dorland's Medical Dictionary Online*, https://www.dorlandsonline.com/dorland/definition?id=70515.

tissue problems causing those symptoms. But swelling was not noticed or diagnosed until December at his follow up and only involved his left lower leg and ankle. Dr. Alper referred petitioner to specialists in diagnostics. *Id*.

Petitioner returned to Dr. McEnaney on May 20, 2016 with persistent and somewhat worsened lower left extremity swelling. Pet. Ex. 6 at 6. He reported that the swelling was significantly worse when he did not wear the compression stocking. He saw an orthopedist for his knee pain and tenderness. Examination revealed swelling from the hip to the toes of the left leg. Lymphoscintigraphy was recommended. He was to wear the compression stocking. *Id*.

Dr. McEnaney discussed the results of the lymphoscintigraphy with petitioner and his parents at a June 3, 2016 visit, advising that the testing demonstrated a lack of radiotracer transit in the left lower extremity consistent with obstructed lymphatic flow, reinforcing the diagnosis of lymphedema. Pet. Ex. 6 at 7, 8-9. Aggressive manual lymphatic drainage was anticipated. *Id*.

On September 22, 2016, petitioner presented to Dr. McCormick for a second opinion on the diagnosis of lymphedema. Dr. McCormick noted a history of hospitalization in the fall of 2015 for left leg weakness, tingling, and numbness. Despite negative imaging and normal CSF, TM was thought to be the best diagnosis. Petitioner received 5 days of high dose steroids with a 6-week oral taper, got better very quickly, and remained neurologically asymptomatic. Pet. Ex. 8 at 19. In December of 2015, he had left ankle and foot swelling but no other symptoms. The swelling then became acutely worse in February of 2016 with the swelling up to the groin. He was admitted to the hospital and diagnosed with May-Thurner syndrome. After discharge, petitioner saw a vascular specialist who diagnosed petitioner with congenital lymphedema not May-Thurner syndrome. *Id*. at 19-20. Lymphoscintigraphy testing was consistent with lymphedema. *Id*. Dr. McCormick recommended an MRI/A of the lower left extremity with vascular anomaly protocol to evaluate for underlying lymphatic malformation. *Id*. at 22. He noted a concern that the underlying lymphatic malformation "accelerated after puberty/steroid treatment vs. May-Thurner syndrome developing after steroid with loss of fat pad between iliac venous and arterial system." *Id*. The MRI performed on October 18, 2016 revealed diffuse left lower leg edema consistent with history of lymphedema and no lymphatic or vascular malformation. *Id*. at 27-28. Lower extremity arteries and veins were patent. There was a nonspecific finding of partial bone marrow edema. *Id*. at 28.

Petitioner underwent unrelated bilateral hydrocele surgery in December of 2019 without event and was treated for a pilonidal cyst in 2022. *See* Pet. Ex. 46. He was otherwise a healthy teenager who continued to play soccer. Pet. Ex. 50.

C. Affidavits by Petitioner's Father, Kary Valentine[27]

Kary Valentine is petitioner's father. In his initial affidavit, he affirmed petitioner's receipt of a flu vaccine as set forth in the Vaccine Injury Table, which was administered in the U.S. and caused transverse myelitis and sequelae in excess of six months. Pet. Ex. 9.

[27] Mr. Kary Valentine was initially the named petitioner in this matter until Mitchell Valentine reached the age of majority. *See* Pet.

Mr. Valentine submitted another affidavit five years later dated December 15, 2022. Pet. Ex. 54. In his second affidavit, Mr. Valentine affirmed that petitioner was healthy and did not have any abnormal swelling of his left leg or elsewhere prior to his August 30, 2015 vaccination. *Id*. at 1. Mr. Valentine further affirmed that petitioner showed no signs of puberty in the year before or after the subject flu vaccine. *Id*.

Mr. Valentine noticed something was not right with petitioner's left foot and ankle in the fall of 2015. Pet. Ex. 54 at 1. "By January or February of 2016, it was evident that the lymphedema has progressed and now encompassed his entire left leg." *Id*. at 1-2.

According to Mr. Valentine, he and his wife sought medical treatment for petitioner for "his neurological symptoms" very quickly. The steroids he was given were very effective in controlling his symptoms. Mr. Valentine was convinced that petitioner's TM would have progressed if not for the steroids. Pet. Ex. 54 at 2.

Mr. Valentine affirmed that he noticed petitioner's left ankle and foot swelling around the time he was losing the weight gained from the steroid treatment. As his parent and "caregiver who observed him every day, it seems almost certain that the lymphedema which followed the TM was caused by it." Pet. Ex. 54 at 2. Prior to the vaccine, he had no signs of lymphedema or other health conditions. *Id*.

D. Deposition Testimony of Andrew McCormick, M.D.

Dr. McCormick was deposed on November 27, 2018. Pet. Ex. 13.

Dr. McCormick[28] is a pediatrician with a specialty in pediatric hospital medicine and a subspecialty in children with vascular anomalies and Down Syndrome. Pet. Ex. 13 at 6-7. He described himself as the pediatrician for children admitted to the hospital for everything from asthma to complex medical needs. *Id*. at 7.

Dr. McCormick described vascular anomalies as a "unique field of medicine" that is multidisciplinary because the patient will need more than one subspecialist to assist in their care. Pet. Ex. 13 at 10-11. Dr. McCormick does the diagnostic evaluation at admission and then decides who the patient needs as a subspecialist going forward. *Id*. at 11.

Dr. McCormick explained that vascular anomalies are divided into vascular tumors and vascular malformations with vascular tumors being the most common and treatable requiring only management because they usually resolve over time. Pet. Ex. 13 at 11, 13. Vascular

[28] Dr. McCormick graduated from University of Pittsburgh Medical School, did his residency at University of Pittsburgh Medical Center ("UPMC"), and has been a hospitalist at the Paul C. Gaffney Hospital Medicine Division for eight years. He has worked in the Vascular Anomaly Center at UPMC for the last five years and has been the medical director for the past three years. Pet. Ex. 13 at 8. He is an associate professor of pediatrics through UPMC and faculty clinician at the Paul C. Gaffney Pediatric Hospital Medicine Division. *Id*. at 9. He said 75% of his time is spent as a clinician and 25% as medical school faculty. He does research on the side. *Id*. Dr. McCormick teaches the residents and medical students in the hospital. Outside of the medical system, he teaches both vascular anomalies and Down Syndrome care. *Id*. at 10. He teaches an advanced physical examination course for first- and second-year medical students on how to take histories from the family and complete physical exams. *Id*.

malformations are congenital abnormalities, typically lifelong, and require multidisciplinary treatment. *Id*. at 11-13. Dr. McCormick stated that data shows "vascular malformations accelerate during puberty and during pregnancy . . .". *Id*. at 42. He said that clinicians "see lymphedema show up in teenagers all the time because of" puberty. *Id*. There are also arterial and venous system malformations purposefully created for dialysis, those that are traumatically induced, or are due to error in correction when the injury occurs. *Id*. at 14-15.

Dr. McCormick saw petitioner once on September 22, 2016. He was questioned at length about his record for that visit. He explained that the first portion of his office record contains the history provided by the parents including the hospitalization and the treatment provided to understand what may have caused the lymphedema. Pet. Ex. 13 at 15-18; Pet. Ex. 8. His "write up of the case" included his review of the medical records prior to meeting the family, confirmation of the history with the family, and his thoughts about what was occurring with the patient. *Id*. at 19, 55-56. He included Dr. Alpers's findings because petitioner's hospitalization in September of 2015 and follow up visits were not for lymphedema; rather, the lymphedema was a new finding and was why he was evaluating petitioner. *Id*. at 21-22.

He discussed the facts that informed his thinking about petitioner's case, including petitioner's presentation of only one swollen leg, no joint swelling, no fever, no rash, no shortness of breath, no loss of appetite, etc. The general screening questions are designed to ascertain whether the unilateral leg swelling was part of a larger process like a systemic illness, a rheumatological disorder, or an oncologic disorder for example. Pet. Ex. 13 at 22-23. Petitioner's presentation, in combination with earlier testing, effectively ruled out those other conditions. *Id*. at 23-24. He included the MRI results as well and explained that Schmorl's nodes are lymph nodes seen at T11, T12, and L1 and were non-enhancing and not inflamed, indicating that the neurological disease petitioner presented with earlier had since resolved and was thus not the etiology of his leg swelling.[29] *Id*. at 24-25. He further explained the significance of seeing the Schmorl's nodes on petitioner's MRI, stating that they could have been enlarged for a "run-of-the-mill virus", bacterial infection, or even cancer but that their enlargement alone "doesn't necessarily tell you anything other than we know that they've been stable over time…". *Id*. at 26-27. He added that the enlarged Schmorl's nodes were likely not connected to the swelling of petitioner's left leg, explaining that unilateral extremity swelling is an abnormality of the lymphatic tract like a unilateral malformation or an obstruction of flow to one side. A central channel would not cause obstruction to an entire leg. *Id*. at 27-28. He agreed that DVT was ruled out before he saw petitioner. *Id*.

Dr. McCormick explained that "left iliac vein compression by the right iliac artery" means that there is a blockage of the fluid draining into the veins. Pet. Ex. 13 at 28-30. He explained that each leg has arteries and veins that run next to each other. An artery delivers blood down the leg and a vein drains fluid and waste product. If there is compression, the return through the veins is blocked and the system becomes inefficient which may cause excessive swelling in a limb or lymphedema. *Id*. at 29-30.

[29] Dr. McCormick stated that if the nodes were enhancing, that would indicate a change in blood flow which could be related to an inflammatory or oncologic process. Pet. Ex. 13 at 26. Enlarged lymph nodes, on the other hand, may be present for several reasons, including upper respiratory infection, a virus, bacterial infection, or lymphoma in the location. Enlargement alone cannot indicate etiology and can exist for a long time after infection. *Id*. at 26-27.

Dr. McCormick explained that May-Thurner syndrome is the compression of the outflow system because of either lymphedema or a blood clot. Pet. Ex. 13 at 30, 32. May-Thurner syndrome can either be congenital or can develop during life. *Id*. at 33. For those that develop later in life, there is a fat pad that exists between the two systems that can be lost in childhood, leading to the compression of venous outflow. *Id*. at 31, 33. High-dose steroids can also redistribute fat, causing someone to lose some protective fat pads. *Id*. at 40.

Petitioner's testing showed both a venous outflow obstruction consistent with May-Thurner syndrome and dysfunction in the lymphatic system. Pet. Ex. 13 at 33-34. It is possible for a person to have both May-Thurner syndrome and a dysfunctional lymphatic system, both occurring together but the two are not necessarily connected. *Id*. at 34-35.

Dr. McCormick's "Impression" included his concern for possible underlying lymphatic malformation of the left lower leg present all of petitioner's life that may have changed over time due to age and the pressures of puberty. Pet. Ex. 13 at 36-37. He stated that May-Thurner syndrome and the loss of fat pad can develop from an illness, but there was insufficient information to make that determination. *Id*. at 36-38. His thought was not May-Thurner syndrome because of the lack of varicosities, so he ordered an MRA to look at petitioner's vasculature system. *Id*. at 38-39, 43. Petitioner's MRA was normal, apart from diffuse swelling suggestive of lymphedema, with no congenital malformation of the major arteries, veins, or lymphatic tissue to cause it, which ruled out both May-Thurner syndrome and congenital lymphatic malformation. *Id*. at 44-45. Rather, "this child fits the category of primary lymphedema." *Id*. at 45.

A classic picture for primary lymphedema in teenagers is praecox lymphedema which is puberty driven. Pet. Ex. 13 at 41-42, 46, 48. However, the mechanism for how the production of steroids in puberty (and in pregnancy) can accelerate an underlying lymphatic malformation is unknown and is only an association. *Id*. Therefore, it is referred to as idiopathic requiring system management with compression garments and lymphedema therapy. There is no medication to prescribe. *Id*. at 45-46. Dr. McCormick thought that high-dose steroids can have the same effect on the body as puberty, which itself is driven by steroids in the form of estrogen and testosterone but was unaware of any case reports pointing to high dose steroids as a cause of primary lymphedema. *Id*. at 48-49. Thus, while puberty is a well-defined disease process for praecox lymphedema, there is little data to suggest that external steroid use could cause progression or changes in lymphatic disease. *Id*.

Dr. McCormick opined that petitioner had primary lymphedema with abnormalities in the peripheral lymphatic drainage system seen on imaging. It would be hard to know the etiology unless you saw what the trigger was. Pet. Ex. 13 at 58-60. Dr. Yilmaz, an interventional radiologist with specialized training in reading vascular anomalies, saw petitioner after Dr. McCormick and had the same impression after reading the test results. *Id*. at 54-55, 58-59.

When asked what the timeframe for onset would be between steroids (either through puberty or medication) and clinically significant lymphedema, Dr. McCormick relied on the

onset of praecox lymphedema in puberty in girls to say that it would be weeks or months, not years. Pet. Ex. 13 at 60-62.

E. Expert Reports

Petitioner submitted expert reports from Dr. Allan Rubenstein and Dr. Tejas Shah. Pet. Ex. 14; Pet. Ex. 27; Pet. Ex. 30; Pet. Ex. 31; Pet. Ex. 47. Respondent submitted reports from Dr. Michael Sweeney and Dr. Caitlin Hicks. Resp. Ex. A; Resp. Ex. C; Resp. Ex. D; Resp. Ex. G; Resp. Ex. H; Resp. Ex. I; Resp. Ex. J. All expert reports were considered and are referenced and discussed in the analysis of this Ruling.

IV. Standard for Adjudication

A petitioner is required to establish his case by a preponderance of the evidence. 42 U.S.C. § 300aa-13(1)(a). The preponderance of the evidence standard requires a "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [they] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly v. Sec'y of Health & Human Servs.*, 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010) (citations omitted). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Human Servs.,* 931 F.2d 867, 873 (Fed. Cir. 1991).

Distinguishing between "preponderant evidence" and "medical certainty" is important because a special master should not impose an evidentiary burden that is too high. *Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1379-80 (Fed. Cir. 2009) (reversing a special master's decision that petitioners were not entitled to compensation); *see also Lampe v. Sec'y of Health & Human Servs.*, 219 F.3d 1357 (Fed. Cir. 2000); *Hodges v. Sec'y of Health & Human Servs.*, 9 F.3d 958, 961 (Fed. Cir. 1993) (disagreeing with the dissenting judge's contention that the special master confused preponderance of the evidence with medical certainty).

A. Legal Standard

The Vaccine Act provides two avenues for petitioners to receive compensation. First, a petitioner may demonstrate a "Table" injury—i.e., an injury listed on the Vaccine Injury Table that occurred within the provided time period. 42 U.S.C. § 300aa-11(c)(1)(C)(i). "In such a case, causation is presumed." *Capizzano v. Sec'y of Health & Human Servs*., 440 F.3d 1317, 1320 (Fed. Cir. 2006); *see* § 13(a)(1)(B). Second, where the alleged injury is not listed on the Vaccine Injury Table, a petitioner may demonstrate an "off-Table" injury, which requires that the petitioner "prove by a preponderance of the evidence that the vaccine at issue caused the injury." *Capizzano*, 440 F.3d at 1320; *see* § 11(c)(1)(C)(ii); *see also Wright v. Sec'y of Health & Human Servs*., 22 F.4th 999, 1006 (Fed. Cir. 2022) (defining the term "residual effects" in the Act, as "detrimental conditions within the patient, such as lingering or recurring signs and symptoms" of the alleged vaccine injury, which are compensable). A petitioner need not show that the vaccination was the sole cause, or even the predominant cause, of the alleged injury; showing that the vaccination was a "substantial factor" and a "but for" cause of the injury is sufficient for recovery. *Pafford v. Sec'y of Health & Human Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006); *Shyface v. Sec'y of Health & Human Servs*., 165 F.3d 1344, 1352 (Fed. Cir. 1999). Petitioners

are not required "to eliminate alternative causes as part of establishing [their] prima facie case." *Doe v. Sec'y of Health & Human Servs*., 601 F.3d 1349, 1357-58 (Fed. Cir. 2010); *see Walther v. Sec'y of Health & Human Servs*., 485 F.3d 1146, 1152 (Fed. Cir. 2007) (holding that a "petitioner does not bear the burden of eliminating alternative independent potential causes"). Once a petitioner has proven causation by preponderant evidence, "the burden then shifts to the respondent to show by a preponderance of the evidence that the injury is due to factors unrelated to the administration of the vaccine." *Deribeaux ex rel. Deribeaux v. Sec'y of Health & Human Servs*., 717 F.3d 1363, 1367 (Fed. Cir. 2013) (citing 42 U.S.C. § 300aa-13(a)(1)(B)).

To prove causation, a petitioner must satisfy the three-pronged test established in *Althen v. Sec'y of Health & Human Servs*., 418 F.3d 1274 (Fed. Cir. 2005). *Althen* requires that a petitioner show by preponderant evidence that a vaccination they received caused their injury "by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Id*. at 1278. Together, these prongs must show "that the vaccine was 'not only a but-for cause of the injury but also a substantial factor in bringing about the injury.'" *Stone v. Sec'y of Health & Human Servs*., 676 F.3d 1373, 1379 (Fed. Cir. 2012) (quoting *Shyface*, 165 F.3d at 1352-53). Causation is determined on a case-by-case basis, with "no hard and fast per se scientific or medical rules." *Knudsen v. Sec'y of Health & Human Servs*., 35 F.3d 543, 548 (Fed. Cir. 1994). Petitioners are not required to identify "specific biological mechanisms" to establish causation, nor are they required to present "epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities." *Capizzano*, 440 F.3d at 1325 (quoting *Althen*, 418 F.3d at 1280). "[C]lose calls regarding causation are resolved in favor of injured claimants." *Althen*, 418 F.3d at 1280.

Each *Althen* prong requires a different showing. Under the first prong, a petitioner must provide a "reputable medical theory" demonstrating that the vaccine received can cause the type of injury alleged. *Pafford*, 451 F.3d at 1355-56 (citation omitted). To satisfy this prong, a petitioner's "theory of causation must be supported by a 'reputable medical or scientific explanation.'" *Andreu*, 569 F.3d at 1379 (quoting *Althen*, 418 F.3d at 1278). This theory need only be "legally probable, not medically or scientifically certain." *Id*. at 1380 (emphasis omitted) (quoting *Knudsen*, 35 F.3d at 548). Nevertheless, "petitioners [must] proffer trustworthy testimony from experts who can find support for their theories in medical literature." *LaLonde*, 746 F.3d at 1341.

The second *Althen* prong requires proof of a "logical sequence of cause and effect." *Capizzano*, 440 F.3d at 1326 (quoting *Althen*, 418 F.3d at 1278). Even if the vaccination can cause the injury, a petitioner must show "that it did so in [this] particular case." *Hodges v. Sec'y of Health & Human Servs*., 9 F.3d 958, 962 n.4 (Fed. Cir. 1993) (citation omitted). "A reputable medical or scientific explanation must support this logical sequence of cause and effect," *Id*. at 961 (citation omitted), and "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," *Paluck v. Sec'y of Health & Human Servs*., 786 F.3d 1373, 1385 (Fed. Cir. 2015) (quoting *Andreu*, 569 F.3d at 1375).

The third *Althen* prong requires that a petitioner establish a "proximate temporal relationship" between the vaccination and the alleged injury. *Althen*, 418 F.3d at 1281. This "requires preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *De Bazan v. Sec'y of Health & Human Servs*., 539 F.3d 1347, 1352 (Fed. Cir. 2008). Typically, "a petitioner's failure to satisfy the proximate temporal relationship prong is due to the fact that onset was too late after the administration of a vaccine for the vaccine to be the cause." *Id*. However, "cases in which onset is too soon" also fail this prong; "in either case, the temporal relationship is not such that it is medically acceptable to conclude that the vaccination and the injury are causally linked." *Id*.; *see also Locane v. Sec'y of Health & Human Servs*., 685 F.3d 1375, 1381 (Fed. Cir. 2012) ("[If] the illness was present before the vaccine was administered, logically, the vaccine could not have caused the illness.").

Finally, although this decision discusses some but not all the literature in detail, I have reviewed and considered all of the medical records and literature submitted in this matter. *See Moriarty ex rel. Moriarty v. Sec'y of Health & Human Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though [s]he does not explicitly reference such evidence in h[er] decision."); *Simanski v. Sec'y of Health & Human Servs.*, 115 Fed. Cl. 407, 436 (2014) ("[A] Special Master is 'not required to discuss every piece of evidence or testimony in her decision.'" (citation omitted)), *aff'd*, 601 F. App'x 982 (Fed. Cir. 2015).

B. Law Regarding Diagnosis

In *Broekelschen v. Sec'y of Health and Human Servs.,* 618 F.3d 1339, 1346 (Fed. Cir. 2010), the Federal Circuit recognized that in some circumstances, the special master may "first determine which injury was best supported by the evidence in the record before applying the *Althen* test." This principle also means that a petitioner must establish that the vaccinee suffers the injury allegedly linked to the vaccination. *Lombardi v. Sec'y of Health & Human Servs.,* 656 F.3d 1343, 1353-54 (Fed. Cir. 2011).

V. Discussion

The outcome of petitioner's case is based on three different findings. First, petitioner must establish that TM was the appropriate diagnosis. Second, he must present persuasive evidence that the flu vaccine was the cause in fact of his TM. Finally, if he proves that TM was the appropriate diagnosis and that the flu vaccine was the cause in fact of his TM, he must show that his TM caused lymphedema in his left lower extremity.

A. Diagnosis

Petitioner alleges that he suffered from TM as a result of the flu vaccine he received on August 30, 2015. Respondent disagrees that TM was the appropriate diagnosis. This dispute involves several issues including the value of published diagnostic criteria, how the pieces of evidence preponderate, and the legal significance of petitioner's lack of success in proving TM.

a. Diagnostic Criteria for TM

The diagnostic criteria for TM includes: 1. Bilateral, not necessarily symmetric, sensorimotor and autonomic spinal cord dysfunction; 2. Clearly defined sensory level; 3. Progression to nadir of clinical deficits between 4 hours and 21 days after symptom onset; 4. Demonstration of spinal cord inflammation through cerebrospinal fluid pleocytosis, elevated IgG index, or MRI revealing a gadolinium-enhancing cord lesion; and 5. Exclusion of compressive, post-radiation, neoplastic, and vascular causes. Pet. Ex. 28 at 6, Table 4;[30] Pet. Ex. 29 at 2, Table 1;[31] Resp. Ex. A Tab 3.[32]

Spinal cord disorders are generally "classified as 'syndromes' due to typical signs and symptoms produced because of the location of the lesion and specific tract involvement." Pet. Ex. 28 at 1-2.[33] Transverse myelitis specifically typically arises as an autoimmune phenomenon after infection, vaccination, systemic autoimmune disease, or acquired demyelinating disease; anywhere from 15% to 30% are classified as idiopathic. Pet. Ex. 29 at 1;[34] Pet. Ex. 28 at 9. Although idiopathic TM can arise at any age, the peak incidence is between 10 and 19 years of age and 30 and 39. Pet. Ex. 29 at 1.

b. Evidence regarding petitioner's health

Petitioner developed tingling in his right lower leg, with numbness of his left lower leg and his left leg "not working" three weeks after his receipt of the flu vaccine, two weeks after mild cold symptoms without fever and following a competitive day of soccer. Pet. Ex. 3 at 5; Pet. Ex. 5 at 39. Neurological exam revealed slightly decreased motor strength in the left lower extremity with normal strength in the right lower extremity. Pet. Ex. 5 at 40, 42. He had diminished pinprick and light touch sensation in both feet. *Id*. at 41-42. His left leg was notably unstable when walking. *Id*. Primary concerns were cortical lesion, cord lesion, mass, or vascular malformation. *Id*. at 42.

Extensive objective testing including MRIs of the brain and spine were normal (other than unrelated Schmorl's nodes on MRI of the spine); a lumbar puncture (performed three days after onset) was normal, cultures for EBV, CMV, VZV, and enterovirus were negative, and EMG testing on September 25, 2015 was normal. Pet. Ex. 5 at 24-26, 31, 80, 137-39, 150-54, 166-69. Rheumatology and endocrine workups were normal. *Id*. at 35.

Despite all normal testing, "[a] presumptive diagnosis of early transverse myelitis was made based on his symptoms and his recent URI symptoms." Pet. Ex. 5 at 31. He received 5 days of IV methylprednisolone followed by oral prednisone taper on discharge beginning on September 26 and ending on November 4, 2015 with symptom improvement. *Id*. at 31, 34-35. At

[30] Anu Jacob, M.D. & Brian G. Weinshenker, M.D., F.R.C.P.(C.), *An Approach to the Diagnosis of Acute Transverse Myelitis*, 28 SEMINAL NEUROLOGY 105 (2008), filed as "Pet. Ex. 28".
[31] Frohman & Wingerchuk, *supra* note 5.
[32] Rohit Bakshi, MD & John C. Mazziotta, MD, PhD, *Acute Transverse Myelitis After Influenza Vaccination: Magnetic Resonance Imaging Findings*, 6.4 J. OF NEUROIMAGING 248 (1996), filed as "Resp. Ex. A Tab 3".
[33] Jacob & Weinshenker, *supra* note 30.
[34] Frohman & Wingerchuk, *supra* note 5.

the time of discharge, he had a normal physical examination with 4+/5 motor strength in the proximal lower left leg muscles and 5-/5 on distal. He had normal bulk and tone and 5/5 strength in the bilateral upper extremities and right lower extremity. *Id*. at 36. Reflexes were normal, and sensory was intact to light touch, temperature, pinprick, and proprioception with no sensory level. *Id*. at 36, 40. He had 7-8 beats of clonus on the left, equivocal toe on the left, and flexor plantar response on the right. He had no ataxia or dysmetria, normal rapid alternating movements, and mild steppage gait with the left foot but able to hop on the left leg with some difficulty. *Id*. at 36. The discharge diagnosis was "[w]eakness of left lower extremity." *Id*.

A month later he was noted to be a well child with "[i]mproving transient myelitis." Pet. Ex. 3 at 3-4.

His parents reported that he was "completely fine" on December 3, 2015, and Dr. Alper noted there was no tingling, buckling, or strength complaints and he was playing soccer without issue. Pet. Ex. 5 at 422-23. However, she noted left ankle swelling and a few beats of clonus. *Id*. 422-24. Petitioner was sent to CHP and underwent further testing, which showed no abnormalities. *Id*. at 105-08.

Petitioner was hospitalized again on February 20, 2016 for left leg swelling. Pet. Ex. 5 at 10. Follow up MRIs of the thoracic and lumbar spine performed during his hospitalization were normal. *Id*. at 10, 127-28. His September MRIs were referred to as having provided no explanation for his symptoms. *Id*. at 10. He had 5/5 strength in all extremities, grossly intact sensation, and normal reflexes in the lower extremities with normal gait and balance on that day. *Id*. at 10-11.

c. Dr. Alper's Opinions

The opinions of treating doctors can be quite probative. *Cappizano v. Sec'y of Health & Human Servs.,* 440 F.3d 1317, 1326 (Fed. Cir. 2006). The views of treating doctors about the appropriate diagnosis are often persuasive because the doctors have direct experience with the patient whom they are diagnosing. *See McCulloch v. Sec'y of Health & Human Servs.,* No. 09-293V, 2015 WL 3640610, at *20 (Fed. Cl. Spec. Mstr. May 22, 2015). However, the views of a treating doctor are not absolute, *Snyder v. Sec'y of Health & Human Servs.,* 88 Fed. Cl. 706, 745 n.67 (2009), even on the question of diagnosis, *R.V. v. Sec'y of Health & Human Servs.,* 127 Fed. Cl. 136, 141 (2016), *appeal dismissed*, No. 16-2400 (Fed. Cir. Oct. 26. 2016).

In her February 2016 visit notes, Dr. Alper noted petitioner's history including September of 2015 when he presented with lower left leg weakness with sensory changes but normal results on testing for Lyme disease, NMO-IgG antibody, MRI of brain and spine, CSF, and EMG. Pet. Ex. 10 at 25. There was no evidence of radiculopathies or neuropathies and no evidence of GBS. He was treated with IV steroids then oral steroids and did well. *Id*. Two months later, he was "completely fine", playing soccer, and slimming down from the steroid weight gain. *Id*. at 25-26. At his December 2015 visit, there was some left ankle swelling with no pain and a few beats of clonus. *Id*. at 26. Dr. Alper wrote that based on his symptoms in September, myelitis could not be ruled out even though all studies were negative, so he was treated for myelitis. However, today, the swollen ankle is concerning, and she questioned if the Lyme testing may have been a

false negative. "He could have had myeloradiculitis before but normal CSF would be unusual" or arthritis. An evaluation at the ER at CHP and repeat Lyme testing was ordered. *Id*. at 27.

Petitioner was seen at CHP with no neurological abnormalities noted. X-rays showed some swelling of the ankle and knee but no bone or joint abnormality. Pet. Ex. 5 at 44, 45, 105-07. A venous doppler was normal. *Id*. at 108.

At his return visit on April 1, 2016, Dr. Alper noted no neurological concerns, but continued left ankle swelling. Pet. Ex. 10 at 14. "Because his diagnosis of myelitis in September was [a] presumed diagnosis, although his symptoms improved at that time, the CSF findings and MRI findings were negative." *Id*. MRI did not show a tumor or any paraspinal mass that would cause both symptoms. Vascular studies showed a vascular malformation, and MRI of the abdominal pelvis showed left iliac vein compression suggestive of May-Thurner syndrome diagnosed by both vascular and radiology. According to the mother, the specialist thought it was an incidental finding that did not explain the left leg swelling. *Id*. Dr. Alper wrote it was "very interesting" that petitioner had lower left extremity edema, possibly lymphedema with no explanation for it. *Id*. at 15. "One possibility in September maybe (sic) he had had mild edema just starting which may expressed (sic) as numbness feeling, but it is hard to explain he had few beats of clonus at initial evaluation also he was complaining [of] some right-sided problem[s] at that time. When I saw the patient the symptoms were more on the left side and he was demonstrating left leg weakness, more proximal saying that his leg was buckling, so it could be some soft tissue problems causing symptoms at that time. But his swelling was not noticed or diagnosed before until December when he came to follow-up with me. It was only involving his left ankle and lower leg that is why I sent him to ED." *Id*.

d. Expert Opinions on Diagnosis[35]

Dr. Rubenstein opined that petitioner had mild, early, asymmetric TM 22 days after receipt of an influenza vaccine, and that petitioner "clearly had an autoimmune event which caused him to have significant neurologic dysfunction which was successfully treated with IV and oral steroids." His subsequent lymphedema was a "direct result of the asymmetric transverse myelitis." Pet. Ex. 14 at 3; Pet. Ex. 27 at 1; Pet. Ex. 30 at 1.

In defending his opinion that petitioner suffered from TM despite all objective testing being normal, Dr. Rubenstein argued that every patient presents differently, and "a diagnosis must be based on a holistic consideration of all the available evidence." Pet. Ex. 27 at 3. He agreed petitioner's presentation was mild and unusual but relied on *Frohman & Wingerchuk* to support that rapid-onset, "unilateral[,] or markedly asymmetric presentations can occur." *Id*. at 3-4; Pet. Ex. 29 at 2.[36] Generally, though, symptoms of TM evolve over the course of hours to days and are bilateral. Pet. Ex. 29 at 2.

Dr. Rubenstein argued that normal imaging or CSF does not rule out TM, citing a study wherein roughly 20% of participants had a TM diagnosis with either normal MRI or normal CSF

[35] Dr. Shah's and Dr. Hicks' opinions assume petitioner had TM and are therefore not relevant to this part of the Ruling.
[36] Frohman & Wingerchuk, *supra* note 5.

findings. In sum, "a lack of abnormal findings on MRI does not undercut a TM diagnosis based on the patient's subjective history, objective clinical findings, and response to treatment – particularly for a mild case." Pet. Ex. 47 at 1; Pet. Ex. 49.[37]

Dr. Rubenstein added that bowel or bladder changes would not be expected in petitioner's case because it was mild and most importantly asymmetric, suggesting that the demyelinating lesions did not cross petitioner's spinal cord and thus did not interrupt signals to the bowel and bladder. Pet. Ex. 27 at 5. He further submitted that petitioner's lack of sudomotor changes (referring to changes in the ability to sweat) and vasomotor changes (blanching or atypical redness of the skin) were either not present in petitioner or were overlooked. *Id*. at 5-6. In sum, Dr. Rubenstein argued that petitioner's lack of the characteristic symptoms of TM can be attributed to how mild his TM was, that not all symptoms are present in every individual, or that the doctors caring for petitioner missed the signs of vasomotor and sudomotor changes. *Id*.

However, Dr. Rubenstein also argued that petitioner's treating physicians were in the best position to render a diagnosis and diagnosed and treated petitioner for TM. Further, the steroid treatment was successful in resolving his neurological symptoms which suggested that he suffered from an autoimmune neuro-demyelination. Pet. Ex. 27 at 2-3.

Dr. Sweeney disagreed that petitioner had TM. He acknowledged that TM following flu vaccine is rare, and he was unable to find published cases in the literature to support a post-influenza myelitis without findings on imaging or evidence of inflammation on CSF. Resp. Ex. A at 3; Resp. Ex. A Tab 1;[38] Resp. Ex. A Tab 2;[39] Resp. Ex. A Tab 3;[40] Resp. Ex. I at 2.

Dr. Sweeney noted that petitioner's TM diagnosis was based on clinical presentation of acute onset of weakness and brisk reflexes including positive Babinski in his left leg several weeks after a flu vaccine. However, there was no evidence of myelitis on MRI of the spine and no evidence of inflammation on CSF testing which was performed more than two days after onset. Petitioner awoke with symptoms on September 20 but his symptoms did not progress. Resp. Ex. A at 3-4. Additionally, petitioner had none of the autonomic changes typically present in early TM including changes to bowel or bladder, vasomotor, or sudomotor functions. *Id*. at 4. Therefore, petitioner's presentation did not meet the diagnostic criteria for TM. *Id*. at 5.

Dr. Sweeney argued that the absence of evidence of inflammation within the CSF and normal MRI of the spine supports that this was not myelitis. Resp. Ex. H at 1. He addressed Dr. Rubenstein's opinion that normal imaging or CSF does not rule out TM noting that one study showed only two out of forty-seven participants with normal imaging and another study showed two out of thirty-eight subjects with normal imaging, but they had CSF abnormalities consistent with inflammation. Resp. Ex. H at 1; Resp. Ex. H Tab 2;[41] Resp. Ex. H at 1; Resp. Ex. H Tab

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[37] J. Sellner et al., *Diagnostic Workup of Patients with Acute Transverse Myelitis: Spectrum of Clinical Presentation, Neuroimaging and Laboratory Findings*, 47 SPINAL CORD 312 (2009), filed as "Pet. Ex. 49".

[38] Naoko Nakamura et al., *Neurologic Complications Associated with Influenza Vaccination: Two Adult Cases*, 42 INTERNAL MEDICINE 191 (2003), filed as "Resp. Ex. A Tab 1".

[39] A.J. Larner & S.F. Farmer, *Myelopathy Following Influenza Vaccination in Inflammatory CNS Disorder Treated with Chronic Immunosuppression*, 7 EUROPEAN J. NEUROLOGY 731 (2000), filed as "Resp. Ex. A Tab 2".

[40] Bakshi & Mazziotta, *supra* note 32.

[41] F.S. Pidcock, MD et al., *Acute Transverse Myelitis in Childhood: Center-Based Analysis of 47 Cases*, 68

3.[42] He also addressed *Sellner et al*., submitted by Dr. Rubenstein, stating that only two out of sixty-three subjects in the study had normal imaging and normal CSF findings but all had sensory disturbances. Resp. Ex. I at 1; Pet. Ex. 49.[43] Summarily, Dr. Sweeney opined that the combination of "the lack of bladder involvement, the absence of sensory abnormalities, the normal CSF WBC count and protein level, and the normal MRI of the spinal cord" all make a diagnosis of TM "extremely unlikely." Resp. Ex. H at 1.

Further, Dr. Sweeney pointed to Dr. Alper's record documenting that petitioner's bilateral sensory symptoms and left leg weakness did not localize well, meaning that Dr. Alper felt that a single lesion could not explain the constellation of findings. Resp. Ex. I at 1. Dr. Sweeney disagreed that a lesion too small to be seen on MRI was capable of causing what Dr. Rubenstein referred to as significant neurological dysfunction. Further, a lesion responsible for causing all petitioner's symptoms "would also be predicted to cause other symptoms that were not seen [], such as a sensory level and bowel/blader dysfunction." *Id*. Dr. Sweeney concluded that the etiology of petitioner's weakness and hyperreflexia were unclear but not consistent with TM. Resp. Ex. A at 5.

e. Summary on Diagnosis

As to whether petitioner suffered from TM, the following conclusions are reached after a complete review of the evidence filed.

The diagnostic criteria for a diagnosis of TM are clear and include acute or subacute motor, sensory, and autonomic spinal cord dysfunction. Pet. Ex. 29 at 1.[44] Symptoms evolve over the course of hours to days and are usually bilateral but can be unilateral and asymmetric. *Id*. at 2. The finding of one or more intrinsic cord lesions, which usually enhance with gadolinium, on MRI is characteristic of myelitis. *Id*.

Frohman & Wingerchuk describe TM as a "heterogeneous group of inflammatory disorders characterized by acute or subacute motor, sensory, and autonomic (bladder, bowel, and sexual) spinal cord dysfunction". Pet. Ex. 29 at 1. They further document that the "pathological hallmark of [TM] is the presence of focal collections of lymphocytes and monocytes, with varying degrees of demyelination, axonal injury, and astroglial and microglial activation, within the spinal cord." *Id*. Clinical symptoms of myelopathy may include urinary incontinence or retention, bowel incontinence or constipation, sexual dysfunction, altered pain and temperature sensations, paresthesias, and paroxysmal tonic spasms. *Id*. at 2. They noted that "[c]linical events that are consistent with" TM without associated CSF or MRI abnormalities "and that have no identifiable underlying cause are categorized as possible idiopathic transverse myelitis." *Id*., Table 1.

NEUROLOGY 1474 (2007), filed as "Resp. Ex. H Tab 2".

[42] Terrence Thomas, MD et al., *The Demographic, Clinical, and Magnetic Resonance Imaging (MRI) Features of Transverse Myelitis in Children*, 27 J. CHILD NEUROLOGY 11 (2012), filed as "Resp. Ex. H Tab 3".

[43] Sellner et al., *supra* note 37.

[44] Frohman & Wingerchuk, *supra* note 5.

During his September 2015 hospitalization, petitioner underwent extensive testing, all of which was normal, including normal MRIs and normal CSF studies. Pet. Ex. 5 at 24-25, 26, 31, 80, 137-39, 150-54, 166-69. He had none of the associated autonomic and sudomotor symptoms characteristic of TM. *Id*. at 82, 84, 100, 177. He showed questionable sensory deficit to pinprick and mild motor dysfunction of the lower left extremity, but reflexes were intact. *Id*. at 33, 38, 40-41, 65, 80. Succinctly, petitioner showed none of the "pathological hallmark[s]" of TM. Pet. Ex. 29 at 1-2.[45] His medical records specifically document TM as a presumed diagnosis based on clinical presentation, which is why he was treated with steroids. Pet. Ex. 5 at 31. Though his condition improved with the steroids, a response to steroids alone does not support a diagnosis of or constitute proof of demyelinating disease. As explained by Dr. Shah, steroids are an effective treatment of a wide variety of conditions, injuries, and illnesses, including lymphedema. Pet. Ex. 31 at 5; Pet. Ex. 41;[46] Pet. Ex. 42.[47]

Petitioner's medical record documents that a "presumptive diagnosis of early transverse myelitis was made based on his symptoms and his recent URI symptoms." Pet. Ex. 5 at 31. On follow up examination one month later, he was referred to as having "[i]mproving transient myelitis." Pet. Ex. 3 at 3-4. Three months later, in December of 2015, he was "completely fine", had returned to all activities with no neurological symptoms to report. Pet. Ex. 5 at 422-23. Dr. Alper noted some swelling of the left ankle at that time and began to question the earlier myelitis diagnosis. Pet. Ex. 10 at 25-27. In April of 2016, Dr. Alper again questioned the "presumed" diagnosis of TM writing that petitioner's symptoms in September could have been a result of the beginning of his edema which manifested as numbness, but that would not explain the clonus or the right-sided complaints. *Id*. at 14-15.

Dr. McCormick explained that he included Dr. Alpers' findings in his notes because the lymphedema was a new finding and was why he was evaluating petitioner; he noted petitioner was hospitalized in September of 2015 but it was not for lymphedema. Pet. Ex. 13 at 21-22. He also included the facts that informed his thinking about petitioner's case, including petitioner's presentation of only one swollen leg, no joint swelling, no fever, no rash, no shortness of breath, no loss of appetite, etc. The general screening questions were designed to ascertain whether the unilateral leg swelling was part of a larger process like a systemic illness, a rheumatological disorder, or an oncologic disorder for example. *Id*. at 22-23. Petitioner's presentation and his earlier testing ruled out those conditions, including systemic illness. *Id*. at 23-24.

The totality of the evidence does not preponderate in favor of a finding that petitioner suffered from TM. When objective testing does not meet the diagnostic criteria for the disease, the impressions of the treating physicians are particularly persuasive. Here, the treating physicians referred to "presumptive" TM in light of presentation attributing it to a recent URI and later referred to as "[i]mproving transient myelitis." Pet. Ex. 5 at 31; Pet Ex. 3 at 3-4. The September 2015 hospital discharge diagnosis was "[w]eakness of left lower extremity"—not TM. Pet. Ex. 5 at 30-33. In December 2015, Dr. Alper questioned the TM diagnosis and by April

[45] *Id*.
[46] N.C.M. Fyfe, F.R.C.S. et al., *Intralymphatic Steroid Therapy for Lymphoedema: Preliminary Studies*, 15 LYMPHOLOGY 23 (1982), filed as "Pet. Ex. 41".
[47] Stanley G. Rockson et al., *Pilot Studies Demonstrate the Potential Benefits of Antiinflammatory Therapy in Human Lymphedema*, 3 JCI INSIGHT e123775 (2018), filed as "Pet. Ex. 42".

2016, she considered that his September 2015 symptoms may have been early, mild edema manifesting as numbness rather than TM. Pet. Ex. 5 at 422-24; Pet. Ex. 10 at 14-15. Dr. McCormick referred to petitioner's lymphedema as "new" at the time he examined him in September 2016 and not related to his hospitalization in September 2015 with testing at that time ruling out other conditions including systemic illness. Pet. Ex. 13 at 21-24.

Dr. Rubenstein submitted that TM is a "diagnosis of exclusion". Pet. Ex. 27 at 3, quoting Pet. Ex. 28 at 7.[48] However, the study on which Dr. Rubenstein relied contained this statement in the context of "[i]nflammatory transverse myelitis" where CSF shows inflammation and in the absence of a specific cause; here, petitioner had normal CSF findings. Pet. Ex. 28 at 7; Pet. Ex. 5 at 24-25, 26, 31, 80, 137-39, 150-54, 166-69. Dr. Rubenstein's opinion that petitioner had TM may have been more persuasive if petitioner met some of the diagnostic criteria or if petitioner's treating physicians believed TM was the correct diagnosis. It would not be reasonable to accept TM as the diagnosis here in the absence of the "hallmark[s]" of the disease, as detailed in the literature relied on by Dr. Rubenstein. *See* Pet. Ex. 29.[49] Further, based on the medical records, it is clear that petitioner's treaters were not confident that he actually had TM. *See* Pet. Ex. 5 at 30-33, 422-24; Pet Ex. 3 at 3-4; Pet. Ex. 10 at 14-15.

I found the opinion of respondent's expert, Dr. Sweeney, to be more persuasive on the issue of diagnosis. He agreed that there are reports of TM following flu vaccination but submitted that there is no support for a post-influenza myelitis without findings on imaging or evidence of inflammation on CSF. Resp. Ex. A at 3; Resp. Ex. A Tab 1;[50] Resp. Ex. A Tab 2;[51] Resp. Ex. A Tab 3;[52] Resp. Ex. I at 2. Without objective findings to support the diagnosis, in addition to the lack of autonomic or sudomotor changes, it is extremely unlikely that petitioner had TM. Resp. Ex. A at 3-5.

Based on the totality of evidence filed—the medical records, opinions of the treating physicians and experts, and the literature—there is not preponderant evidence to support that petitioner suffered from TM or an inflammatory spinal process.

B. *Althen* Prongs

Because petitioner does not allege an injury listed on the Vaccine Injury Table, his claim is classified as "off-Table." As noted above, for petitioner to prevail on an "off-Table" claim, he must show by preponderant evidence that his claimed injury resulted from the vaccination at issue. *Capizzano*, 440 F.3d at 1320. Doing so shifts the burden to respondent to show that the injury was caused by factors unrelated to the vaccination. *Deribeaux*, 717 F.3d at 1367.

Presenting a sound and reliable theory is essential to petitioner's case. A theory causally connecting the vaccine to the injury is the first *Althen* prong. When petitioner fails to establish this element, compensation is denied. *Boatmon v. Sec'y of Health & Human Servs*., 941 F.3d

[48] Jacob & Weinshenker, *supra* note 30.
[49] Frohman & Wingerchuk, *supra* note 5.
[50] Nakamura et al., *supra* note 38.
[51] Larner & Farmer, *supra* note 39.
[52] Bakshi & Mazziotta, *supra* note 32.

1351, 1360-62 (Fed. Cir. 2019). Moreover, the theory advanced for *Althen* prong one influences the remaining two *Althen* prongs. For the second *Althen* prong, which addresses whether a logical sequence connects the vaccine to the injury, special masters may consider whether the vaccinee responded in a way consistent with the theory being offered. *Hibbard v. Sec'y of Health & Human Servs*., 698 F.3d 1355, 1364 (Fed. Cir. 2012); *Miller v. Sec'y of Health & Human Servs.*, 172 Fed. Cl. 762, 784 (2024) (finding special master did not err in denying entitlement when petitioner did not establish that she had immune complexes after asserting a theory involving immune complexes); *Dodd v. Sec'y of Health & Human Servs*., 114 Fed. Cl. 43, 52-57 (2013); *La Londe v. Sec'y of Health & Human Servs*., 110 Fed. Cl. 184, 205 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). Similarly, the third *Althen* prong, which concerns timing, depends at least in part upon the theory being offered. *Langland v. Sec'y of Health & Human Servs*., 109 Fed. Cl. 421, 443 (2013); *see also Koehn v. Sec'y of Health & Human Servs*., 773 F.3d 1239, 1244-45 (Fed. Cir. 2014) (holding that special master was not arbitrary in finding an onset of injury seven months after vaccination was incompatible with a theory based upon cytokines). Without a defined theory, attempting to determine whether preponderant evidence supports the logical sequence or timing is difficult if not impossible.

The parties never addressed the consequences of a finding that petitioner did not suffer from TM or an inflammatory myelitis. Rather, they focused on whether petitioner did or did not have TM, how individual symptoms or subjective complaints did or did not satisfy the diagnostic criteria of TM, and the various explanations as to why petitioner had TM despite the lack of any objective findings.

It is undisputed that petitioner suffers from lymphedema of the left leg. The remaining issues to be resolved is whether a flu vaccine can cause lymphedema and to a lesser extent, whether petitioner's lymphedema is primary or secondary lymphedema. To that end, I find that petitioner's flu vaccine played no role in his development of lymphedema.

a. Petitioner Has Not Provided a Sound and Reliable Medical Theory

The first *Althen* prong requires petitioner to provide a "reputable medical theory" demonstrating that the vaccines received *can* cause the type of injury alleged. *Pafford*, 451 F.3d at 1355-56 (citation omitted). To satisfy this prong, petitioner's "theory of causation must be supported by a 'reputable medical or scientific explanation.'" *Andreu ex rel. Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1379 (Fed. Cir. 2009) (quoting *Althen*, 418 F.3d at 1278). This theory need only be "legally probable, not medically or scientifically certain." *Id.* at 1380 (emphasis omitted) (quoting *Knudsen*, 35 F.3d at 548). This standard was recently clarified by the Federal Circuit. *See Boatmon*, 941 F.3d at 1359-60 (stating that the correct standard for *Althen* prong one is "reputable," and "sound and reliable" not a "lower reasonable standard" (internal quotations omitted)). Nevertheless, "petitioners [must] proffer trustworthy testimony from experts who can find support for their theories in medical literature." *LaLonde v. Sec'y of Health & Human Servs.*, 746 F.3d 1334, 1341 (Fed. Cir. 2014).

Special masters may consider the relative expertise of testifying experts when weighing the value of their opinion. *See Depena v. Sec'y of Health & Human Servs.*, No. 13-675V, 2017 WL 1075101 (Fed. Cl. Spec. Mstr. Feb. 22, 2017), *mot. for rev. denied*, 133 Fed. Cl. 535, 547-48

(2017), *aff'd without op.,* 730 Fed. App'x 938 (Fed. Cir. 2018); *Copenhaver v. Sec'y of Health & Human Servs.,* No. 13-1002V, 2016 WL 3456436 (Fed. Cl. Spec. Mstr. May 31, 2016), *mot. for rev. denied*, 129 Fed. Cl. 176 (2016).

Petitioner presents two different causation theories. First, Dr. Rubenstein submits that the flu vaccine caused petitioner's TM and the damage from TM caused him to develop lymphedema. Second, Dr. Shah opines that the flu vaccine caused an inflammatory response that caused petitioner to develop lymphedema.

Petitioner's first causation theory hinges on the presence of spinal cord myelitis. Specifically, Dr. Rubenstein's theory is predicated on petitioner having suffered from TM. He opined that the flu vaccine caused petitioner's TM which subsequently resulted in his development of secondary left leg lymphedema due to the improper functioning of the sympathetic nervous system,[53] which is a complication of spinal cord myelitis and likely occurred during the process of sympathetic re-innervation.[54] Pet. Ex. 27 at 4-5; Reply at 8. He submitted that re-innervation following the nerve damage caused by the TM led to dysregulation of lymphatic flow in the left lower extremity where the TM symptoms localized, causing secondary lymphedema. *Id*. Dr. Rubenstein failed to explain how a flu vaccine could have caused lymphedema in the absence of TM.

According to Dr. Rubenstein petitioner's lymphedema was "a complication of 'dysfunction' of the sympathetic nervous system. In other words, this was not a matter of a simple increase or decrease in sympathetic outflow but of improper functioning of the sympathetic nervous system as a complication of spinal myelitis". Pet. Ex. 27 at 4-5. Using complex regional pain syndrome ("CRPS") as an example, Dr. Rubenstein explained that "improper 'wiring'" can cause lymphedema in a patient with re-innervation after spinal myelitis just as it can cause pain in a patient with CRPS. *Id*. at 5. He argued that "there is every reason to think that sympathetic dysfunction in the context of TM could likewise lead to secondary lymphedema." Pet. Ex. 30 at 3. Importantly, Dr. Rubenstein failed to propose a theory of sympathetic dysfunction causing secondary lymphedema *outside* the context of TM.

Dr. Rubenstein's opinions for the most part are no longer relevant since they depend on a finding that petitioner suffered from TM or spinal cord inflammation. As detailed at length above, there is not preponderant evidence that petitioner suffered from TM or spinal cord

[53] The sympathetic nervous system is the portion of the autonomic nervous system that receives its fibers of connection with the central nervous system through the thoracolumbar outflow of visceral efferent fibers. These fibers (preganglionic fibers) arise from cells in the thoracic and upper lumbar levels of the spinal cord, leave by way of ventral roots, and, by way of rami communicantes, enter sympathetic trunks, where some synapse with ganglion cells. Distal to these ganglia, there are postganglionic fibers that either return to spinal nerves by way of rami communicantes to supply blood vessels, smooth muscles, and glands of the trunk and limbs, or go as visceral branches to blood vessels, smooth muscles, glands of the head and neck, and viscera of the thorax, abdomen, and pelvis. Some preganglionic fibers pass through the sympathetic trunks and synapse in the prevertebral ganglia; postganglionic fibers from those ganglia supply adjacent viscera. Sympathetic nervous system, *Dorland's Medical Dictionary Online*, https://www.dorlandsonline.com/dorland/definition?id=111901&searchterm=sympathetic+nervous+system.

[54] Reinnervation is the restoration of nerve function to a part from which it was lost; it may occur spontaneously or be achieved by nerve grafting. Reinnervation, *Dorland's Medical Dictionary Online*, https://www.dorlandsonline.com/dorland/definition?id=43257&searchterm=reinnervation.

inflammation/myelitis. However, even if objective evidence existed that supported TM or spinal cord inflammation, and assuming Dr. Rubenstein's theory based on molecular mimicry was sufficient to prove that flu vaccine can cause TM, there is not preponderant evidence to support the remainder of Dr. Rubenstein's theory that petitioner's left leg lymphedema that developed three months after his September 2015 symptoms was "a direct complication of dysfunction of the peripheral sympathetic nervous system" resulting from his TM. Pet. Ex. 14 at 4.

Dr. Shah built his opinion on Dr. Rubenstein's causation theory "assuming Dr. Rubenstein's description of the effects of TM on the sympathetic nervous system is accurate, it would then follow that this type of sympathetic nervous system dysfunction could cause or exacerbate lymphedema . . . particularly [in a patient like petitioner] who was already very likely developing lymphedema as a direct side effect of the vaccine." Pet. Ex. 51 at 1. He opined that it is most likely that petitioner had an abnormal inflammatory response to the vaccine, causing both his TM and lymphedema, "with the lymphedema exacerbated by damage to the sympathetic nervous system as outlined by Dr. Rubenstein." *Id*. at 2; Pet. Ex. 31 at 4, 5. "Since transverse myelitis is known to result from inflammation of the spinal cord, it is highly probable that the same inflammatory process that triggered spinal cord inflammation and transverse myelitis subsequently resulted in an inflammatory process in the lymphatic system thus triggering lymphedema . . .". Pet. Ex. 31 at 5.

While conceding that no literature points to causality between lymphedema and flu vaccines, Dr. Shah argued that "one can largely conclude that [petitioner]'s lymphedema was secondary lymphedema" in that "his lymphedema was in all likelihood secondary to an external cause whether that be trauma, surgery, radiation, infection or any other inflammatory cause." Pet Ex. 51 at 3. Dr. Shah opined that TM is the result of inflammation of the spinal cord making it "highly probable" that the inflammatory process that caused petitioner's TM progressed over time and slowly caused obstructed lymphatic channels that the body relies on for drainage of lymph fluid. Pet. Ex. 31 at 5. Even after the inflammatory process ends, obstruction can persist resulting in edema. The lack of any other factor that could trigger secondary lymphedema is notable, "as should the consistency of [petitioner's] vascular symptoms with the theory of post-vaccinal inflammation." Pet. Ex. 31 at 5; Pet. Ex. 44.[55]

As to the second proposed causation theory, Dr. Shah stated that having ruled out other obvious causes and "given the well accepted fact that the flu vaccine can cause excessive systemic inflammation in a small number of patients, it is reasonable to conclude that the flu shot resulted in a pathological inflammatory reaction resulting in secondary lymphedema." Pet. Ex. 51 at 3; Pet. Ex. 52.[56] He added that whatever the correct diagnosis for petitioner's neurological

[55] Ly et al., *supra* note 9.

[56] "This study examined the extent to which baseline inflammatory status predicted subjective symptoms and the extent to which subjective symptoms corresponded with objective inflammatory responses in the days following receipt of a flu vaccine." The sample included 56 women, 28 of whom were pregnant. *Id*. at 6. Previous studies showed that inflammatory responses to flu infection are more severe in females than in males. *Id*. at 8. The researchers found that reports of subjective symptoms following vaccination (like pain or systemic symptoms) are "accompanied by measurable differences in vaccine-induced serum proinflammatory cytokine responses as well as some possible associations with body temperature." *Id*. Given the study's conclusion that subjective complaints of symptoms correspond with objective markers of inflammation, it is not clear how this study supports Dr. Shah's proposition that petitioner—who had no subjective symptoms or objective testing indicative of inflammation—had a

symptoms, "they provide strong evidence of a systemic inflammatory response to the vaccine given that they followed it so closely in time." Pet. Ex. 51 at 3. There is no other "stronger causation" for petitioner's unilateral edema of his entire left leg. *Id*.

Dr. Shah claimed that his inflammation theory of causation was proposed "as an independent cause of [petitioner's] symptoms irrespective of the diagnosis of transverse myelitis." Pet. Ex. 51 at 3. Dr. Shah contended that his opinion and Dr. Rubenstein's were consistent but added that in general, inflammation caused by a vaccine could lead to lymphedema. *Id*. at 1. "This process can operate alongside and can be exacerbated by the one described by Dr. Rubenstein." *Id*. at 1-2.

Dr. Shah added that there is "much supporting literature" to support flu vaccines causing inflammatory reactions citing to only one article. *Id*. at 4; Pet. Ex. 52.[57] Such an inflammatory response could result in secondary lymphedema, and petitioner suffered "an episode of systemic inflammation in the fall of 2015". Pet. Ex. 51 at 4. Dr. Shah claimed that studies have associated lymphedema with an inflammatory process, but failed to explain how this can occur. Pet. Ex. 31 at 5.

Dr. Sweeney focused mainly on refuting the diagnosis of TM but also stated that the loss of sympathetic innervation has not been shown to be a driver of peripheral edema. Resp. Ex. A at 4; Resp. Ex. A Tab 5;[58] Pet. Ex. 53.[59] Dr. Sweeney maintained that lymphedema without the presence of other autonomic disturbances is unlikely to be caused by alterations in sympathetic innervation to a limb. Resp. Ex. A at 5. Most importantly, the proposed mechanism of sympathetic reinnervation elucidated here is not described in any literature. Resp. Ex. C at 3.

Dr. Sweeney added that even if he accepted the TM diagnosis, he could not "find a plausible link between myelitis and lymphedema", particularly when the lymphedema presented three months later. Resp. Ex. H at 2. There is no literature that shows myelitis to be associated with lymphedema or how it would present three months later. *Id*.

Dr. Hicks disagreed with Dr. Shah's opinion that the flu vaccine caused systemic inflammation that resulted in TM and lymphedema; rather, she believed the two were coincidental and that petitioner's lymphedema is likely primary lymphedema. Resp. Ex. G at 3. Dr. Hicks noted Dr. Shah's acknowledgment that primary lymphedema affects about 1.2 per 100,000 people and that no reports exist tying TM to secondary lymphedema or that a cause and effect relationship has ever being observed. *Id*.; Resp. Ex. G Tab 3.[60] Further, there is no literature that supports flu vaccine as a cause of lymphedema. Resp. Ex. D at 6. She explained that "[l]ymphedema is a disease of the lymphatic network, not the nervous system. . . While it is theoretically possible that sympathetic nervous system dysfunction may somehow be associated

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"pathological inflammatory reaction resulting in secondary lymphedema." Pet. Ex. 51 at 3.

[57] *See id.*

[58] Douglas Howarth, M.D. et al., *Autonomic Regulation of Lymphatic Flow in the Lower Extremity Demonstrated on Lymphoscintigraphy in Patients with Reflex Sympathetic Dystrophy*, 24 CLINICAL NUCLEAR MEDICINE 383 (1999), filed as "Resp. Ex. A Tab 5".

[59] Noel McHale, *Nervous Control of the Lymphatic System*, 4 VASCULAR MEDICINE REVIEW 307 (1993), filed as "Pet. Ex. 53".

[60] Smeltzer et al., *supra* note 8.

with lower extremity swelling, it is highly unlikely that 'improper crosstalk among neurons and neural groups can produce a variety of symptoms, including lymphedema.'" *Id*.

As already noted, petitioner's theory of causation is largely predicated on a finding that petitioner had TM and that his TM was caused by the flu vaccine. Thus, my finding that petitioner did not suffer from TM dramatically undercuts his claim. "Because causation is relative to the injury, a petitioner must provide a reputable medical or scientific explanation that pertains specifically to the petitioner's case, although the explanation need only be 'legally probable, not medically or scientifically certain.'" *Broekelschen*, 618 F.3d at 1345 (quoting *Knudsen*, 35 F.3d at 548-49). The basis of the theory proposed by Dr. Rubenstein and accepted by Dr. Shah was that petitioner had inflammation of the spinal cord, which caused sympathetic reinnervation and ultimately lymphedema. Because I found no evidence to support the diagnosis of TM or inflammation of the spinal cord, this theory fails.

The remaining theory implicating a systemic inflammatory response to the vaccine is insufficient to meet petitioner's burden. Vaccines are meant to cause an inflammatory response, which is how antibodies are generated and what makes vaccines effective. Similar theories involving excessive inflammation have been accepted as persuasive in other vaccine cases. However, here, the theory was underdeveloped and insufficient. Conclusory assertions that a petitioner suffered an abnormal inflammatory response after a vaccine with nothing more are insufficient to meet petitioner's burden under *Althen* prong one. Further, petitioner's experts provided no persuasive evidence to support any connection between a flu vaccine, a subsequent inflammatory response, and lymphedema. Accordingly, petitioner has failed to satisfy prong one.

b. Petitioner Has Not Demonstrated a Logical Sequence of Cause and Effect

The second *Althen* prong requires proof of a "logical sequence of cause and effect." *Capizzano*, 440 F.3d at 1326 (quoting *Althen*, 418 F.3d at 1278). In other words, even if the vaccinations can cause the injury, petitioner must show "that it did so in [this] particular case." *Hodges v. Sec'y of Health & Human Servs.*, 9 F.3d 958, 962 n.4 (Fed. Cir. 1993) (citation omitted). "A reputable medical or scientific explanation must support this logical sequence of cause and effect," *id.* at 961 (citation omitted), and "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," *Paluck v. Sec'y of Health & Human Servs.*, 786 F.3d 1373, 1385 (Fed. Cir. 2015) (quoting *Andreu*, 569 F.3d at 1375). Petitioner is not, however, required "to eliminate alternative causes as part of establishing [their] prima facie case." *Doe v. Sec'y of Health & Human Servs.*, 601 F.3d 1349, 1357-58 (Fed. Cir. 2010); *see Walther v. Sec'y of Health & Human Servs.*, 485 F.3d 1146, 1152 (Fed. Cir. 2007) (holding that a "petitioner does not bear the burden of eliminating alternative independent potential causes").

Petitioner here proposed two theories of causation. Not only do I not find either theory to be sound and reliable, but I also do not find either theory to be consistent with petitioner's medical history. Succinctly, without objective proof of a neurological injury as the cause of petitioner's September 2015 symptoms, Dr. Rubenstein's theory of improper functioning of the sympathetic nervous system as a complication of spinal myelitis, likely occurring during the process of sympathetic re-innervation resulting in secondary lymphedema, fails on prong two.

See Pet. Ex. 27 at 4-5. Dr. Shah's additional theory that petitioner suffered a systemic inflammatory process that led to his development of secondary lymphedema also fails as there is no evidence to support that petitioner suffered from an excessive, abnormal inflammatory response to the vaccine. *See* Pet. Ex. 51 at 3; *see also* Pet. Ex. 13 at 24-27 (Dr. McCormick explaining that the Schmorl's nodes seen on petitioner's MRI were non-enhancing therefore not inflamed, meaning that they had been "stable" for some time.). Dr. Sweeney pointed out that there was no evidence of inflammation; rather, petitioner had normal CSF testing and normal MRI, little reported sensory loss, numbness, and/or weakness, and no bowel, bladder, or sexual dysfunction, or pain. Resp. Ex. A at 3-4, Resp. Ex. H at 1, 2; Resp. Ex. H Tab 2;[61] Resp. Ex. H Tab 3;[62] Resp. Ex. H Tab 4.[63]

Petitioner developed tingling and numbness of his left lower leg three weeks after his receipt of the subject flu vaccine. Pet. Ex. 3 at 5; Pet. Ex. 5 at 39. He had slightly decreased motor strength in the left lower extremity and diminished pinprick and light touch sensation in both feet. Pet. Ex. 5 at 40-42. All testing was normal. *Id*. at 24-26, 31, 35, 80, 137-39, 150-54, 166-69. Despite negative testing but because of his presentation and recent upper respiratory infection, "[a] presumptive diagnosis of early transverse myelitis was made". *Id*. at 31. His symptoms improved with steroid treatment, and he was stable upon discharge. *Id*. at 31, 34-36, 40. The discharge diagnosis was "[w]eakness of left lower extremity" not TM. *Id*. at 36.

Thereafter, petitioner was "completely fine" and resumed his regular activities. Pet. Ex. 5 at 422-23. However, on December 3, 2015, at a follow up visit, Dr. Alper noted left ankle swelling and a few beats of clonus. *Id*. 422-24. Petitioner underwent further testing, which showed no significant abnormalities. *Id*. at 105-08.

Three months later, on February 20, 2016, petitioner was then hospitalized for left leg swelling since December 2015 that had increased in the last few days. Pet. Ex. 5 at 10. MRIs of the thoracic and lumbar spine were normal. MRI of the pelvis showed left iliac vein compression by the right iliac artery. *Id*. at 10, 15, 127-28. The discharge diagnosis was swelling of the lower extremity. *Id*. at 10-11, 13.

Petitioner's left leg swelling increased throughout the following months. Pet. Ex. 3 at 2; Pet. Ex. 4 at 2-3; Pet. Ex. 6 at 3-4, 6; Pet. Ex. 8 at 19-20; Pet. Ex. 10 at 6. The lymphedema diagnosis was confirmed following a lymphoscintigraphy performed in June 2016 and an MRI/A performed in October 2016. Pet. Ex. 6 at 7, 8-9; Pet. Ex. 8 at 27-28.

Summarily, petitioner's MRIs did not indicate myelitis or any other inflammatory process. Likewise, petitioner's CSF studies refute the notion that there was an ongoing inflammatory response with the WBC within normal limits. Pet. Ex. 5 at 80, 150-154. Thus, the objective test results refuted petitioner's main theory that he had TM which caused lymphedema

[61] Pidcock et al., *supra* note 41.
[62] Thomas et al., *supra* note 42.
[63] Thomas F. Scott et al., *Acute Partial Transverse Myelitis with Normal Cerebral Magnetic Resonance Imaging: Transition Rate to Clinically Definite Multiple Sclerosis*, 11 MULTIPLE SCLEROSIS 373 (2005), filed as "Resp. Ex. H Tab 4".

as well as the secondary theory that he had an abnormal inflammatory response to the vaccine which caused his lymphedema.

Further, none of petitioner's treating physicians associated his leg swelling with his flu vaccine or with the symptoms he had in September 2015 other than to question whether he in fact had the early manifestations of lymphedema in September of 2015. *See* Pet. Ex. 10 at 14-15 (Dr. Alper questioning whether vascular issues may have been the cause of the neurological symptoms in September of 2015 rather than the other way around). Instead, petitioner's treating providers opined that petitioner had primary lymphedema, which is consistent with respondent's position. Pet. Ex. 6 at 4 (noting Dr. McEnaney's impression of May Thurner syndrome v. primary lymphedema); Pet. Ex. 10 at 15 (Dr. Alper noting that the lower leg swelling/edema was possibly lymphedema without explanation and no underlying diagnosis); Pet. Ex. 13 at 21-24, 45 (Dr. McCormick including Dr. Alpers' findings and petitioner's hospitalization in September of 2015 not for lymphedema and with larger processes like systemic illness, rheumatological disorder, or an oncological disorder ruled out; stating that after examining petitioner, that he believed he "fits the category of primary lymphedema"); *see also* Resp. Ex. D at 6; Resp. Ex. G at 3 (Dr. Hicks opining that petitioner most likely had "pre-existing primary lymphedema" that began to manifest as he entered adolescence).

While there are incidences where vaccines cause excessive inflammatory responses resulting in injuries through the mechanisms highlighted by Dr. Rubenstein and Dr. Shah, there is no objective evidence in this case that the flu vaccine administered to petitioner caused an excessive systemic inflammatory response three weeks after vaccination.

Accordingly, petitioner has failed to provide a logical sequence of cause and effect connecting the subject flu vaccine to either his symptoms in September 2015 or his leg swelling ultimately diagnosed as lymphedema. Thus, petitioner has failed to satisfy *Althen* prong two.

c. Petitioner Has Not Demonstrated a Proximate Temporal Relationship

To satisfy the third *Althen* prong, petitioner must establish a "proximate temporal relationship" between the vaccination and the alleged injury. *Althen*, 418 F.3d at 1281. This "requires preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *de Bazan*, 539 F.3d at 1352. Typically, "a petitioner's failure to satisfy the proximate temporal relationship prong is due to the fact that onset was too late after the administration of a vaccine for the vaccine to be the cause." *Id.* However, "cases in which onset is too soon" also fail this prong; "in either case, the temporal relationship is not such that it is medically acceptable to conclude that the vaccination and the injury are causally linked." *Id.*; *see also Locane v. Sec'y of Health & Human Servs.*, 685 F.3d 1375, 1381 (Fed. Cir. 2012) ("[If] the illness was present before the vaccine was administered, logically, the vaccine could not have caused the illness.").

Other than concluding that the "best supported hypothesis" in this case was that petitioner's lymphedema was the direct result of his TM which occurred 22 days after a flu

vaccine, Dr. Rubenstein did not discuss the medically appropriate timeframe for a flu vaccine causing TM, TM causing lymphedema, or a flu vaccine causing lymphedema. Pet. Ex. 14 at 4.

Dr. Shah opined that the onset of petitioner's lower leg edema was on or around December 3, 2015, consistent with his theory of lymphedema secondary to vaccine-induced inflammation, "since a systemic inflammatory process affecting the lymphatic system would require anywhere from several weeks to several months' time to manifest itself as clinically significant lower extremity edema." Pet. Ex. 51 at 2. He further argued that Dr. Alper's suggestion that petitioner's lymphedema may have begun in September 2015 was speculative with no objective support in the medical records and no treating physician examining petitioner having noted any left leg swelling. *Id.*; Pet. Ex. 10 at 14-15.

Dr. Sweeney did not discuss timing. Dr. Hicks wrote that "[w]hile there is a very loose (months) temporal relationship between vaccination and [petitioner's] symptoms, temporal relationships do not mandate causation." More likely, petitioner developed primary lymphedema/praecox in the months following the flu vaccine. The onset of petitioner's lymphedema is consistent with the literature for onset of lymphedema praecox, "whereas a causal pathway between vaccination, transverse myelitis, and secondary lymphedema has never been described." Resp. Ex. J at 3; *see also* Resp. Ex. G Tab 6;[64] Resp. Ex. J Tab 5;[65] Resp. Ex. J Tab 6.[66]

Given the findings that petitioner did not have TM and did not meet *Althen* prongs one or two, petitioner's claim necessarily fails on prong three as well. Petitioner failed to present persuasive evidence regarding any causal association between a flu vaccine, his September 2015 symptoms, and lymphedema—much less a medically appropriate timeframe for onset. As such, petitioner has failed to prove prong three.

VI. Conclusion

Upon careful evaluation of all the evidence submitted in this matter—including the medical records, expert reports, medical literature, and other submitted arguments—I conclude that petitioner has not shown by preponderant evidence that he is entitled to compensation under the Vaccine Act. **The Clerk shall enter judgment accordingly.**[67]

IT IS SO ORDERED.

s/ Mindy Michaels Roth
Mindy Michaels Roth
Special Master

[64] Carolyn C. Schook, B.A. et al., *Primary Lymphedema: Clinical Features and Management in 138 Pediatric Patients*, 127 PLASTIC RECONSTRUCTION SURGERY 2419 (2011), filed as "Resp. Ex. G Tab 6".
[65] Isabelle Quere, M.D., Ph.D. et al., *Incidence of Cellulitis Among Children with Primary Lymphedema*, 378 N. ENG. J. MEDICINE 2047 (2018), filed as "Resp. Ex. J Tab 5".
[66] Colmant et al., *supra* note 17.
[67] Pursuant to Vaccine Rule 11(a), entry of judgment can be expedited by each party filing a notice renouncing the right to seek review.