In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. [redacted]V
October 16, 2008
Reissued redacted: October 31, 2008
To be Published

JANE DOE/23,	*	
	*	
Petitioner,	*	
	*	
v.	*	Entitlement; hepatitis B vaccine;
	*	visual difficulties one month
SECRETARY OF THE DEPARTMENT OF HEALTH AND HUMAN SERVICES,	*	later and MS diagnosis three months later
	*	
Respondent.	*	

Clifford J. Shoemaker, Vienna, VA, for petitioner.
Richard F. Topping, Washington, DC, for respondent.

MILLMAN, Special Master

RULING ON ENTITLEMENT[1]

[1] 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 e a clearly unwarranted invasion of privacy. When such a decision or designated substantive order is filed, petitioner has 14 days to identify and move to delete 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 delete such material from public access. On October 30, 2008, petitioner moved to redact her name from the decision. The undersigned grants her motion.

Petitioner filed a petition on May 17, 1999, under the National Childhood Vaccine Injury Act, 42 U.S.C. §300aa-10 et seq., alleging that she received hepatitis B vaccine on November 12, 1992, December 18, 1992, and May 25, 1993, and experienced an adverse reaction.

Petitioner had optic neuritis and has multiple sclerosis (MS). Onset is the issue of the case. A hearing was held on June 17, 2008. Testifying for petitioner was Dr. Carlo Tornatore. Testifying for respondent was Dr. Aaron E. Miller.

This is one of the cases affected by the undersigned's rulings in the Omnibus proceeding dealing with hepatitis B vaccine and demyelinating diseases such as transverse myelitis (TM), Guillain-Barré syndrome (GBS), chronic inflammatory demyelinating disease (CIDP), and multiple sclerosis (MS). The undersigned held that hepatitis B vaccine can cause these diseases if the onset is between three days and one month based on the Omnibus testimony of petitioners' expert Dr. Vera Byers and respondent's expert Dr. Roland Martin. Stevens v. Secretary of HHS, No. 99-594, 2006 WL 659525, at *12, *15 (Fed. Cl. Feb. 24, 2006).[2]

Recently, in Pecorella v. Secretary of HHS, No. 04-1781V, 2008 WL 4447607 (Fed. Cl. Spec. Mstr. Sept. 17, 2008), the undersigned ruled that an appropriate onset could be up to two months.

FACTS

[2] Stevens v. Secretary of HHS, No. 99-594, 2006 WL 659525 (Fed. Cl. Spec. Mstr. Feb. 24, 2006) (hepatitis B vaccine caused TM; onset was12 or 13 days after first vaccination with recovery; onset of TM was one week after second vaccination); Gilbert v. Secretary of HHS, No. 04-455V, 2006 WL 1006612 (Fed. Cl. Spec. Mstr. Mar. 30, 2006) (hepatitis B vaccine caused GBS and CIDP; onset was 21 days after second vaccination); Werderitsh v. Secretary of HHS, No. 99-310V, 2006 WL 1672884 (Fed. Cl. Spec. Mstr. May 26, 2006) (hepatitis B vaccine caused MS; onset was one month after second vaccination); Peugh v. Secretary of HHS, No. 99-638V, 2007 WL 1531666 (Fed. Cl. Spec. Mstr. May 8, 2007) (hepatitis B vaccine caused GBS and death; onset of GBS was eight days after fourth vaccination).

Before the Vaccinations

Petitioner was born on March 13, 1960.

On September 18, 1987, petitioner went to St. Elizabeth Hospital Medical Center, complaining of back pain which began the prior day after she was lifting at work. She also had numbness. Med. recs. at Ex. 7, p. 5. Dr. J.P. Brown's impression was thoracic spine strain and he advised petitioner to avoid heavy lifting. Med. recs. at Ex. 7, p. 7. She had some numbness over the right shoulder and leg region but denied weakness in the upper or lower extremities. She was treated in the recent past for episodes of back pain. Back examination revealed minimal spasm amid the thoracic spine, particularly on the right. There were no objective sensory or motor findings. She walked without difficulty. *Id.*

On May 1, 1989, petitioner complained of right knee pain. Med. recs. at Ex. 7, p. 2.

On September 4, 1990, petitioner was diagnosed with right knee tendinitis. *Id.* She went to the Emergency Department of Lafayette Home Hospital. Two years previously, she had fluid behind the right knee but no problems since. She complained of pain for two days behind the right knee which radiated to the thigh and the calf. She had intermittent sharpness with tingling in her right toes. She had no swelling. She had a burning sensation in her foot. She had good range of motion. The diagnosis was tendinitis in the right knee. Med. recs. at Ex. 7, p. 9.

After the Vaccinations

Petitioner received hepatitis B vaccinations on November 12, 1992, December 18, 1992, and May 25, 1993. Med. recs. at Ex. 2, p. 2.

On June 28, 1993, one month after the third hepatitis B vaccination, petitioner saw Dr. Nicholas F. Hrisomalos, a retina specialist, for photopsias[3] and film that she had noted in her right eye over the prior week. Examination showed vitreous floaters in each eye with an annulus[4] appearing on the right. There was no vitreous hemorrhage or pigment. The macula in the right eye had some conspicuous pigmentary changes and drusen.[5] Peripheral retinal examination of the left eye showed peripheral cystoid degeneration but no significant retinal tear, hole, or detachment. In the right eye was a patch of lattice degeneration inferiorly and early developing traction inferiorly temporally. Superiorly, there was another area of light without traction. Dr. Hrisomalos's impression was high myopia with posterior vitreous detachment and some peripheral retinal changes in the right eye. No treatment was recommended. Med. recs. at Ex. 4, p. 4.

On August 2, 1993, petitioner returned to Dr. Hrisomalos, complaining that the film, sparklers, flashing lights, and distorted vision were just the same or worse. Her headaches were less and her vision was still slightly blurred but not worse. She reported some possible changes in her color vision. Angiography was performed which showed a small area in the macula inferior nasally which looked like a small laquer crack. There was no evidence of subretinal

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[3] Photopsia is "an appearance as of sparks or flashes due to retinal irritation." Dorland's Illustrated Medical Dictionary, 30th ed. (2003) at 1431.

[4] An annulus is "a ring or ringlike structure...." Dorland's Illustrated Medical Dictionary, 30th ed. (2003) at 93.

[5] Drusen (German for "bumps") are "hyaline excrescences in Bruch's membrane (lamina basalis choroideae); they usually result from aging, but sometimes occur with pathologic conditions...." Dorland's Illustrated Medical Dictionary, 30th ed. (2003) at 565. Basal lamina of choroid is "the transparent inner layer of the choroid, which is in contact with the pigmented layer of the retina." *Id.* at 993.

neovascularization or other change. He called petitioner and recommended she check her central vision daily and report any distortion, and check her peripheral vision for any sign of retinal detachment. Med. recs. at Ex. 4, p. 3.

On September 21, 1993, at 7:54 p.m., petitioner went to Lafayette Home Hospital Emergency Department, complaining of pain in her medial right leg proximal and distal to her knee, which started at noon that day and was tender to the touch. She was at work and tried to walk the pain out for about 20 minutes. The pain went away and she felt lightheaded as if she would pass out, but she did not. She came to the hospital and felt better. She had a history of phlebitis in her right leg. Her left inner thigh felt like it was on fire. On examination, her gait and squat were normal. There was no heat or redness on either leg. She was diagnosed with a vasovagal[6] episode and leg pain. Med. recs. at Ex. 8, p. 31.

On September 22, 1993, Dr. Anna L. Welch worked petitioner into her schedule because of pain in petitioner's left leg with numbness. She had had trouble with both legs intermittently for years and had a history of phlebitis. She had pain and burning in the medial left thigh. She had shortness of breath the prior night and went to the emergency room. Her deep tendon reflexes were normal. Her left thigh was tender to palpation but not red or warm. Med. recs. at Ex. 6, p. 14.

On September 23, 1993, Dr. Welch noted that petitioner could return to work. *Id.*

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[6] Vasovagal attack is "a transient vascular and neurogenic reaction marked by pallor, nausea, sweating, bradycardia, and rapid fall in arterial blood pressure which, when below a critical level, results in loss of consciousness and characteristic electroencephalographic changes. It is most often evoked by emotional stress associated with fear or pain." Dorland's Illustrated Medical Dictionary, 30th ed. (2003) at 178.

On September 27, 1993, Dr. Welch noted petitioner's legs were numb and her feet cold. She had difficulty ambulating and slid down a hill one week previously on her right buttock. She had no deep tendon reflex on the right. Her left leg was cooler than her right leg. *Id.*

On September 29, 1993, petitioner underwent a left lumbar spinal MRI, which was normal except for mild scoliosis and early dehydration of the L5-S1 intervertebral disc due to age. Med. recs. at Ex. 1, p. 13.

On September 30, 1993, she saw Dr. Michael A. Sermersheim, a neurologist, on referral from Dr. Welch, complaining of clumsiness, numbness, and weakness of her lower extremities for about 10 days. Med. recs. at Ex. 1, p. 17. Initially she felt it more in the left lower extremity, but she very quickly started experiencing the sensation in both lower extremities. *Id.* She denied any recent illnesses. *Id.* She had never experienced this sensation before in her life. She had never had any other transient neurologic deficits, specifically no visual disturbances, numbness and tingling in the upper extremities, or personality changes. She was basically healthy and had no significant health problems in the past. *Id.*

On October 1, 1993, petitioner underwent a nerve conduction study and right peroneal F-wave latency test. Petitioner had complained of 10 days of numbness and tingling in her lower extremities. The study was essentially normal. Dr. Sermersheim stated she had mild delay in the posterior tibial distal latency which was of uncertain clinical significance. The study results did not suggest peripheral neuropathy or Guillain-Barré syndrome. Med. recs. at Ex. 8, p. 27.

On October 14, 1993, petitioner underwent a brain MRI, which was abnormal due to increased T2 signal in the posterior parietal white matter bilaterally and in the medial left brachium pontis. The primary consideration would be MS. Med. recs. at Ex. 1, p. 15.

On October 20, 1993, Dr. Sermersheim wrote a letter to Dr. Welch to update her on petitioner. Petitioner's lumbar puncture and brain MRI were most consistent with the diagnosis of MS. Med. recs. at Ex. 1, p. 3.

On October 13, 1994, petitioner saw Dr. Catherine I. Hatvani who, in reviewing petitioner's history, stated that petitioner had MS with some involvement of the lower extremities and not much involvement of her vision or upper extremities. Med. recs. at Ex. 16, p. 27. She had headaches associated with tension. She had tension and pain in the back of the neck associated with work-related tension. *Id.*

Five years later, on December 17, 1999, petitioner saw Dr. Hamid S. Hamdi, a neurologist, complaining of sudden onset of loss of vision in her left eye, decreased visual acuity, and burning sensations and hyperesthesias in both lower extremities. On examination, Dr. Hamdi did not see any relative afferent pupillary defect. The fundi appeared normal on the left side. The right side showed some atrophy. Petitioner had a patchy loss of sensation in all extremities. Her strength, tone, and deep tendon reflexes were normal. His impression was left optic neuritis. Med. recs. at Ex. 17, p. 10.

Later on December 17, 1999, at Lafayette Home Hospital, Dr. Hamdi wrote a history and physical examination of petitioner, stating that petitioner had a history of MS diagnosed in 1993 when she presented with right optic neuritis from which she had partially recovered. She came to his office, complaining of sudden onset of decreased vision in the left eye which started the day before. Dr. Hamdi saw petitioner in his office that afternoon and she was admitted to Lafayette Home Hospital, Inc. for IV steroid treatment. Petitioner had decreased visual acuity in both eyes

with left pupillary defect. She had pallor in the right fundus and a normal fundus in the left side. He diagnosed petitioner with acute left optic neuritis. Med. recs. at Ex. 17, p. 16.

On January 25, 2000, petitioner underwent another brain MRI which was compared to her prior MRI of October 14, 1993. Dr. Douglas M. Dunco's impression was interval decrease in the left bracium pontis signal abnormality with stable deep white matter foci of signal abnormality in the parietal regions and with new lesions in the centrum semiovale bilaterally, with an acute lesion in the temporooccipital region. The findings were consistent with MS. Med. recs. at Ex. 17, p. 15.

On September 29, 2000, petitioner suffered cardiac arrest and associated hypoxia with memory and other cognitive difficulties. Med. recs. at Ex. 12, p. 24.

Other Submitted Material

Petitioner filed Ex. #22, an article entitled "Retinal Venous Sheathing and the Blood-Retinal Barrier in Multiple Sclerosis," by M.K. Birch, et al., 114 *Arch Ophthalmol* 34-39 (1996). There is a well-recognized association between retinal venous sheathing and MS. *Id.* at 34. The inflammatory nature of some of the retinal venous changes associated with MS has been shown by disruption of the blood-retinal barrier (BRB) in a few patients. *Id.* Intracranial venulitis in white matter in or between demyelinated plaques in MS may be associated with retinal venous sheathing and have a common cause. *Id.*

Petitioner filed Ex. #23, which is an excerpt from a text called Neuro-Ophthalmology. Diagnosis and Management, eds. Liu, et al., "Inflammatory Optic Neuropathies," pp. 128, 129. The authors state, at 128:

> The inflammatory optic neuropathies represent a heterogeneous group of conditions characterized by acute or subacute, often painful vision loss that results from inflammation of the optic nerve. . . . The term *optic neuritis* has come to have an even more specific implication: inflammatory optic neuropathy that accompanies demyelinating disease. . . . These processes may be immune mediated, granulomatous, or infectious. . . . In many patients optic neuritis is the heralding manifestation of multiple sclerosis.

The authors continue, at 128-29:

> Vision loss is rapid in onset, generally being recognized over a period of hours to a few days. . . . Reduced color vision invariably accompanies the vision loss in optic neuritis. . . . Characteristic pain precedes the vision loss by a few days and is present in the majority of patients. . . . Global tenderness and worsening of pain on eye movements are typical. The exact origin of the pain is unknown but presumably pain is the result of the pulling on the dura (in contact with the inflamed nerve) by the eye muscle origin . . . . Phosphenes or flashing lights described by patients with optic neuritis generally take a variety of forms, including lights, sparkles, and shifting squares. . . . These symptoms were present in 30% of patients . . . . Uhthoff's symptom is transient visual obscuration associated with elevation in body temperature. . . . Vision loss generally takes the form of blurring, greying, or reduced color vision . . . . Patients with optic neuritis almost always have some visual field loss.

Petitioner filed Ex. #24, an excerpt from Principles and Practice of Ophthalmology, 2nd ed., Vol. 5, eds. Albert & Jakobiec, pp. 4120-23. The authors list clinical signs of optic neuritis on p. 4120 as: reduced visual acuity at near and far distance, dyschromatopsia (abnormal color vision), impaired contrast sensitivity, reduced stereoacuity, general depression of the visual field, afferent pupillary defect, and hyperemia and acute swelling of the optic disc. Abnormal color vision occurs in patients with acute and recovered optic neuritis. *Id.* The authors state, "Color vision defects are highly sensitive indicators of a previous attack of optic neuritis." *Id.* at 4121.

"In acute optic neuritis, the cardinal field defect is a widespread depression of sensitivity, which is particularly pronounced centrally as a central scotoma." *Id.* at 4123. A unilateral relative afferent pupillary defect is present in 44-76% of patients with acute optic neuritis. *Id.*

Petitioner filed Ex. #25, an article entitled "Retinal Venous Sheathing in Optic Neuritis. Its Significance for the Pathogenesis of Multiple Sclerosis," by S. Lightman, et al., 110 *Brain* 405-14 (1987). The authors found vascular abnormalities and/or cells in the media of one-quarter of patients presenting with their first attack of an apparently isolated optic neuritis which the authors considered predictive in assessing the risk of developing clinically expressed MS. *Id.* at 405-06, 411. They thought it possible that the primary events leading to demyelination occurred at the vascular endothelium. *Id.* at 412.

TESTIMONY

Dr. Carlo Tornatore, a neurologist, testified for petitioner. Tr. at 5. He is the director of the MS Center at Georgetown. Tr. at 7. On June 28, 1993, about a month after petitioner's third hepatitis B vaccination, petitioner had flashing lights or photopsias and a film over her right eye. Tr. at 12. The timing is appropriate for the onset of an autoimmune response to a vaccination. *Id.* Dr. Nicholas F. Hrisomalos wrote that petitioner also had headache, sparkles, and the sensation of looking through a screen. *Id.* She said these symptoms began about a week earlier, putting onset at June 21, 1993. *Id.*

In a follow up visit on August 2, 1993, petitioner reported some possible change in her color vision. Tr. at 13. This would mean color loss. Tr. at 14. Dr. Hrisomalos was a retinal specialist. *Id.* He was looking only at petitioner's retina. *Id.* Petitioner's change in her color vision may have been color loss. *Id.* Dr. Tornatore emphasized four symptoms about which

petitioner complained to Dr. Hrisomalos: flashing lights (sparkles or photopsias), visual obscuration (the screen in front of her eyes), pain (headache), and loss of or changing color. Tr. at 17-18.

Phosphenes or flashing lights are present in 30 percent of patients with optic neuritis. Tr. at 18-19. They could also occur in neuroretinitis. Tr. at 19. Dr. Tornatore thinks that Dr. Hrisomalos did not diagnose petitioner with optic neuritis because, being a retinal specialist, he thought there was something wrong with her retina. *Id.*

To find out if petitioner's symptom of headaches really was pain in her eye, Dr. Tornatore questioned her the day before the hearing. Tr. at 20. But petitioner did not recall where the pain was. *Id.* Dr. Tornatore stated that Dr. Hrisomalos, being a retina specialist, did not describe petitioner's pupils or her optic nerve to say whether they were normal or abnormal in his written notes and diagram. Tr. at 23. He spent a lot of time looking at the periphery of petitioner's retina and found changes which could be old in someone with myopia. *Id.*

Someone with optic neuritis would have an afferent pupillary defect. Tr. at 23-24. Dr. Hrisomalos recognized the complaint of decreased vision and color vision and did a fluorescein angiogram. Tr. at 28. This helps see the blood vessels in the back of the eye. *Id.* Petitioner had a small area in the macular region inferior nasally which seemed like a small laquer crack, i.e., a little break in a blood vessel or small, leaky vessel. Tr. at 29. In optic neuritis, there is some focus leakage out of the blood vessel of the eye. Tr. at 31. Dr. Hrisomalos did not indicate if the leak were in a vein or an artery. *Id.* Leaky blood vessels are well recognized in someone with optic neuritis. *Id.*

Six years after petitioner's first symptoms, Dr. Hamdi at Lafayette Home Hospital notes that petitioner had right optic neuritis in 1993 from which she had partially recovered. Tr. at 33-34. This was after she had been diagnosed with MS. Tr. at 34. She then had problems with her left eye. *Id.* Dr. Hamdi wrote petitioner had right fundal pallor. *Id.* Normally, it is yellowish. *Id.* Pallor is a hallmark of someone with atrophy of the optic nerve. Tr. at 34-35. Optic neuritis, but not retinal detachment, would cause pallor of the optic nerve. Tr. at 35. In Dr. Hamdi's letter of December 17, 1999, he states that petitioner's right fundus showed some atrophy, meaning her right optic nerve had been injured, and there was no other episode of right eye problem other than in 1993. *Id.*

In a follow-up visit to Dr. Hamdi in January 2000, he again notes that petitioner's fundus was normal on the left but atrophied on the right. *Id.* On three separate occasions, Dr. Hamdi noted that petitioner's optic nerve on the right was pale or atrophied and that she had optic neuritis in 1993. *Id.* Petitioner had no other eye problems between 1993 and 2000 so that one could say she had optic neuritis after 1993 (but not in 1993) which was not recognized. Tr. at 37. Seeing pallor in the optic nerve depends on where the inflammation is. Tr. at 38. If the inflammation is further back than the retinal part of the optic nerve, it could take several years before one sees the sequelae of that inflammation. *Id.*

Petitioner returned to Dr. Hamdi on December 17, 1999 (Ex. 17, p. 10) with sudden onset of loss of vision in her left eye, decreased visual acuity, burning sensations, and hyperaesthesias in both lower extremities, which mean she had an acute problem. Tr. at 39. But, on examination, petitioner did not have any relative afferent pupillary defect, meaning that she was in that group of people who did not have a change in her pupils when she had optic neuritis. *Id.*

Dr. Tornatore did not believe that petitioner had retinopathy or central serous retinopathy in 1993 when she had a leaking blood vessel. Tr. at 40. To diagnose petitioner in 1993, Dr. Hrisomalos used the code 362.63 (Ex. 4, p. 5) for lattice degeneration. *Id.* That indicates myopia in the very fringes of the eye where the retina is pulled a bit. *Id.* Dr. Tornatore doubts that petitioner had a retinal or a myopic problem for just one time. Tr. at 41. A few months later, petitioner had all the symptoms to diagnose MS. *Id.* These included the symptoms of optic neuritis. It would not make sense for her to have retinal detachment and then optic neuritis. *Id.* Dr. Hrisamolos should have examined petitioner's pupils and he did not. Tr. at 42. Dr. Hamdi felt that what happened in 1993 was optic neuritis. *Id.*

Comparing the symptoms of petitioner in 1993 in the right eye with her symptoms in 1999 in the left eye, Dr. Tornatore stated that in 1993, she had decreased acuity as if she had a screen in front of her eye, but in 1999, she had sudden loss of vision. Tr. at 43. The basic symptoms for 1993 and 1999 were loss of or change of vision in one eye that occurred relatively suddenly. *Id.* On June 28, 1993, petitioner's vision in her right eye was 20/60. Tr. at 45. The same was for the left eye. *Id.* On August 2, 1993, petitioner's vision in her right eye was 20/80, and in her left 20/50, showing some changes in both eyes. *Id.* In optic neuritis, patients typically have reduced visual acuity ranging from nearly normal to no light perception, citing to Ex. 23. Tr. at 46. One does not have to have complete loss of vision to have optic neuritis. *Id.* In reduced visual acuity, the sharpness of vision is reduced. *Id.*

Dr. Tornatore's opinion is that petitioner's onset of MS occurred within a month of her third hepatitis B vaccination. Tr. at 47. His basis is his interpretation of petitioner's visit to Dr. Hrisomalos in June 1993, her symptoms, and Dr. Hamdi's 1999 concurrence that petitioner had

optic neuritis in 1993. Dr. Hamdi was petitioner's treating neurologist. *Id.* Dr. Tornatore connects petitioner's onset of optic neuritis one month after her third hepatitis B vaccination with her subsequent diagnosis of MS three months later. *Id.* His basis is that optic neuritis is an inflammatory autoimmune disease of the optic nerve which is frequently the heralding symptom of someone who is going to develop MS. Tr. at 47-48. The percentage of patients who have optic neuritis and subsequently develop MS is very high. Tr. at 48.

On cross-examination, Dr. Tornatore admitted that headache, sparklers (photopsias), a veil-like film over the eyes, and color change can be symptoms of a retinal disorder. Tr. at 50. Petitioner had some lattice degeneration at the periphery of her retina. Tr. at 51. But changes at the periphery would not entail changes throughout the eye such as the screen. *Id.* Dr. Tornatore questioned why if petitioner had retinal detachment, she did not continue to have this as a problem. Tr. at 52. Patients with high myopia such as petitioner are at high risk for retinal detachment. Tr. at 53-54.

Dr. Tornatore stated that Dr. Hamdi gave us the piece of information six years later when he wrote that he saw petitioner's right disk was atrophied and pale, and that she had optic neuritis in 1993. Tr. at 54. Dr. Hamdi said that because no eye problem intervened between 1993 and 1999. *Id.*

Dr. Aaron Miller testified for respondent. Tr. at 56. He is a neurologist specializing in MS. Tr. at 58. His opinion is that it is extremely improbable that petitioner had optic neuritis in June 1993. Tr. at 59. Petitioner lacked all the common symptoms of optic neuritis in June 1993. *Id.* The symptoms she had are more likely related to retinal disease. *Id.* The hallmark of her complaints was photopsias or sparklers. Tr. at 59-60. Her symptoms in 1999 comprised the

hallmarks of optic neuritis when she saw Dr. Hamdi, who made the appropriate diagnosis of optic neuritis, i.e., loss of vision in one eye that occurs suddenly and is usually accompanied by a scotoma which is like a hole in the field of vision. Tr. at 60. Pain frequently accompanies it when the eye moves. In the aftermath, people with optic neuritis have an afferent pupillary defect. *Id.* But petitioner never had an afferent pupillary defect in her right eye. *Id.* Dr. Miller admitted on questioning from the undersigned that petitioner did not have an afferent pupillary defect in her left eye when Dr. Hamdi saw her. *Id.*

Dr. Miller stated he had seen hundreds of patients with optic neuritis and cannot recall anyone complaining of sparklers or photopsias. Tr. at 60-61. Patients with optic neuritis tell him they lost vision in one eye or their vision in one eye is very blurry, often with pain on eye movement. Tr. at 61. On examination, he may or may not see optic pallor. The inflammation is frequently behind the optic nerve preventing the examiner from seeing acute inflammation. *Id.*

Dr. Sermersheim, the neurologist who diagnosed petitioner in September 1993 with MS, specifically looked at her pupils and did not note an afferent pupillary defect. Tr. at 61-62. He also did not note any optic atrophy which was at least three full months after petitioner's initial visual symptoms. Tr. at 62.

Dr. Hrisomalos in 1993 found a number of abnormalities in petitioner's retina: photopsias, flashing lights, sparklers. They are far more typical of retinitis. *Id.* Petitioner did not have any of the hallmarks of optic neuritis in June 1993: sudden or rapid loss of vision, afferent pupillary defect, scotoma. *Id.* Her visual acuity was the same in both eyes. *Id.*

Dr. Miller stated he was not surprised that Dr. Hamdi found pallor indicating atrophy in petitioner's right optic nerve in 1999 and that finding did not indicate she had a bout of optic

neuritis in 1993. Tr. at 63, 64. Acute optic neuritis means an episode in which symptoms of acute inflammation occur: loss of vision, very blurry unilateral vision, a hole in the field of vision, pain on movement, resulting in the loss of myelin. Tr. at 63. This results ultimately in the pallor of the disk. *Id.* A lot of MS patients will have damage to their optic nerve insidiously over time without an acute episode. It is not uncommon to find them with an afferent pupillary defect in one eye when they never noticed symptoms. *Id.* Dr. Miller believes that petitioner's optic atrophy which Dr. Hamdi noticed in 1999 in petitioner's right optic nerve occurred after the time Dr. Sermersheim examined petitioner's optic nerves in September 1993 and did not notice optic atrophy and before 1999 when Dr. Hamdi examined petitioner's eyes. Tr. at 64-65.

Dr. Miller stated that, in June 1993, petitioner did not have loss of visual acuity, afferent pupillary defect, pallor of the optic nerve, or a scotoma. Tr. at 65-66. He admitted on cross-examination that a patient with loss of visual acuity will most typically complain of blurred vision in one eye. Tr. at 67. Petitioner did complain her vision was blurred, as if she were looking through a screen in front of her eye. *Id.* Dr. Miller admitted that complaining of looking through a screen could be a symptom of optic neuritis. *Id.* But petitioner in June 1993 had no difference in the visual acuity between her two eyes. Tr. at 68. In the second visit, in August 1993, she had lower visual acuity in the right eye than in the left. *Id.* A very small percent of patients with optic neuritis have no change in visual acuity. *Id.* There was a difference of 20 points between the first and second eye examinations (June and August 1993). Tr. at 69. Dr. Miller's experience is that patients with optic neuritis do not complain about flashing lights. Tr. at 70. He disagreed with Dr. Tornatore that a retinal problem would not have gone away. Dr. Miller testified that retinal problems can be intermittent. Tr. at 71.

Dr. Miller agreed that color changes can be a symptom of optic neuritis. *Id.* A washing out of color is a very common manifestation of optic neuritis. Tr. at 72. Someone looking at the retina would also be looking at the optic nerve. *Id.* He thinks it ridiculous that Dr. Hrisomalos would not have commented on petitioner's optic nerve if he had seen anything abnormal about it in 1993. Tr. at 73. He agreed that if the inflammation was further down the optic nerve, someone would not see it on examination by just looking at the retina. Tr. at 73-74. It usually takes a few months to develop atrophy after optic neuritis. Tr. at 74. It might take longer than from June to September 1993 to develop atrophy, but Dr. Miller said an afferent pupillary defect should be present from the beginning. *Id.* However, Dr. Miller agreed that petitioner never had afferent pupillary defect in either eye. *Id.*

Dr. Miller disagrees with Dr. Hamdi's diagnosis of bilateral optic neuritis, because the "itis" implies an active inflammatory process. Tr. at 74-75. Dr. Miller prefers to say that petitioner had bilateral optic neuropathy, i.e., a disease of both optic nerves. Tr. at 75. Dr. Miller thinks it was inappropriate for Dr. Hamdi to assume that what he observed in petitioner's eye in 1999 was the result of optic neuritis in 1993 when the person examining petitioner in 1993 did not diagnose that. Tr. at 76. Petitioner denied having had any visual symptoms at all in September 1993 when she saw Dr. Sermersheim. Tr. at 77.

Upon questioning from the undersigned, Dr. Tornatore stated that three months between the time petitioner saw Dr. Hrisomalos in June 1993 and Dr. Sermersheim in September 1993 was a relatively short time to start to develop optic atrophy in her right eye since her degree of inflammation of that optic nerve was not great. Tr. at 77-78. If there were an insidious inflammation, it would take longer. Tr. at 78.

In answer to the undersigned's question, Dr. Miller stated that Dr. Hamdi in 1999 would not have known when petitioner's optic neuritis in her right eye began and therefore there was no reason for him to assume she had optic neuritis in 1993. Tr. at 80. He thinks there is no clear evidence that petitioner ever had acute optic neuritis in her right eye. She did have it in her left eye in 1999. Tr. at 82. She had optic neuropathy, not neuritis, in her right eye because Dr. Hamdi observed pallor or atrophy of her optic disk, implying that at some point over the years she had damaged the right optic nerve. Tr. at 82-83.

Dr. Tornatore thought that petitioner had optic neuritis, not neuropathy, on June 28, 1993. Tr. at 85. It came on in the course of a week meaning something abrupt happened. This indicates an acute inflammatory process rather than slow smoldering, but it was not bad because her visual acuity was 20/60 in June 1993. *Id.* One-quarter of optic neuritis patients do not have afferent pupillary defect. *Id.*

Dr. Miller agreed that petitioner's visual acuity improved to 20/20 bilaterally without correction. Tr. at 86. He does not doubt that petitioner had optic neuritis in her left eye in 1999 when she had MS. Tr. at 88. She never had an afferent pupillary defect in either eye. Tr. at 88-89.

DISCUSSION

This is a causation in fact case. To satisfy her burden of proving causation in fact, petitioner must offer "(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. Secretary of HHS, 418 F. 3d 1274, 1278 (Fed. Cir. 2005). In Althen, the Federal Circuit quoted its opinion in Grant v. Secretary of HHS, 956 F.2d 1144, 1148 (Fed. Cir. 1992):

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

In Capizzano v. Secretary of HHS, 440 F.3d 1274, 1325 (Fed. Cir. 2006), the Federal Circuit said "we conclude that requiring either epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect is contrary to what we said in Althen...."

Close calls are to be resolved in favor of petitioners. Capizzano, 1440 F.3d at 1327; Althen, 418 F.3d at 1280. *See generally*, Knudsen v. Secretary of HHS, 35 F.3d 543, 551 (Fed. Cir. 1994).

Without more, "evidence showing an absence of other causes does not meet petitioners' 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 the vaccine, she would not have had optic neuritis and MS, but also that the vaccine was a substantial factor in bringing about her optic neuritis and MS. Shyface v. Secretary of HHS, 165 F.3d 1344, 1352 (Fed. Cir. 1999).

The Federal Circuit in Capizzano emphasized that the special masters are to consider seriously the opinions of petitioner's treating doctors. In that case, four of petitioner's treating doctors opined that hepatitis B vaccine caused her rheumatoid arthritis. 440 F.3d at 1326.

In Werderitsh v. Secretary of HHS, No. 99-310V, 2006 WL 1672884 (Fed. Cl. Spec. Mstr. May 26, 2006), the undersigned ruled that hepatitis B vaccine can cause MS and did so in that case. Petitioner's transient monocular visual symptoms in her right eye were suggestive of optic neuritis and occurred several days to a week after her first hepatitis B vaccination. 2006 WL 1672884, at *19. She had symptoms of transverse myelitis a month after her second vaccination, and was diagnosed with MS. Respondent's expert, Dr. Roland Martin, testified that the appropriate onset interval, if a vaccination were to cause an acute reaction, would be a few days to three to four weeks. Stevens v. Secretary of HHS, No. 99-594V, 2006 WL 659525, at *15 (Fed. Cl. Spec. Mstr. Feb. 24, 2006).

Both experts in the instant action are neurologists specializing in MS. Both agree that petitioner had optic neuritis. Both experts also agree that optic neuritis is frequently the hallmark of MS. Their difference is in attributing the optic pallor or atrophy in petitioner's right eye to either optic neuritis (according to Dr. Tornatore with onset in June 1993) or to optic neuropathy (according to Dr. Miller with onset some time between 1993 and 1999 when Dr. Hamdi, petitioner's treating neurologist, noted the pallor in petitioner's right optic nerve when she had come to him with symptoms of optic neuritis in her left eye).

One of Dr. Miller's reasons for doubting that petitioner had optic neuritis in her right eye when Dr. Hrisomalos saw her in June 1993 was the absence of an afferent pupillary defect in her right eye. But he agrees that petitioner had optic neuritis in her left eye in 1999 when petitioner did not have an afferent pupillary defect in her left eye. Therefore, Dr. Miller accepts that in an MS patient, the patient may have optic neuritis without an afferent pupillary defect, as the literature shows is the case with a minority of optic neuritis patients. It lessens Dr. Miller's

credibility for him to deny petitioner had optic neuritis in her right eye in 1993 because of the absence of an afferent pupillary defect when that sign was equally missing from her left eye in 1999 when he agrees she had optic neuritis. This point of the defense was not well-considered.

Whereas Dr. Tornatore interprets petitioner's June 1993 complaints of sparkles, a film over her eye, blurry vision, and headache as indicative of optic neuritis, Dr. Miller interprets these symptoms as symptoms of retinal problems. But petitioner never had a retinal problem again. Both doctors agreed that these symptoms could be consistent with optic neuritis as the literature shows. Petitioner also experienced less visual acuity and her eyes had disparate acuity in her next visit to Dr. Hrisamolos in August 1993.

The tie-breaker in this case must be the opinion of petitioner's treating neurologist Dr. Hamdi who, in 1999, when petitioner came to him complaining of undisputed optic neuritis in her left eye, diagnosed her with having had optic neuritis in her right eye in 1993 after he examined her eyes and found pallor in her right optic nerve. The symptoms about which she complained in June 1993 to Dr. Hrisomalos, although consistent with a retinal problem, are also consistent with optic neuritis. As Dr. Tornatore stated, and Dr. Miller agreed, if the inflammation were further back in her eye, Dr. Hrisomalos, and later Dr. Sermersheim, a neurologist who diagnosed petitioner with MS in September 1993, would not have seen the inflammation. Optic neuritis is inflammation of the optic nerve.

The Federal Circuit in Capizzano emphasized taking into serious consideration the opinions of petitioner's treating doctors. There is no reason to doubt Dr. Hamdi's diagnosis after evaluating petitioner's eyes that her right optic nerve pallor or atrophy was due to a prior optic neuritis in 1993, the only time that she voiced visual complaints before 1999.

It seems an extraordinary stretch to adopt Dr. Miller's opinion that some time after petitioner saw Dr. Sermersheim in September 1993 but before she saw Dr. Hamdi in 1999, she had a smoldering inflammatory process which did not reach the acuteness of optic neuritis but did cause her right optic nerve to atrophy and yet ignore totally her complaints of sparkles, blurry or filmy vision, and screened vision in June 1993 which stayed the same or worsened when she saw Dr. Hrisomalos again in August 1993. Dr. Hamdi reached his opinion before there was any litigation. He was her treating neurologist. Dr. Tornatore's opinion is consistent with Dr. Hamdi's diagnosis and the undersigned accepts it as accurate. Because petitioner's optic neuritis was not severe, both eyes recovered 20/20 visual acuity. But this does not negate the occurrence of petitioner's earlier right eye optic neuritis, even though it was relatively mild.

Since petitioner's onset of right optic neuritis was within a month of her third hepatitis B vaccination, the onset is medically appropriate for causation. In Werderitsh, petitioner also had optic neuritis before her diagnosis of MS. The undersigned discussed in Werderitsh the biologically plausible medical theory connecting hepatitis B vaccine and MS. 2006 WL 1672884, at *24-*26. The undersigned notes that Althen concerned the onset of optic neuritis two weeks after tetanus toxoid vaccination followed by the demyelinating disease acute disseminated encephalomyelitis. 418 F.3d at 1281.

Each expert in the instant case agrees that optic neuritis is frequently a hallmark for MS. There is a logical sequence of cause and effect connecting petitioner's exposure to hepatitis B vaccine and her onset of demyelinating disease. Petitioner has proved causation in fact.

CONCLUSION

Petitioner is entitled to reasonable compensation. The undersigned hopes that the parties may reach an amicable settlement, and will convene a telephonic status conference soon to discuss how to proceed in resolving the issue of damages.

IT IS SO ORDERED.

October 31, 2008
DATE

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