In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 12-475V
Filed: October 4, 2017
Not to be Published

KIMBERLY S. THOMPSON,	*	
	*	
Petitioner,	*	
	*	
v.	*	Influenza ("flu") vaccine; H_1N_1 vaccine; peripheral neurological injury
	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
	*	
Respondent.	*	
	*	

Clifford J. Shoemaker, Vienna, VA, for petitioner.
Voris E. Johnson, Washington, DC, for respondent.

MILLMAN, Special Master

RULING ON ENTITLEMENT[1]

On July 27, 2012, petitioner filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10-34 (2012), alleging that influenza ("flu") vaccine administered on September 15, 2009 caused her neurologic injury. Pet. at ¶¶ 5, 6. Petitioner also received H_1N_1 influenza vaccine on October 23, 2009, which is not covered under the Vaccine Act during the 2009-2010 flu season.

On April 11, 2017, the undersigned held a hearing in this case. Testifying for petitioner was Dr. Carlo Tornatore, her treating neurologist. Testifying for respondent was Dr. Vinay Chaudhry, a neurologist. At the end of the hearing, the undersigned provisionally ruled on the

[1] Because this unpublished decision contains a reasoned explanation for the special master's action in this case, the special master intends to post this unpublished decision on the United States Court of Federal Claims' website, in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 note (2012) (Federal Management and Promotion of Electronic Government Services). Vaccine Rule 18(b) states that all decisions of the special masters will be made available to the public unless they contain trade secrets or commercial or financial information that is privileged and confidential, or medical or similar information whose disclosure would constitute a clearly unwarranted invasion of privacy. When such a decision is filed, petitioner has 14 days to identify and move to redact such information prior to the document's disclosure. If the special master, upon review, agrees that the identified material fits within the banned categories listed above, the special master shall redact such material from public access.

record in favor of petitioner with the understanding that respondent's expert Dr. Chaudhry had the opportunity to respond to a medical article (exhibit 126) which petitioner filed just a few days before the hearing. Dr. Chaudhry's response did not alter the undersigned's view that petitioner has prevailed in this case. Below, the undersigned gives a short summary of the medical records, the testimony at the hearing, and then the undersigned's provisional ruling on entitlement, which now remains the undersigned's opinion.

At the close of the hearing, the undersigned encouraged the parties to negotiate a settlement of damages. On September 27, 2017, respondent filed a status report stating that settlement was not feasible at this time and requesting a ruling on entitlement.

FACTS

On September 15, 2009, petitioner received trivalent flu vaccine. Med. recs. Ex. 29, at 3.

On October 23, 2009, petitioner received H_1N_1 monovalent flu vaccine.[2] Med. recs. Ex. 5, at 14.

Petitioner complained that she experienced facial drooping four days after receiving H_1N_1 vaccine. She had a brain MRI on October 28, 2009, which did not reveal acute disseminated encephalomyelitis ("ADEM"). Med. recs. Ex. 34, at 1. The 2009 brain MRI showed several foci of abnormal increased signal intensity involving the left forceps, major white matter tract, and the centrum semiovale white matter tracks at the frontal lobes bilaterally. There are not typical locations for multiple sclerosis ("MS") plaque formation, but the doctor stated MS plaque formation should be considered. These findings might represent old post-traumatic areas of gliosis change. A cervical spine MRI done the same day was normal.

On November 18, 2009, petitioner saw Dr. Preeti Yonker, a neurologist, stating she began noticing numbness in both sides of her face five days after receiving H_1N_1 flu vaccine. Now, she noticed a very mild tingling in both legs. She said her arms and legs felt weak. She returned after being admitted to Johns Hopkins Hospital where her weakness was felt to be functional. Her lumbar puncture results were normal. Dr. Yonker diagnosed petitioner with functional weakness, i.e., there was no physical basis for it, and stated she did not have Guillain-Barré Syndrome ("GBS"). Med. recs. Ex. 8, at 5, 6. On December 17, 2009, Dr. Yonker did an EMG and nerve conduction study on petitioner, whose results were normal. Id. at 2.

On February 9, 2010, Dr. Jeffrey W. Anderson performed a nerve conduction study and EMG on petitioner. Med. recs. Ex. 58, at 2. He concluded that petitioner examination was consistent with a demyelinating polyneuropathy affecting the bilateral lower extremities. Id. at 4. He thought she likely had acute inflammatory demyelinating polyneuropathy ("AIDP") given her clinical history and the present scenario.

[22] The Vaccine Injury Table does not include the H1N1 monovalent vaccine administered in the flu season from fall 2009 to spring 2010.

On May 5, 2010, Dr. Richard E. Bird, petitioner's neurologist, noted it was very difficult for petitioner to walk. Med. recs. Ex. 47, at 1. She could not work her feet, but she had no arm complaints. The facial numbness had resolved. He thought, looking at all the data, included decreased reflexes in the upper extremities and absent reflexes at the ankles, that petitioner had a mild GBS.

On May 27, 2010, petitioner saw Dr. Michael S. Sellman, a neurologist and electromyographer. Med. recs. Ex. 1, at 5. She told Dr. Sellman she received the October 23, 2009 H_1N_1 flu vaccine because her work required it. She said she received it at 7:30 a.m. and felt ill immediately. She had malaise. Within a few days, she had facial drooping and tingling, then numbness and weakness of her legs. She had had two normal EMGs. She was at Johns Hopkins from October 28, 2009 to November 2, 2009, which concluded she did not have GBS. Initially, Dr. Richard Bird, petitioner's neurologist, diagnosed her with apparent GBS or GBS-like syndrome following an H_1N_1 vaccination. But in April 2010, Dr. Bird reported that all of petitioner's symptoms had largely resolved except for bilateral footdrop. Her nerve conduction study was normal. Her reflexes were normal except for her ankles. Dr. Sellman concluded that petitioner did not have GBS.

On September 9, 2010, Dr. James W. Russell performed an EMG and nerve conduction study on petitioner, both of which were normal. Med. recs. Ex. 36, at 1, 2. Petitioner on examination demonstrated give-away weakness with poor effort. Dr. Russell noted petitioner had non-anatomical sensory loss, meaning her sensory complaints did not match the anatomical presence of nerves. He wrote that it was unlikely that petitioner had a history of GBS or recurrent neuropathy by her history and examination as well as by electrodiagnostics. He agreed with the Johns Hopkins conclusion that the most likely diagnosis was conversion disorder and he also agreed with the Johns Hopkins recommendation that she follow up with a psychologist.

On February 2, 2011, petitioner saw Dr. Bonnie Gerecke, a neurologist. Med. recs. Ex. 7, at 4. A lumbar puncture showed petitioner's protein count was 38, which is normal. Dr. Gerecke's diagnosis was that petitioner did not have GBS. She also did not have muscle atrophy. She had preserved reflexes and a normal nerve conduction study. Dr. Gerecke stated petitioner did not have myelitis. Petitioner's sensory examination was non-physiologic, i.e., petitioner's sensory complaints did not coincide with anatomical norms. Dr. Gerecke stated a conversion disorder must be considered. Id. On March 14, 2011, Dr. Gerecke did an EMG and nerve conduction study. The nerve conduction study was normal, but the EMG was mildly abnormal and could be consistent with a very mild bilateral S1 radiculopathy. Petitioner did not have an axonal or demyelinating peripheral neuropathy. Id. at 11.

On May 25, 2011, Dr. Sellman wrote an addendum in his notes that petitioner did not have GBS, transverse myelitis, central nervous system or peripheral nervous system disorder, polyneuropathy, or any neurological disorder that H_1N_1 vaccine caused. Med. recs. Ex. 1, at 10.

On January 9, 2012, petitioner saw Dr. Daniel Becker, a neurologist, at the Johns Hopkins transverse myelitis clinic. Med. recs. Ex. 24, at 3. He stated petitioner did not have

transverse myelitis. Her spinal MRI about five days after the onset of her symptoms was normal. Her lumbar puncture showed a normal protein count. She had no evidence of inflammation. On lower extremity testing, she either had no activation or a trace activation of her leg muscles, yet she was able to ambulate unassisted. Id. at 4, 5.

On May 1, 2012, Dr. Richard Bird, petitioner's neurologist, did a nerve conduction study and EMG on petitioner's right and left legs. Med. recs. Ex. 37, at 1. Petitioner had mild acute denervation bilaterally in the gastrocnemius (calf) and abductor halluces (inner portion of the feet) muscles with slight irritation in the L5 paraspinal muscles. Dr. Bird thought petitioner had a possible radiculopathy (which would be consistent with Dr. Gerecke's conclusion on March 14, 2011).

From September 24-28, 2012, petitioner was at Georgetown Hospital under the care of Dr. Carlo Tornatore, her neurologist who testified at the hearing. He noted that her brain MRI of July 12, 2012 showed demyelinating lesions and that her EMG was concerning for chronic inflammatory demyelinating polyneuropathy ("CIDP"). Med. recs. Ex. 25, at 4.

On October 15, 2012, Dr. Bird noted that Dr. Tornatore had made a tentative diagnosis of ADEM with possible CIDP. Id. at 4. Petitioner's follow-up brain MRI revealed a few more scattered white periventricular lesions, particularly in the posterior hemispheres with one small hemosiderin deposit off the centrum semiovale which was not seen before. Dr. Bird wrote that petitioner had no evidence before of prior hemangiomas in this area even though she has had a few in her spine. Dr. Bird noted this might be consistent with early development of amyloid angiopathy. He wrote Dr. Tornatore thinks it is consistent with ADEM, but Dr. Bird stated he was not certain that is the correct diagnosis. He added that petitioner has had consistently normal nerve conduction studies and "I do not think that she had CIDP although she might have had some kind of postinflammatory radiculopathy at some point as she has continued to have positive EMG abnormalities in her gastrocnemius." Id.

On March 22, 2013, Dr. Tornatore testified in favor of petitioner's workers' compensation claim. Ex. 57. He testified that petitioner had GBS in October 2009 and components of CIDP. Id. at 6. He also testified that the first flu vaccination (the trivalent flu vaccine given September 15, 2009) primed petitioner's immune system, and the second flu vaccination (the H_1N_1 monovalent flu vaccine) caused an acute onset of GBS. Id. at 8, 15.

On April 1, 2013, Dr. Sellman wrote a letter saying petitioner was on Social Security disability and remained very weak and was homebound. Med. recs. Ex. 65, at 1. Dr. Sellman opined that petitioner did not have a disorder of the peripheral nervous system. Id. at 4, 6.

On April 25, 2013, petitioner and her employer settled her workers' compensation claim for $60,000.00. Ex. 54, at 30.

On July 16, 2013, Dr. William Reid did a brain MRI, which showed areas of abnormal, increased signal intensity on flair and T2-weighted images in the periventricular and subcortical

regions unchanged from the prior brain MRI done on July 2, 2012. Med. recs. Ex. 60, at 1.

On May 26, 2015, Dr. Robert L. Kane, a psychologist, performed an evaluation of petitioner. Med. recs. Ex. 105, at 1. After administering various tests, he concluded that she was not an individual who deliberately engaged in symptom exaggeration. Id. at 3.

TESTIMONY

Dr. Carlo Tornatore testified first for petitioner. Tr. at 5. He is Chairman of the Department of Neurology at Georgetown University Hospital. Id. During his six years at the National Institutes of Health as a post-doctoral fellow, he did molecular neuroimmunology and neurovirology, looking at how the immune system and viruses interact in the nervous system. Id. at 6. Dr. Tornatore is also Chairman of the Clinical Department of Neurology at MedStar. Id. at 7. That means he is the neurologist and chief for the neurology departments at Georgetown Hospital, Washington Hospital Center, Montgomery General Hospital, and other hospitals, for a total of nine hospitals. Id. Georgetown has two basic science laboratories looking at remyelination in the nervous system and trying to understand the pathogenesis of different diseases in the nervous system. Id. at 8. Georgetown has strong collaboration with the neuroimmunology branch at the National Institutes of Health. Id. Georgetown's residency program has combined with NIH into a joint residency program. Id.

Dr. Tornatore has a very wide perspective on petitioner's case. Id. at 15. Prior to petitioner receiving H_1N_1 vaccine On October 23, 2009, she was doing well. Id. at 16. She had no significant issues. She was walking between 12,000 and 15,000 steps per day over a five-week period. She had some minor medical problems. She was working and not seeing physicians frequently. On October 23, 2009, everything changed and this was striking to Dr. Tornatore. Id. The vaccine stimulated her immune system. Id. at 17. Looking back to September 15, 2009, six weeks before, petitioner received the seasonal flu vaccine. Id. at 18. Dr. Tornatore stated these are not two independent events. Rather they are related. The seasonal flu vaccine is inactivated and acts against H_3N_2 epitopes of the seasonal flu. H_1N_1 is inactivated and acts against the H_1N_1. They look like two different sets of antigens to which the vaccinee is exposed. Dr. Tornatore referred to petitioner's exhibit 126, an article entitled Pandemic H1N1 influenza vaccine induces a recall response in humans that favors broadly cross-reactive memory B cells.[3] Id.

The authors of exhibit 126 asked if immunization with H_1N_1 vaccine could develop an immune response against other types of influenza virus that do not have the H_1N_1 serotype. Id.

[3] G-M Li, et al., Pandemic H1N1 influenza vaccine induces a recall response in humans that favors broadly cross-reactive memory B cells, 109 PNAS 23:9047-52 (2012). Ex. 126. The authors state that the 2009 H_1N_1 flu vaccine "induces a broadly cross-reactive antibody response." Id. at 9049-50. "[I]mmunization with a dramatically altered [hemagglutinin] induces a recall response in humans that favors broadly cross-reactive memory B cells." Id. at 9050. "[T]he response to the [pandemic] H_1N_1 2009 vaccine arises from preexisting memory B cells. This was supported by the presence of memory B cells specific for [pandemic] $H1N_1$ 2009 [hemagglutinin] in individuals even before the emergence of the new virus, strongly implying they were induced by exposure to previous [flu] strains." Id. at 9051.

at 19. The answer is yes. The vaccinee will develop an immune response against H_3 and H_5. This result leads the authors of exhibit 126 to surmise that with just one vaccine, maybe even H_1N_1, doctors can immunize people against a variety of flu viruses. Id. at 18-19.

This is relevant to petitioner's case because she received H_3N_2 seasonal flu vaccine six weeks prior to receiving H_1N_1 flu vaccine. Id. at 19. When she received H_1N_1 vaccine, she had an anamnestic response to the earlier H_3N_2 flu vaccine because her immune system was already primed against H_3N_2. When she received H_1N_1 flu vaccine, the H_3N_2 plasmablasts started up again. Id. Had petitioner not had the H_3N_2 seasonal flu vaccine but only the H_1N_1 vaccine, Dr. Tornatore stated her plasmablasts would have been stimulated only against H_1N_1. But she had the H_3N_2 seasonal flu vaccine and, when she got the H_1N_1 vaccine, it activated the H_3N_2 even further. Id. at 21. In the article that is exhibit 126, serum from someone vaccinated with H_1N_1 had an immune response against H_3N_2. Id. at 22. Without having received the seasonal flu vaccine before the H_1N_1 vaccine, petitioner would not have had the symptoms she had. Id.

Plasmablasts are white blood cells that produce antibodies. Id. at 24. In order to produce antibodies, they have to be stimulated. To Dr. Tornatore, the speed of petitioner's medical decline after receiving H_1N_1 vaccine is quite striking because one would not expect an immune response to occur that quickly. Id.

Dr. Tornatore called petitioner's case complex. Id. at 26. He did not disrespect petitioner's other treating doctors who denied she had a neurologic illness or was sick at all. Id. He stated petitioner had objective symptoms that numerous different doctors noted which involved both the peripheral and autonomic nervous systems, symptoms she did not have pre-vaccination. Id. at 26-27. These were not trivial symptoms. Id. at 27. After petitioner had H_1N_1 flu vaccine, she had right-sided facial palsy. Her personal care physician Dr. Joan Smith notes there was evidence of motor, sensory, and lateralizing deficit on the left side of petitioner's face with occasional slurring of words. Id. Dr. Smith concludes petitioner had a GBS-like syndrome. Id. at 27-28.

Several days later, petitioner sees Dr. Preet Yonker, a neurologist, who also makes some very striking observations in petitioner's physical examination. Id. at 28. Petitioner had decreased sensation bilaterally in the second division of the fifth cranial nerve, meaning numbness. Dr. Preet also notes petitioner had bilateral peripheral seventh nerve weakness, which controls the strength of the face. Thus, whereas Dr. Smith saw weakness on the left side of petitioner's face, Dr. Yonker notices weakness on both sides of petitioner's face. Dr. Yonker notes bilateral, nasolabial-fold flattening. Id. That flattening is evidence of weakness. There are only three causes of bilateral weakness of the facial nerve: Lyme disease, sarcoidosis, or GBS. Id. at 29. Petitioner does not have Lyme or sarcoidosis. Dr. Preet also found weakness in all muscle groups of the legs: the bilateral iliopsoas, the quadriceps, the hamstrings, the gastrocnemius, and the tibialis anterior. Dr. Yonker diagnosed petitioner with acute inflammatory demyelinating polyneuropathy/GBS post-vaccination. Id. Petitioner's facial weakness was so significant that she could not close her eyes completely. Id. at 30. Dr. Tornatore said petitioner could not have faked these symptoms. Id.

Petitioner next went to Atlantic General Hospital where the doctors found bilateral lower extremity weakness, but not facial palsy. Id. Petitioner's physical examination changed over the course of the next several years. Id. Facial weakness later became twitching on the right side of her face. Id. at 30-31. Then petitioner went to Johns Hopkins where the doctors found bilateral weakness in the legs, but giveaway weakness. Id. at 31-33. Dr. Tornatore does not equate giveaway weakness with conversion disorder. Id. at 33. All he can say is that petitioner did not give her full effort when she underwent physical examination. Id. However, a physiatrist felt her weakness was significant enough that she should undergo physical therapy. Id. at 33-34.

Petitioner also had urinary retention. Id. at 34. A catheter inserted in her bladder produced 1,000 ml of urine, which is an astounding amount. Petitioner did not have a urinary tract infection. Dr. Tornatore stated he has patients with urinary tract infections and none of them develops acute urinary retention. In fact, they urinate constantly. Id. Petitioner had to self-catheterize for several days. Id. at 35. Petitioner could not fake urinary retention. That is the part of the nervous system called the autonomic nervous system.

Several years later, petitioner developed another autonomic deficit, this time in her pupil, called the Adie's pupil. Id. When petitioner saw her primary care physician, Dr. Walsh, on November 17, 2009, petitioner had no ankle reflexes and had weakness in her legs, besides left foot drop. Id. at 36. Dr. Walsh concludes this is post-vaccination polyneuropathy. Id. at 36. Petitioner sees Dr. Yonker, the neurologist, who finds weakness in petitioner's upper and lower extremities plus nasolabial fold flattening. Id. at 37. Dr. Tornatore thinks petitioner had fluctuating symptoms. Id. He attributes the fluctuation to antibodies binding to pores, thus impeding sodium and potassium moving across the membranes. But then the antibodies fall off. Id. at 38-41. This would also explain the variable results of petitioner's EMGs. Id. at 42-43.

Dr. Tornatore testified that petitioner's examination never reverted to normal. Id. at 43. On January 10, 2010, two months after vaccination, petitioner saw Dr. Bird, another neurologist, who also finds her lower extremities weak. He finds sensory loss and absent ankle reflexes. Dr. Bird diagnosed petitioner with GBS or GBS-like symptoms following her H_1N_1 vaccination. He thought this might not be classical GBS because of the quick timing after vaccination, but maybe a toxic reaction. Id. Dr. Tornatore notes that most of petitioner's doctors were unaware that petitioner had a trivalent flu vaccination before her H_1N_1 vaccination. Id. at 44. Dr. Yonker did an EMG whose results were normal. On February 9, 2010, Dr. Anderson does an EMG whose results are abnormal. Id. The results are consistent with demyelinating polyneuropathy. Id. at 45. On examination, Dr. Anderson found weakness and concludes that petitioner has acute inflammatory demyelinating polyneuropathy. In May 2010, petitioner sees Dr. Bird again, and he finds weakness, sensory loss, and absent reflexes, and he starts to think this might be transverse myelitis. Id.

Dr. Michael Sellman saw petitioner on May 27, 2010 and he found she had normal reflexes except for absent ankle reflexes. Id. at 47. She also had sensory loss in her legs. Id. Petitioner had an EMG/nerve conduction study at the University of Maryland whose results were

normal. Id. at 54. On physical examination, petitioner had poor effort and giveaway weakness. Id. at 55.

In July 2013, petitioner reported blinding light sensitivity and blurred vision with bright light. Id. at 56. The ophthalmologist finds Adie's pupil in her left eye. That means that her pupil did not constrict in bright light, which is a problem with the autonomic nervous system. Dr. Tornatore said this is an objective neurologic finding that was new. Id. An autoimmune event such as acute inflammatory demyelinating polyneuropathy and chronic inflammatory demyelinating polyneuropathy can involve the autonomic nervous system. Id. at 57. Dr. Tornatore has been petitioner's treating neurologist for five years. He has treated her for neuropathy and she has slowly improved. Id. He thinks she had an AIDP-like presentation which lingered and became CIDP, noting how in Dr. Peter Dyck's book The Peripheral Nervous System, the division between them has changed over time. Id. at 59-60.

In sum, Dr. Tornatore believes the trivalent flu vaccination administered in September 2009 stimulated petitioner's immune system. Id. at 63. Six weeks later, petitioner received H_1N_1 flu vaccine which further activated the trivalent flu plasmablasts, resulting in precipitous symptoms. The antigens in the vaccine shared homology with epitopes on the nervous system, causing cross-reactivity, otherwise known as molecular mimicry. Id.

Dr. Tornatore continues to treat petitioner with IVIG. Id. at 83. He admitted on cross-examination that petitioner would not meet the Brighton criteria for GBS or CIDP. Id. at 97. An antibody-mediated event could be quickly reversible. Id. at 107. If petitioner had not had seasonal flu vaccine six weeks before she received H_1N_1 vaccine, Dr. Tornatore said her onset of symptoms after H_1N_1 vaccine would have seemed very fast. Id. at 115. But she did have seasonal flu vaccine and a time frame of six weeks fits very well with an anamnestic response after the H_1N_1 vaccination. He feels H_1N_1 vaccine by itself was less probably the sole cause of petitioner's symptoms. Id. Dr. Tornatore thinks it interesting that a number of petitioner's doctors felt that H_1N_1 could cause injury. Id. at 116. Dr. Tornatore said that petitioner did not seem to him to be someone with a fictitious disorder or with other psychiatric issues. Id. at 129.

Dr. Vinay Chaudhry testified for respondent. Id. at 132. He is a professor of neurology at Johns Hopkins University and co-runs the EMG laboratory. Id. at 133. He is board-certified in neurology, neuromuscular diseases, electrodiagnostic medicine, and clinical neurophysiology. Id. at 133-34. Besides seeing patients with neuromuscular diseases, he helps other doctors diagnose their patients by performing EMGs. Id. at 134. He gives lectures on GBS and CIDP to medical students, residents, fellows, and colleagues on national fora. Id. at 137.

Dr. Chaudhry testified that AIDP is a form of GBS. Id. at 138. There are three other forms of GBS: AMAN, AMSAN, and Fisher syndrome. Id. at 140. They peak within less than four weeks and all recover with time. Id. Of these four forms of GBS, only AMAN involves molecular mimicry. Id. at 141. Dr. Chaudhry has never met petitioner. Id. His opinion is that petitioner did not have GBS or AIDP or any other form of GBS. Id. at 142. His first reason is that GBS of any kind must peak within four weeks. Someone cannot have a fluctuating

weakness continuing for years and diagnose it as GBS. The reflexes must be gone and not be brisk. Dr. Chaudhry follows the Brighton criteria for GBS. Id. Petitioner met the criterion of bilateral flaccid weakness of her lower extremities, but she did not have decreased or absent reflexes. Id. at 143-44. Her course was not monophasic and she continued to have other findings after 28 days. Id. at 144. In addition, petitioner's electrophysiologic findings were not consistent with GBS and she did not have spinal fluid results consistent with GBS. Id. Generally, GBS requires progressive weakness in both arms and legs in an ascending pattern. Id. at 145. Any bladder or bowel dysfunction at onset should raise doubts about a GBS diagnosis. Petitioner had one lumbar puncture and the result was normal spinal fluid. Id. She had seven EMGs of which six were normal. Id. at 146. Dr. Chaudhry thinks there was a lack of temperature control for the one abnormal EMG. Id. Twelve different doctors at six or seven different facilities said she did not have GBS. Id. at 155. Nerve conduction studies are critical to a diagnosis of GBS. Id. at 158. Petitioner had a skin biopsy done whose result was normal. Id. at 159. That makes less likely that the smaller, unmyelinated fibers and autonomic nerves were affected. In GBS, the myelinated fibers are more affected. Id.

Dr. Chaudhry said that sometimes reflexes can be present initially and then go away, but there should be other features to GBS, i.e., monophasic course, abnormal spinal fluid and EMG, before you treat with IVIG. Id. at 161. Only petitioner's ankle jerks were reduced, but she retained her other reflexes and sometimes they were brisk. Id. at 163. For petitioner to have CIDP, she also needs abnormal reflexes and EMG results. Id. Dr. Chaudhry finds it hard to call petitioner's condition a neuropathy because peripheral neuropathy causes numbness and weakness generally in a stocking-glove pattern, distal to proximal. Id. at 164. He doubts petitioner has CIDP. Id. at 165. Even if the disease is chronic and there is some numbness and weakness, the patient needs to have localization to the nerves either clinically (diminished or absent reflexes) or physiologically (abnormal EMG or skin biopsy). Id. at 166. Petitioner's treating doctors other than Dr. Tornatore did not diagnose her with CIDP. Id. at 167. Dr. Chaudhry does not accept Dr. Tornatore's channelopathy explanation for petitioner's fluctuating symptoms. Id. at 168-69. Nerves do not jump back and forth like that. Id. at 169. Dr. Chaudhry has never seen fluctuating symptoms in any of his GBS/CIDP patients without medical intervention. Id. at 170-71. But with medical intervention, the patient would not then get worse. Id. at 171.

Dr. Chaudhry does not think petitioner's urinary retention was neurological. Id. Neurological urinary retention is not transient. Her doctors attributed it to a urinary tract infection. Id. None of her other doctors attributed her urinary retention to an autonomic neuropathy. Id. at 172. Autonomic neuropathy usually presents with lightheadedness when someone stands, blood pressure going down, tachycardia, fluctuating blood pressure, and gastrointestinal symptoms. Adie's pupil is a totally separate issue. He does not know to what to attribute petitioner's Adie's pupil, but it did not have to be an autonomic neuropathy. It was a transient symptom. Id. Autonomic neuropathies present with blurred vision, not with Adie's pupil. Id. at 172. Most of the relapsing and remitting neuropathies occur over weeks and months, related more to treatment. Relapsing and remitting does not occur over days, hours, or weeks. Id. A lot of people have Adie's pupil because they inherited it. Id. at 174-75. It has

nothing to do with autonomic neuropathy. Id. at 175.

Dr. Chaudhry does not accept Dr. Tornatore's analysis of other treating doctors' records because Dr. Tornatore emphasizes the symptoms but disregards the doctors' assessments, whereas Dr. Chaudhry thinks the assessments are the most valuable part of the record. Id. at 177. Dr. Chaudhry thinks that neither petitioner's seasonal flu vaccination nor her H_1N_1 vaccination caused either GBS or CIDP because petitioner did not have these diseases. Id. at 179. He does not think the vaccines acting together changes his mind. Id. Petitioner had a set of symptoms without a diagnosis. Id. at 179-80. He does not know if her set of symptoms is neurological. Id. at 180. He needs to know a diagnosis. Id. at 181. These symptoms do not involve the peripheral nerves. Id. Dr. Chaudhry said he does not diagnose anyone based on symptoms. Id. at 182. He has to know where the lesion is. Id. at 183. None of the physicians localized it to the peripheral nerve. Id. at 183-84.

Dr. Chaudhry said that autonomic dysfunction is much more common in GBS than in CIDP, and it mostly (more than 50 percent) involves the heart. Id. at 187. Autonomic dysfunction could be central or peripheral in terms of the nervous system involved. Id. But autonomic dysfunction should not be transient if it is connected to a central or peripheral nervous system disorder. Id. at 188. Instead of picking up individual symptoms here and there, Dr. Chaudhry said he looks at the entire picture of the clinical symptoms, the signs, the EMG, and the laboratory data, and concludes that it is hard to diagnose this as an autonomic neuropathy, although it could be. Id.

Dr. Chaudhry said he was familiar with the terms "lumpers" and "splitters." Id. at 189. He thinks that when one does not know a disease pattern or pathogenesis, it is okay to lump all the types of GBS together. Id. at 190. But when one knows the pathogenesis is different, then it is better to be a splitter for treatment. Id. If he had to choose, he would say he is a splitter. Id. at 191-92. If someone does not know what the condition is, it is hard to know the cause. Id. at 192.

On cross-examination, Dr. Chaudhry admitted that someone could have GBS that looks acute at first and then becomes chronic. Id. There is both an acute onset CIDP and a chronic CIDP that is slowly progressive. Id. None of petitioner's other treating doctors knew she had had two flu vaccinations. Id. at 205.

On rebuttal, Dr. Tornatore said that petitioner's Adie's pupil was not transient. Id. at 218. When Dr. Tornatore saw petitioner May 4, 2016, it was two and one-half years after she was diagnosed with Adie's pupil and she still had it. Id. at 218-19. Dr. Tornatore testified that Dr. Chaudhry's emphasis on AMAN having a very rapid response to IVIG treatment supports Dr. Tornatore's testimony on fluctuation of symptoms pertaining to channelopathy because the rapidity of an AMAN patient's recovery would be too soon for remyelination. Id. at 219-20.

DISCUSSION

To satisfy her burden of proving causation in fact, petitioner must prove by preponderant

evidence: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." Althen v. Sec'y of HHS, 418 F.3d 1274, 1278 (Fed. Cir. 2005). In Althen, the Federal Circuit quoted its opinion in Grant v. Secretary of Health and Human Services, 956 F.2d 1144, 1148 (Fed. Cir. 1992):

> A persuasive medical theory is demonstrated by "proof of a logical sequence of cause of and effect showing that the vaccination was the reason for the injury [,]" the logical sequence being supported by a "reputable medical or scientific explanation[,]" i.e., "evidence in the form of scientific studies or expert medical testimony[.]"

418 F.3d at 1278.

Without more, "evidence showing an absence of other causes does not meet petitioner's affirmative duty to show actual or legal causation." Grant, 956 F.2d at 1149. Mere temporal association is not sufficient to prove causation in fact. Id. at 1148.

Petitioner must show not only that but for her trivalent flu vaccination, she would not have her neurological illness, but also that her trivalent flu vaccination was a substantial factor in causing her neurological illness. Shyface v. Sec'y of HHS, 165 F.3d 1344, 1352 (Fed. Cir. 1999). An added question is whether her H_1N_1 monovalent flu vaccination was a substantial factor in causing her neurological illness.

The first problem is that many neurologists have tested petitioner and found her to be normal. But others, including Dr. Tornatore, have found her to have neurological problems. Respondent's expert Dr. Chaudhry based his opinion that petitioner did not have any neurological illness on the reports and testing of neurologists who said she was normal and who also suggested she had conversion disorder. Dr. Tornatore reached the opposite conclusion. Close calls are to be resolved in favor of petitioners. Capizzano, 1440 F.3d at 1327; Althen, 418 F.3d at 1280.

Dr. Tornatore has been petitioner's treating neurologist for the last five years. The Federal Circuit in Capizzano emphasized that the special masters are to evaluate seriously the opinions of petitioner's treating doctors since "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." 440 F.3d at 1326. See also Broekelschen v. Sec'y of HHS, 618 F.3d 1339, 1347 (Fed. Cir. 2010); Andreu v. Sec'y of HHS, 569 F.3d 1367, 1375 (Fed. Cir. 2009). Balancing this one treating neurologist, i.e., Dr. Tornatore, versus numerous other treating doctors has been a challenge, but the undersigned made a provisional holding on the record, with the understanding that respondent's expert Dr. Chaudhry would respond in writing to late submissions of petitioner and petitioner's expert Dr. Tornatore would reply to respondent's expert's response.

At the end of the hearing, the undersigned stated the following:

> Before I walked into court this morning, I thought this is not a case that's going to succeed because there is so much ambiguity about what it is that Ms. Thompson had. . . .
>
> In my experience here, however, and what I heard from Dr. Tornatore changed my mind completely. . . . I have had cases in which somebody just doesn't fit into neat categories. They're causation [in] fact cases obviously. And what is the big deal?
>
> If it's causation [in] fact, and if the petitioner satisfies the three [Althen] criteria to prove causation [in] fact, then petitioner can have little pieces of all kinds of problems. In the Dunbar[4] case, . . .although the experts there were immunologists, not neurologists, . . . [petitioner] had so many different things, and it was the immunologists that tied it together. And I ruled for petitioner then.
>
> If I believe the theory underlying it, then you've made good progress in bringing that case home for the petitioner. Yes, it was disturbing to me to read doctor after doctor after doctor saying she doesn't have GBS, or she doesn't have – I don't even know if anybody said she didn't have CIDP, but she has no peripheral neuropathy, no this, no that, normal this, normal that. What could be wrong with her?
>
> And yet we have – and I wanted to – nobody brought this up, but we have a report, which is unusual in these kinds of cases. Usually, you don't have somebody giving a negative report. And this is Dr. Kane's report. This is Exhibit 105, and his CV is 106. And he's the psychologist. And he examined and listened to and talked to Ms. – he calls her Payne,[5] but it's Thompson, and he concludes – this is Exhibit 105, he gave her the MMPI, and the result of that test – it's actually MMPI-2-RF, did not indicate that she is someone who over-reports symptoms, that is exaggerates symptomatology.
>
> Also on page 3 of Exhibit [10]5, he says, taken together, and he talks about all the things he had done, Mrs. Payne's response patterns on different tests, instruments, were not indicative of an individual who was deliberately engaging in symptom exaggeration. And the final page, which is page 4, she [has] no known previous psychiatric history; she's functioned fine prior to obtaining the flu vaccine; and test data do not support malingering or deliberate noncredible reporting of symptoms. And he

[4] Dunbar v. Sec'y of HHS, No. 98-627V, 2007 WL 2844826 (Fed. Cl. Spec. Mstr. Sept. 14, 2007) (petitioner alleged that hepatitis B vaccine caused him a lupus-like reaction with demyelinating disease/vasculitis).

[5] Petitioner filed her petition under the name of Payne. On April 11, 2017, the undersigned issued an Order changing the caption to Thompson.

concludes she did not evidence symptom over-reporting that would support the explanation of deliberate exaggeration and malingering as the reason for her persistent symptoms.

So when I weigh that with Dr. Tornatore's assessment that . . . we have specific symptoms that cannot be manufactured. We've got the weakness in her face initially and the nasolabial folds of flattening because there's some kind of . . . muscular paralysis there, and then she started out with one-sided facial weakness, and then she had the urinary retention, and she had no ankle reflexes and she had foot drop. [W]hether this . . . giveaway weakness or whatever the reason might be for some of the test results, there's enough symptoms there that convey to me that these are actually real symptoms. Whatever the significance is, they're real symptoms.

So then the question is what do we have. And I was very taken with – and, of course, you would be commenting on this, no doubt, with Exhibit 126,[6] which is the discussion of the effect of receiving H_1N_1 flu vaccine because it not only protects you against that particular type of bird flu but also against five other types of flu strains, which are not in H_1N_1. And that's on page 4 of Exhibit 126.

The pandemic, which is the H_1N_1, of course that doesn't count, so it's against five. There's an H_1N_1 that's related to A/Fort Monmo[u]th number 247. There's another H_1N_1 that [is] A[/]Solomon Islands. There's an A[/]New Caledonia. There's an A[/]Brisbane, which is the H_3N_2 because it's not H_1N_1.

And [Dr. Tornatore's] thesis, his theory that the trivalent [flu] vaccine, which is on the [Vaccine Injury] [T]able, in September 2009, which contained the H_3N_2 provoked, as it should, antibody response to that particular viral strain. And when [petitioner] received H_1N_1 six weeks later in October 2009, it then stimulated her again to the same viral strain, H_3N_2, and the rapidity of her symptoms is explained only by that being a trigger.

And it's because we talk about this, and I did talk about this in the prehearing conference, she could recover [damages] if the . . . trivalent vaccination in September 2009 was [a] substantial factor causing her illness. There could be another noncompensible substantial factor in which this case is H_1N_1, which is also substantial, but as long as the vaccine that is covered by the Vaccine Act is a substantial factor, then she's entitled to compensation.

And *Shyface* is a Federal Circuit decision, [in] which [the

[6] G-M Li, et al., Pandemic H1N1 influenza vaccine induces a recall response in humans that favors broadly cross-reactive memory B cells, 109 PNAS 23:9047-52 (2012).

vaccinee] was facing an infection – [E.] coli and DPT vaccine. DPT vaccine was held to be a substantial factor by the Federal Circuit. In that case, both the [E.] coli infection and the DPT were 50 percent each liable or responsible for causing this poor baby's high fever, which led to his death.

But the Federal Circuit said in *Shyface* that it isn't necessary for the vaccine that is on the [V]accine [I]njury [T]able to be the predominant factor as long [as] it's a substantial factor, without which she would not have been afflicted with whatever illness she had. . . . And, so, [Dr. Tornatore] said if [petitioner] had not been exposed prior to the [H_1N_1 vaccination] to the H_3N_2 [in the trivalent flu vaccine] that anamnestic response would not have occurred because there wouldn't have been that double H_3N_2 provoking of the immune system. That theory I find credible.

And then the timing, when you look at this as someone who's already primed to respond to the H_3N_2 [viral strain] of one day [it] is appropriate. And I had Dr. Tornatore in a case that Mr. Homer brought ten years ago also, . . . *Augustynski*,[7] . . . in which the individual had hepatitis [B] vaccine 30 days earlier – or maybe more than that – and then 30 days later he had another hepatitis B vaccine, and of course they're made the same way. There's total identity.

And then [Augustynski] had the onset of transverse myelitis one day later [after vaccination], which ultimately turned into multiple sclerosis. And I held for petitioner, based upon Dr. Tornatore's testimony that the prior hep B vaccine had primed [Augustynski] to respond more quickly after the second – the hepatitis B vaccine that we were dealing with, which was only one day away from the onset of symptoms. So that doesn't bother me, which would satisfy the third criterion [of *Althen*].

… I think everything that Dr. Tornatore said makes sense, that – and, also, I'm not unaware that the treatment that he devised for [petitioner], which is the IVIg, which is suitable for those with autoimmune diseases, is working because he's been monitoring her for five years.

And we know from the Federal Circuit's decision in *Capizzano* that the special masters are to take seriously the opinions of treating doctors. Here, I've got many treating doctors who say there's nothing wrong with Ms. Thompson or I don't know what's wrong with her, but it's not peripheral neuropathy, versus some other treating doctor, which is Dr. Tornatore. So I have to weigh one side of treating doctors with the other side of the treating doctor, who happens to be treating her successfully.

[7] Augustynski v. Sec'y of HHS, No. 99-611V, 2007 WL 3033614 (Fed. Cl. Spec. Mstr. Sept. 28, 2007).

> And since she doesn't have conversion disorder according to the psychologist, Kane, and I have no reason to believe that she does because the symptoms that she has manifested, which have not been explained necessarily by many of the treating doctors, are not those that can be faked. You can't fake the pupillary response with the Adie's pupil; you can't fake the paralysis in the face, which makes the nasolabial – the wrinkles that women try and get rid of [to] get flatter.
>
> You can't fake the foot drop, or maybe you could, but there's testing that can be done. The ankle reflex can't be faked. You can't fake the urinary retention of a thousand milliliters of urine. There's an awful lot here that is not actually being treated, if you will, seriously, because nobody can figure out what it is.
>
> And that's where the lumper versus splitter comes in. I think Dr. Tornatore is a lumper. I think that's appropriate. I found it very interesting that . . . over time, Dr. Dyck's[8] distinction between acute inflammatory demyelinating polyneuropathy and chronic inflammatory demyelinating polyneuropathy is not so distinct because you've got the mix and match, and I've seen it in other cases.

Transcript at 227-34.

On July 28, 2017, respondent filed respondent's expert Dr. Chaudhry's supplemental report disagreeing with petitioner's expert Dr. Tornatore's interpretation of Ex. 126.[9] Ex. L, at 2. Dr. Chaudhry wrote the article does not have any example of an exaggerated response after seasonal flu vaccine and the peak response to H_1N_1 occurs at day 7 and returns to background level by day 14. Dr. Chaudhry's view was that petitioner's prior flu vaccination on September 15, 2009 and her subsequent H_1N_1 vaccination on October 23, 2009 would have reduced, not exaggerated, her response. Id.

On August 28, 2017, petitioner filed petitioner's expert Dr. Tornatore's supplemental report disagreeing with Dr. Chaudhry, stating that petitioner was first immunized with an H_3N_2 vaccination, followed six weeks later with an H_1N_1 vaccination. Ex. 131, at 1. The H_1N_1 vaccine resulted in an anamnestic activation of H_3N_2 plasmablasts, an idea that exhibit 126 demonstrates. Id. Dr. Tornatore states Dr. Chaudhry concedes that this phenomenon can happen though infrequently. To Dr. Tornatore, "The fact that it can happen at all is the salient point." Id.

To summarize, petitioner satisfied Althen prong one by proving that trivalent flu vaccine can cause GBS and CIDP with the stimulant boost that H_1N_1 monovalent vaccine provided. The

[8] Dr. Peter J. Dyck is co-editor with P.K. Thomas of the textbook Peripheral Neuropathy (4th ed. 2005).

[9] Pandemic H1N1 influenza vaccine induces a recall response in humans that favors broadly cross-reactive memory B cells, by G-M Lei, et al., 109 PNAS 23:9047-52 (2012).

undersigned holds that both vaccines were substantial factors in causing petitioner's illnesses. Without having received the seasonal flu vaccination, petitioner would not have had neurological illness because she would not have had the plasmablast anamnestic response to H_3N_2 flu virus with solely receipt of H_1N_1 vaccine. Petitioner satisfied Althen prong two by proving that there was a logical sequence of cause and effect in trivalent flu vaccine causing her GBS and CIDP through the trigger effect of the H_1N_1 monovalent flu vaccine. Petitioner satisfied Althen prong three by proving that rapid onset of ADEM and CIDP one day after the trigger H_1N_1 vaccine can occur within six weeks of the Table seasonal flu vaccination and be an appropriate interval to convey causation in fact.

The undersigned rules in favor of petitioner on entitlement. This case is now in damages

IT IS SO ORDERED.

Dated: October 4, 2017

/s/ Laura D. Millman
Laura D. Millman
Special Master