In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

(E-Filed: August 31, 2009)
No. 01-707V

MICHAEL STEPHEN SHAW, Petitioner, v. SECRETARY OF THE DEPARTMENT OF HEALTH AND HUMAN SERVICES, Respondent.	**TO BE PUBLISHED** Hepatitis B Vaccine; Small Nerve Fiber Neuropathy; Sequence of Cause and Effect not Logical; Theory of Causation Based on Injury that Record does not Show

Ronald Homer, Boston, MA, for petitioner.

Voris R. Johnson, Department of Justice, Civil Division, Torts Branch, Washington, DC, for respondent.

RULING ON ENTITLEMENT[1]

Petitioner, Michael Shaw, has alleged that the hepatitis B vaccines he received on

[1] As provided by Vaccine Rule 18(b), each party has fourteen days within which to request the redaction "of any information furnished by that party (1) that is trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Rules of the United States Court of Federal Claims (RCFC), Appendix B, Vaccine Rule 18(b). In the absence of timely objection, the entire document will be made publicly available.

May 5, 1999, and June 11, 1999, caused him to suffer a neuropathy.[2] See Petition (Pet.) at 2-8.[3] On December 20, 2001, he filed a petition pursuant to the National Vaccine Injury Compensation Program (Vaccine Program or Program).[4] 42 U.S.C. §§ 300aa-1 to -34 (2006).

Mr. Shaw relies on a theory of causation in fact. In support of his claim, Mr. Shaw has filed: (1) his affidavit; (2) his medical records; (3) the medical opinion of Sherri Tenpenny, D.O., an osteopathic physician;[5] (4) supporting medical literature, and (5) post-hearing briefs. Respondent challenges Mr. Shaw's claim and Dr. Tenpenny's expertise in neurology. Respondent offered the expert opinion of Thomas Leist, M.D., a neurologist, and a post-hearing memorandum.

During a recorded proceeding on March 12, 2008, in Sacramento, California, the undersigned heard the testimony of Mr. Shaw, Dr. Tenpenny and Dr. Leist. Based upon the developed factual record, the supporting medical literature, and the testimony of the parties' medical witnesses and for the reasons set forth in this ruling, the undersigned finds that petitioner has failed to satisfy his burden of proving vaccine-related causation.

I. Facts

In general, the parties do not dispute the underlying facts in this case, and as directed by the Vaccine Act, the undersigned has carefully considered, "in addition to all other relevant medical evidence contained in the record" the diagnoses, conclusions, and medical judgments contained in the record regarding the nature, causation and aggravation of petitioner's condition as well as the results of diagnostic tests contained in

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[2] A neuropathy is defined as "a functional disturbance or pathological change in the peripheral nervous system." Dorland's Illustrated Medical Dictionary 1257 (30th ed. 2003).

[3] Unrepresented by counsel at the time he filed his petition, petitioner filed with the petition an unnumbered collection of medical records and a summary of his medical records. The unnumbered pages are cited in sequential order.

[4] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C.A. § 300aa-10 et seq. (2006) (Vaccine Act or the Act). All citations in this Decision to individual sections of the Vaccine Act are to 42 U.S.C.A. § 300aa.

[5] Dr. Tenpenny explained that as an osteopathic physician, she received medical school training as well as training in manipulation such as a chiropractor receives. Transcript of March 12, 2008 hearing (Tr.) at 31.

the record. See 42 U.S.C. §300aa-13(b)(1). The undersigned does not review here, however, all of the voluminous medical records filed by petitioner. Rather, the undersigned now reviews the records upon which the parties have relied most heavily and upon which the undersigned relies most particularly in this case.

Petitioner was born on June 15, 1959. Petitioner's Exhibit (Pet.'s Ex.) 1 at 1. His medical history is most notable for a couple of concussive head injuries, a cracked pelvis, a chipped tailbone, a fractured nose, and broken hands and feet. Pet.'s Ex. 4 at 5; Tr. at 12, 16. He also has a history of herpes. Pet.'s Ex. 2 at 4.

Prior to receiving the vaccinations at issue in this case, petitioner traveled extensively in his professional capacity as the corporate general manager for a large, multi-national trading firm. Pet.'s Ex. 43 at 1-2. He had responsibilities for approximately 30 offices throughout the Asian Pacific. Id.

Recreationally, Mr. Shaw enjoyed extreme sports activities, including motorcross riding, mountain biking, roller blading, hang gliding, parachuting, rafting and mountain climbing. Id. at 1. He also enjoyed golf, tennis, skiing, softball, and basketball. Id.

In anticipation of scheduled business travel and as part of an employment-related immunization program, Mr. Shaw received his first hepatitis B vaccination on May 5, 1999. Pet.'s Ex. 2 at 67; Transcript of March 12, 2008 Hearing (Tr.) at 5. He did not recall experiencing any effects after that vaccination. Tr. at 5.

The next month, on June 11, 1999, he received his second hepatitis B vaccination and a polio vaccination. Pet.'s Ex. 2 at 67; Pet.'s Ex. 43 at 2. He recalled experiencing tingling and numbness in his "great toe" within 48 hours of his receipt of the second hepatitis B vaccination. Pet.'s Ex. 43 at 2; Tr. at 7. Contrary to petitioner's testimony, however, the most contemporaneous medical records indicate that the onset of numbness occurred six rather than two days after petitioner received a second administration of the hepatitis B vaccine. Compare Pet.'s Ex. 43 at 2 and Tr. at 7 (petitioner's affidavit and hearing testimony) with Pet.'s Ex. 1 at 31 and Pet.'s Ex. 5 at 3 (contemporaneous medical records reflecting a different time period for onset). Consistent with the guidance set forth in Curcuras v. Secretary of Health and Human Services, 993 F.2d 1525, 1528 (1993),[6] the undersigned would afford greater weight to the contemporaneous medical

[6] Noting that in United States v. United States Gypsum Co., 333 U.S. 364, 396 (1947), "the Supreme Court counsel[ed] that oral testimony in conflict with contemporaneous documentary evidence deserves little weight," the Federal Circuit in Curcuras stated:

records than to petitioner's later-offered affidavit and hearing testimony in this case. But, the undersigned need not resolve this particular fact discrepancy because petitioner has asserted that either time frame--whether found to be two or six days--would be medically appropriate based on petitioner's theory of causation.

The medical records indicate that on June 21, 1999, 10 days after receiving the hepatitis B vaccination of interest, petitioner visited his primary care physician, John Roberts, M.D., of Blackhawk Medical Group, with complaints of recurrent numbness in his right leg below the knee. Pet.'s Ex. 1 at 31; Petitioner's Post-Hearing Brief (P's Brief) at 2. Petitioner reported that the numbness had begun on June 17, 1999, four days prior to his visit to Dr. Roberts and six days after he received his second hepatitis B vaccine.[7] Pet.'s Ex. 1 at 31. Dr. Roberts noted that petitioner had a history of "lots of trauma" due to his motorcross riding. Id. Dr. Roberts diagnosed petitioner with a lumbar strain with right-leg radiculopathy.[8] Id. Dr. Roberts prescribed prednisone[9] and urged petitioner to obtain x-rays and an MRI.[10] Id.

Petitioner began an international business trip on June 23, 1999. Pet.'s Ex. 43 at 2. In his affidavit prepared on October 17, 2006, he recalled that:

> Medical records, in general, warrant consideration as trustworthy evidence. The records contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium. These records are also generally contemporaneous to the medical events.

993 F.2d at 1528.

[7] The pertinent medical record indicates that the numbness began on the Thursday preceding the office visit. Pet's Ex. 1 at 31. According to the June 1999 calendar, the referenced Thursday was June 17, 1999. See id.; see also http://www.timeanddate.com/calendar/index.html?year=1999&country=1

[8] Radiculopathy is a "disease of the nerve roots." Dorland's at 1562.

[9] Prednisone belongs to a class of drugs called steroids. See http://www.drugs.com/prednisone.html. A synthetic glucocorticoid, it is used as an antiinflammatory and as an immunosuppressant. See Dorland's at 1500.

[10] Magnetic Resonance Imaging (or an MRI) is "a method of visualizing soft tissues of the body by applying an external magnetic field that makes it possible to distinguish between hydrogen atoms in different environments." Dorland's at 908.

> By the time I reached my first stop in England, both my feet [and] legs were affected. During business meetings in India, I began to experience tremors in my limbs, cognitive memory/speech problems, and coordination difficulties. Prior to returning home from the two-week trip, my arms were also affected. The symptoms now included, not only, numbness and tingling but also sharp, shooting, burning, and throbbing pain. I managed to complete the trip in defiance of significant pain.
>
> Once home, the pain continued. I experienced numbness in both of my hands and legs and had spasms in my back.

Id.

Upon his return, petitioner reported that the prednisone trial had been unsuccessful. See Pet.'s Ex. 1 at 29 ("Had prednisone x 6 days before trip"), 31 (reference to "prednisone taper"). Petitioner underwent imaging of his spine on July 6, 1999. Pet.'s Ex. 1 at 95. The MRI of his cervical spine produced an impression of "[e]arly disc degeneration without extrusion." Id. The MRI of his lumbar spine was normal. Id. at 95-96.

On July 9, 1999, three days after his spinal MRI, petitioner returned to his primary care provider. See Pet.'s Ex. 1 at 29. He complained of flu-like symptoms and of continued numbness in his right leg. Id. Although the office notes reflect a history of numbness in petitioner's left leg and hands, no time frame is specified. Id.; but see Pet.'s Ex. 1 at 25 (petitioner reporting, during a visit to his primary care physician on October 27, 1999, that his numbness had progressed to all of his extremities in late June). The diagnostic impression was sinusitis and strain in the lumbar and cervical regions of the spine. See Pet.'s Ex. 1 at 29. The examining physician prescribed Lorabid and Xanax[11] and ordered physical therapy. Id.

Five weeks later, on August 18, 1999, petitioner visited Samuel Jorgenson, M.D.,

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[11] Lorabid "is used to treat mild-to-moderate bacterial infections of the lungs, ears, throat, [and] sinuses." http://www.pdrhealth.com/drugs/rx/rx-mono.aspx?contentFileName=Lor1237.html&contentName=Lorabid&contentId=317.

Xanax is indicated for the treatment of panic disorder which can include symptoms of paresthesias (specifically, numbness or tingling sensations). See http://www.rxlist.com/xanax-drug.htm.

an orthopedist. Pet.'s Ex. 4 at 11. Petitioner reported a two-month history of right foot pain and intermittent numbness and tingling in his arms, hands, and feet. Id. Petitioner also reported that he did not take the prescribed Xanax because it caused drowsiness. Id. at 10. Dr. Jorgenson's physical examination revealed a "decreased sensation to sharp pin prick" in petitioner's right foot when compared with his left one. Id. at 12. It was the orthopedist's assessment that petitioner had a possible entrapment neuropathy in his lower right extremity. Id.

Dr. Jorgenson referred petitioner for an electromyogram[12] that was conducted on September 2, 1999. See Pet.'s Ex. 1 at 66. The electromyogram (or EMG) revealed no evidence of "acute or chronic lumbosacral radiculopathy, plexopathy, or peripheral neuropathy." Id. at 67. In other words, there was no evidence in petitioner's lower back of any disease of the nerve roots (radiculopathy, see Dorland's at 1562), or any disorder of the network of nerves (plexopathy, see Dorland's at 1453), or any functional disturbance of the nervous system affecting petitioner's extremities (peripheral neuropathy, see Dorland's at 1257). Petitioner had described symptoms of progressive burning pain and intermittent numbness from his foot to his ankle that, at times, emanated to his knee. Pet.'s Ex. 1 at 66. The physician interpreting the EMG results noted that "the patient is most likely exhibiting very early symptoms of idiopathic peripheral neuropathy" and recommended a trial of Neurontin[13] to reduce the burning parasthesias. Id. at 67.

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[12] An electromyogram (or EMG) "is a test that is used to record the electrical activity of muscles. When muscles are active, they produce an electrical current. This current is usually proportional to the level of the muscle activity. . . . EMGs can be used to detect abnormal electrical activity of muscle that can occur in many diseases and conditions, including . . . inflammation of muscles, pinched nerves, [and] peripheral nerve damage (damage to nerves in the arms and legs)." http://www.medicinenet.com/electromyogram/article.htm. A physician may order the performance of an EMG when a patient has unexplained muscle weakness. Id. "The EMG helps to distinguish between muscle conditions in which the problem begins in the muscle and muscle weakness due to nerve disorders. The EMG can also be used to detect true weakness, as opposed to weakness from reduced use because of pain or lack of motivation. EMGs can also be used to isolate the level of nerve irritation or injury." Id.; see also Mosby's Manual of Diagnostic and Laboratory Tests at 571-573 (3d ed. 2006) (stating same).

[13] Neurontin is an anticonvulsant. See http://www.drugs.com/neurontin.html. Because it affects chemicals and nerves in the body that are involved in the cause of seizures and some types of pain, it may be used in the treatment of epilepsy. Id. It also may be used to treat nerve pain caused by the herpes virus or shingles. Id.

Approximately two months later, on November 9, 1999, petitioner saw Janet Lin, a neurologist, on referral from Dr. Roberts, his primary care physician. Pet.'s Ex. 2 at 1, 3. Dr. Lin noted that petitioner's neurologic exam was normal "except for some minimal sensory abnormalities in his hands and feet." Pet.'s Ex. 2 at 3. Although petitioner reported feeling fatigued, there was no evidence of muscle weakness. Id. Dr. Lin believed that petitioner was "suffering [due to] a post-inflammatory neuropathy related to immunizations." Id. She surmised that the "culprit" might be the hepatitis B immunization that petitioner received because petitioner had received all the other immunizations previously. Id.

Petitioner sought treatment from a variety of specialists over the next five years. On referral from his primary care doctor, petitioner consulted on February 28, 2000, with Benedict Villanueva, M.D., an infectious disease specialist. See Pet.'s Ex. 6 at 1, 4. As reflected in the notes from the consultation, Dr. Roberts had referred petitioner to Dr. Villanueva for an evaluation of whether his symptoms of diffuse sensory neuropathy were a "[p]ossible post vaccine adverse reaction." Id. at 1. The particular vaccine under examination was the polio vaccine--not the hepatitis B vaccine--that petitioner received in June 1999. Id. Dr. Villanueva noted that petitioner had a normal EMG, a "basically" normal MRI of his cervical and lumbar area, and, with the exception of a slightly elevated protein level, a normal spinal tap.[14] Id. In Dr. Villanueva's assessment, among the "[p]ossible etiologies" for petitioner's "subjective diffuse sensory polyneuropathy" would be a "rare/remote adverse reaction to the polio vaccine." Id. at 3 (emphasis added). But, Dr. Villanueva observed, such reactions occur within a few weeks after immunization and, to his knowledge, do not last for several months after the inoculation. Id.

Two months later, petitioner underwent further neurological examination by Catherine Lomen-Hoerth, M.D., at the University of California in San Francisco. See Pet.'s Ex. 9 at 107. He returned to Dr. Lomen-Hoerth on May 10, 2000, for a follow-up of continuing pain and numbness. Id. Dr. Lomen-Hoerth noted that petitioner's discomfort had progressed and was worse than when she had examined him for the first time one month earlier. See id. It was Dr. Lomen-Hoerth's impression that petitioner had "a progressive small fiber neuropathy rather than a static neuropathy related to his vaccinations last summer." Id. Other diagnoses that Dr. Lomen-Hoerth considered were

[14] A spinal tap or cerebrospinal fluid (CSF) examination that yields an elevated protein level may be indicative of an underlying infectious or inflammatory process. See Mosby's at 677, 681.

chronic Epstein Barr Virus[15] and chronic fatigue syndrome. Id. at 34.

On referral from Dr. Lomen-Hoerth, petitioner saw David Martin, M.D., a rheumatologist, on July 31, 2000. Pet.'s Ex. 2 at 9. The purpose of the referral was to evaluate petitioner's severe fatigue, weight loss, intermittent burning rash on both arms and joint pain. Id. at 10. It was Dr. Martin's impression that extensive laboratory work and physical examination failed to produce any clear evidence of connective tissue disease. Id. at 11-12. In his view, petitioner suffered from "an idiopathic syndrome associated with chronic fatigue and . . . is possibly related to a vaccine exposure or possibly a toxin." Id. at 12 (emphasis added). Dr. Martin suspected that petitioner's condition had an underlying psychiatric component with possible depression. Id. Dr. Martin recommended increased aerobic exercise, continued use of antidepressants and decreased consumption of marijuana. Id.

Nearly five months later, on January 3, 2001, petitioner presented to the emergency room "[a]cting strange and confused" and complaining of worsening pain in his extremities. Pet.'s Ex. 3 at 25. The admission notes indicate that petitioner has a neuropathic condition that has waxed and waned but is slowly progressive. Id. Petitioner also has experienced some changes in mental status including poor memory and decreased alertness and concentration. Id. The diagnosis on discharge was "[a]cute severe exacerbation of chronic neuropathy pain." Id. at 27.

Petitioner filed a workers' compensation claim with his employer on February 5, 2001. Pet.'s Ex. 8 at 13. On April 25, 2001, petitioner returned to his primary care physician for the purpose of completing disability forms. Pet.'s Ex. 1 at 8. Ultimately found to have a disability for which future medical expenses were likely, petitioner entered into a Workers' Compensation Medicare Set-aside Arrangement[16] with his employer and began receiving payments. See Tr. at 11-12; see also http://www.cms.hhs.gov/WorkersCompAgencyServices/04_wcsetaside.asp.

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[15] Epstein Barr Virus (or EBV) is the virus that causes infectious mononucleosis. Dorland's at 2044.

[16] A Workers' Compensation Medicare Set-aside Arrangement is a method of resolving a workers' compensation case that includes future medical expenses. http://www.cms.hhs.gov/WorkersCompAgencyServices/04_wcsetaside.asp. The arrangement involves an allocation of a portion of the workers' compensation settlement for future medical expenses. Id. The amount of the set aside is determined on a case-by-case basis to pay for future Medicare covered expenses related to the compensable injury. Id.

On May 8, 2001, petitioner saw Rex Chiu, M.D., an internist at Stanford Hospital and Clinics, on referral from Dr. Lomen-Hoerth. Pet.'s Ex. 5 at 13. Dr. Chiu noted that petitioner experienced an onset of numbness and tingling in his left toe six days after receiving a polio vaccination and a hepatitis B vaccination in anticipation of business travel to India. Id. Petitioner's developing symptoms produced "concern for a postinflammatory reaction to the immunizations," but a trial course of prednisone provided no relief. Id. Following a series of visits to diverse medical specialists, the "consensus diagnosis" is small fiber neuropathy. Id. Dr. Chiu wrote that because petitioner's neurologic changes seem to have arisen after his immunization in 1999, "there is a question as to whether there is some type of autoimmune or other reaction to this vaccination, which may now be worsening in a progressive fashion." Id. at 16 (emphasis added). Dr. Chiu noted, "[T]he patient is hepatitis B negative." Id. Dr. Chiu planned to refer petitioner for further neurological and rheumatological examination at Stanford. Id.

On referral from Dr. Chiu, Yuen So, M.D., a neurologist at Stanford, examined petitioner on July 21, 2001. Pet.'s Ex. 5 at 21. Dr. So noted that petitioner had seen a number of neurologists over a two year period. Id. Dr. So further noted that the "most disabling" feature of petitioner's illness has been his diffuse pain. Id. Based on a physical examination of petitioner and a review of petitioner's laboratory test results, Dr. So wrote: "It is conceivable that [petitioner] had an acute, predominantly sensory polyneuropathy back in 1999." Id. at 21-22. But without the records of petitioner's medical evaluation during that time period, Dr. So found it "difficult" to ascribe petitioner's complaint of progressive symptoms since 1999 to the received vaccinations. Id. at 22. Disturbing to Dr. So about petitioner's condition was the "very diverse nature" of petitioner's symptoms. Id. Also disturbing to Dr. So was the lack of objective evidence of neuropathic abnormality in a patient who has had ongoing disease for a course of two years. Id. Contrary to normal expectations for a patient suspected of having a prior acute neuropathy, petitioner did not demonstrate a slow and steady course improvement. See id. Dr. So described the case as a "very difficult" one to diagnose and to treat. Id.

In September 2001, petitioner and his wife moved from northern California to Delaware. See Pet.'s Ex. 18 at 46. Approximately, two months later, on November 8, 2001, petitioner visited Gail Berkenblit, M.D., an internist at Johns Hopkins, for ongoing chronic pain. Pet.'s Ex. 13 at 161; see also http://www.hopkinsmedicine.org/gim/faculty/berkenblit.htm. Dr. Berkenblit conducted a physical examination and reviewed the records that petitioner presented regarding his extensive laboratory work. See id. at 161-165. Dr. Berkenblit took an extensive patient history and noted that petitioner's evaluations have been essentially normal, including his autonomic function testing. See id. Petitioner's initial diagnosis was a possible

postinflammatory neuropathy. Id. at 162. Subsequently, petitioner has received evaluations for a possible small fiber neuropathy. Id. Repeated testing, however, has not disclosed any "definite evidence of a small fiber neuropathy." Id. Rather, swelling noticed in the distal leg sites during a neurological examination at Johns Hopkins by Dr. Nicholas Maragakis was suggestive of "early possible nerve fiber degeneration." Id. During the office visit, Dr. Berkenblit addressed concerns expressed by petitioner and his wife that petitioner's symptoms resulted from his hepatitis B vaccination. Id. at 165. Dr. Berkenblit observed that there is no clear link between hepatitis B vaccination and progressive neuropathic pain, but noted that "[i]f [petitioner] did develop symptoms of a sensory neuropathy as a consequence of the vaccine it would most likely be an autoimmune type mechanism" and not a vaccine contamination issue as petitioner's wife speculated. Id.

Petitioner filed his vaccine claim on December 20, 2001. See Pet. at 1.

A little more than one month later, on February 7, 2002, petitioner visited Lee Dresser, M.D., a neurologist, for an evaluation. Pet.'s Ex. 18 at 44. Dr. Dresser noted that previous evaluations by neurologists included an assumption that petitioner developed a sensory neuropathy as a response to his vaccination, but that diagnosis has been modified following extensive negative testing. Id. at 45. It was Dr. Dresser's impression that petitioner suffers from "[d]iffuse dysesthetic pain following remote vaccinations." Id. at 47. Of interest to Dr. Dresser was the finding of mild elevation of petitioner's spinal fluid protein following petitioner's extensive and otherwise unremarkable testing. Id. Dr. Dresser observed that petitioner's symptoms were "essentially 100% subjective with no significant objective findings on his testing or examination." Id. Dr. Dresser found petitioner's case to be a "very complicated" one. Id.

To assist petitioner with his pending vaccine claim, Dr. Roberts, the primary care physician who examined petitioner when his symptoms first began in 1999, wrote a letter to petitioner dated February 13, 2002. Pet.'s Ex. 18 at 6. Dr. Roberts stated that petitioner had no significant neurological symptoms prior to the petitioner's receipt of the hepatitis B vaccination and that petitioner began to develop neuralgic complaints shortly after his immunization. Id. It was Dr. Roberts' belief that the temporal relationship between the received vaccination and the onset of petitioner's symptoms "strongly correlate[d]" with the hypothesis that the symptoms were caused by the vaccination. Id.

Thereafter, other treating doctors offered views about what may have caused petitioner's symptoms.

On January 21, 2003, Robert Allen, M.D., an evaluator retained by the defense in connection with petitioner's workers' compensation claim, saw petitioner. Pet.'s Ex. 8 at 12-24; Pet.'s Ex. 33 at 43. Dr. Allen observed that petitioner's neurological evaluations (including biopsies) have not documented any progressive neurological disease. Pet.'s Ex. 8 at 22. In Dr. Allen's opinion, petitioner's clinical history and physical examination, together with the extensive objective work-up, suggested "a diagnosis of fibromyalgia." Id. He explained that "[t]he diagnosis of fibromyalgia involves the presence of widespread musculoskeletal pain, as well as multiple tender points . . . that occur[] both above and below the waist." Id. He stated that "[t]he etiology of his fibromyalgia remains unclear and may have developed as a result of the June 1999 vaccination[s]." Id. (emphasis added). But, Dr. Allen acknowledged, such causation "is impossible to confirm or deny." Id. Dr. Allen was one of two evaluators to diagnose petitioner with fibromyalgia, a diagnosis that is disputed by petitioner's treating physicians.[17] See Pet.'s Ex. 33 at 48.

On April 29, 2003, Harold Buttram, M.D., an internist with Woodland Healing Research Center, examined petitioner. Pet.'s Ex. 25 at 1. Dr. Buttram noted that petitioner had become ill following chelation efforts to eliminate mercury, and subsequent testing indicated that mercury toxicity was not an issue for petitioner. Id. Dr. Buttram further noted that Dr. Tenpenny, the treating physician who testified at hearing on petitioner's behalf, had directed petitioner's mercury detoxicification process. Id. Aware that petitioner's vaccine claim was pending, Dr. Buttram wrote, "For the records, it is my opinion that the patient's peripheral neuropathy is directly related to (was caused by) a series of two hepatitis B vaccines." Id. (emphasis added). Noting that petitioner "ha[d] been diagnosed by neurologists as having chronic neuropathic pain," Dr. Buttram prepared an opinion letter dated June 6, 2003, stating that he agreed with the diagnosis of the neurologists and reiterating that petitioner's condition was caused by a series of hepatitis B vaccines. See Pet.'s Ex. 33 at 95.

On December 23, 2004, David Waldman, M.D., issued an extensive report concerning petitioner's disability status. See Pet.'s Ex. 33 at 2-52. Dr. Waldman's report was informed by his review of petitioner's medical records, his review of medical

[17] The diagnosis of fibromyalgia was first considered by the defense evaluator, Dr. Robert Allen. Another defense evaluator, Dr. Skomer, diagnosed a chronic pain condition but allowed that petitioner's symptoms were "possibly consistent with [a finding of] fibromyalgia." Pet.'s Ex. 33 at 43, 48-49. But, there is no evidence in the multiple neurological evaluations contained in petitioner's medical records to support a finding that Mr. Shaw has fibromyalgia.

literature,[18] and a physical examination of petitioner. See id. Contained in Dr. Waldman's report was a detailed, chronological summary of petitioner's medical evaluations and laboratory results. Id. at 35-45. Also contained in Dr. Waldman's report was a summary of medical articles that he had reviewed, in connection with his evaluation of petitioner, concerning "complications from the hepatitis B vaccination." Id. at 45. Dr. Waldman concluded:

> There is no evidence within the records submitted that, prior to 6/11/99, Mr. Shaw had any neurological injury and was not able to function . . . . After the vaccinations of 6/11/99, Mr. Shaw began a very complex medical history, resulting in a chronic pain disorder syndrome. . . . Mr. Shaw has a problem with pain medicine addiction, which he did not have prior to his industrial injury. As stated within his multiple medical records, as a consequence of his work-related chronic pain disorder, he has developed a drug dependence. . . . There is no evidence in review of the medical records that Mr. Shaw has a fibromyalgia syndrome. . . . [Rather,] Mr. Shaw has developed a chronic neuropathic pain syndrome. Although the exact etiology has not been determined, based on the review of the medical records and medical literature, it is with medical probability that this syndrome was a consequence of the vaccinations received on 6/11/99. This opinion that this syndrome occurred post vaccination has also been supported by multiple clinical evaluators . . . includ[ing] Dr. Janet Lin and Dr. [Catherine] Lomen-Hoerth[, two neurologists] at UCSF Medical Center. This has also been supported by recent evaluations which Mr. Shaw has sought to obtain relief from his pain syndrome . . . with multiple sequelae,

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[18] The literature that Dr. Waldman reviewed included: (1) A. Tourbah et al., Encephalitis after hepatitis B vaccination. Recurrent disseminated encephalitis or MS? Neurology 53(2): 396-401 (Jul 22, 1999); (2) M. Hernan et al, Recombinant hepatitis B vaccine and the risk of multiple sclerosis, BJM 309(6974): 94 (July 9, 1994); (3) Science Magazine, Vol. 281, Immunology: A shadow falls on the hepatitis B vaccination effort (July 31, 1998); (4) C. M Poser, Neurological complications of vaccinations, Mealey's Litigation Report, Thimerosal & Vaccines (April 2003); (5) B. Dunbar, Professor of cell biology investigates hepatitis B vaccine damage, Baylor College of Medicine, Houston, TX; (6) Y. Shoenfield, Center for Autoimmune Diseases, Dept. of Internal Medicine, B. Sheba Medical Center, Tel-Hashomer, Israel. Vaccination and autoimmunity "vaccinosis": a dangerous liaison? Academic Press (2000); (7) B. A. Waisbren, Demyelinizing diseases occurring after hepatitis B vaccination, Wisconsin Medical J. 95(3): 148; (8) D. A. Geier and M. R. Geier, Chronic adverse reactions associated with hepatitis B vaccination, an examination of the VAERS database following adult HBV from 1997-2000, Ann. Pharmacother. 2002; (9) Vaccine Reaction Special Report, Hepatitis B vaccine: the untold story (September 1998).

including drug dependence, and these conditions are industrial in nature.

Id. at 48-49 (emphasis added).

A little more than three years after Dr. Waldman's evaluation of petitoiner, petitioner testified at the hearing for his vaccine claim that he continues to experience fluctuating levels of pain. See Tr. at 13. His pain is best managed by the opiate therapy he has been prescribed. Id. at 15.

II. Applicable Legal Standards

The Vaccine Act provides two separate methods by which to obtain Program compensation: Vaccine Injury Table (Table) claims and causation in fact claims. Andreu v. Sec'y of Health and Human Servs., 569 F.3d 1367 (Fed. Cir. 2009). When asserting a Table claim, a claimant is afforded a presumption of causation if he shows that he received a vaccination listed on the Table, 42 C.F.R 100.3(a), and suffered an injury listed on the table within the prescribed period. 42 U.S.C. § 300aa-11(c)(1)(C)(i); see Pafford v. Sec'y of Health and Human Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). If unable to avail himself of the Table method of establishing causation, however, the claimant must show that his injury was "caused in fact" by the vaccine he received. See Capizzano v. Sec'y of Health and Human Servs., 440 F.3d 1317, 1320 (Fed. Cir. 2006).

The Vaccine Act provides for the compensation of "any illness, disability, injury, or condition not set forth in the Vaccine Injury Table but which was caused by a vaccine" covered under the Program. 42 U.S.C. § 300aa-11(c)(1)(C)(ii)(I). The Act does not require a petitioner bringing a non-Table claim "to categorize [the suffered] injury." Kelley v. Sec'y of Health and Human Servs., 68 Fed. Cl. 84, 100 (Fed. Cl. 2005). Rather, a petitioner is required only "to show that the vaccine in question caused [him] injury--regardless of the ultimate diagnosis." Id. But when, as in this case, the conditions at issue present with many of the same symptoms but the underlying causes and required treatments are different and when, as in this case, the evidence for causation depends on the particular diagnosis of petitioner's condition, a special master acts properly in considering whether the record supports the diagnosis proposed by petitioner. See Broekelschen v. Sec'y of Health and Human Servs., No. 07-137, 2009 WL 2569734 (Fed. Cl. 2009).

A petitioner may prove that a received vaccine in fact caused the injury sustained by satisfying the three-part test set forth by the Federal Circuit in Althen v. Secretary of Health and Human Services, 418 F.3d 1274 (Fed. Cir. 2005):

> Concisely stated, [a claimant's] burden is to show by preponderant evidence that the vaccination brought about [his] 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. If [a claimant] satisfies this burden, [he] is entitled to recover unless the [government] shows, also by a preponderance of evidence, that the injury was in fact caused by factors unrelated to the vaccine.

418 F.3d at 1278 (quotation marks omitted).

Althen also makes clear that a claimant's theory of causation must be supported by a "reputable medical or scientific explanation." 418 F.3d at 1278 (citations and internal quotation marks omitted); see also Knudsen v. Sec'y of Health and Human Servs., 35 F.3d 543, 548 (Fed. Cir. 1994) (requiring a "sound and reliable medical or scientific explanation"). Although a claimant need not produce medical literature or epidemiological evidence to establish causation under the Vaccine Act, where such evidence is submitted, the special master may consider it in reaching an informed judgment as to whether a particular vaccination more likely than not caused a particular injury. See Andreu, 569 F.3d at 1379; Althen, 418 F.3d at 1280; Capizzano, 440 F.3d at 1325. See also Daubert, 509 U.S. at 593-97 (noting that one factor in assessing the reliability of expert testimony is whether the theory espoused enjoys general acceptance within a relevant scientific community).

While Althen contemplates that the support for a claimant's theory of causation is based on a "reputable medical or scientific explanation." 418 F.3d at 1278 (citations and internal quotation marks omitted); see also Knudsen, 35 F.3d at 548 (requiring a "sound and reliable medical or scientific explanation"), the support need not rise to the level of medical or scientific certainty for a petitioner to prevail on a vaccine claim. In Andreu, 569 F.3d at 1380, the Federal Circuit recently reiterated that submitted medical literature and epidemiological evidence "must be viewed . . . from the vantage point of the Vaccine Act's preponderant evidence standard:

> The standard of proof required by the [Vaccine] Act is simple preponderance of evidence; not scientific certainty. . . . [I]t is not plaintiff's burden to disprove every possible ground of causation suggested by defendant nor must the findings of the court meet the standards of the laboratorian.

(quoting Bunting v. Sec'y of Health and Human Servs., 931 F.2d 867 , 931 F.2d at 873 (Fed. Cir. 1991) (citations and internal quotation marks omitted)). When reviewing the

scientific evidence, a special master must take into account that "a finding of causation in the medical community may require a much higher level of certainty than that required by the Vaccine Act to establish a prima facie case." Broekelschen, No. 07-137, 2009 WL 2569734 at *5 (internal citation omitted).

The Federal Circuit also reiterated in Andreu the importance of considering medical records and medical opinion testimony in vaccine cases. Andreu, 569 F.3d 1367. Such testimony, explained the Circuit Court, can be "'quite probative' 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.'" 569 F.3d 1375 (quoting Capizzano, 440 F.3d at 1326 (citations and internal quotation marks omitted)). See also Althen, 418 F.3d at 1279-80 (noting that the use of "medical opinion as proof" of causation is contemplated under the Vaccine Act)."

Consistent with the Vaccine Act, however, a special master is not bound by any diagnosis, conclusion, judgment, test result, report, or summary contained in the record. 42 U.S.C. § 300aa-13(b)(1). Rather, the special master must consider the entire record and the course of the injury when evaluating the weight to be afforded to any offered diagnosis, conclusion, judgment, test result, report, or summary contained in the record. 42 U.S.C. § 300aa-13(b)(1).

III. Analysis

In support of his vaccine claim, Mr. Shaw has relied on the opinions of the treating physicians contained in his filed medical records as well as the opinion testimony of Dr. Tenpenny, who testified at hearing on his behalf. Questions about whether Dr. Tenpenny possessed the qualifications to offer an expert opinion on causation arose during the hearing. Because the issue of Dr. Tenpenny's qualifications is a pivotal one, the undersigned addresses it first.

A. Qualifications of Petitioner's Testifying Medical Witness, Dr. Tenpenny

Dr. Tenpenny attended Kirksville College of Osteopathic Medicine in Kirksville, Missouri. Tr. at 30. After graduating in 1984, she completed a one-year rotating internship. Id. She practiced emergency medicine from 1985 until 1998 in Finley, Ohio. See id. at 31-32. Desiring to "open an office practice to do primary care and to do osteopathic manipulation," she took a medical acupuncture course offered by University of California at Los Angeles in 1994, moved to Cleveland, Ohio in 1996, and opened a practice in 1998 treating women and children using both conventional and alternative

medicine. Id.

Dr. Tenpenny is a doctor of osteopathic medicine (known as a D.O. rather than a M.D.) and is board certified in emergency medicine and in osteopathic manipulative medicine. Id. at 30. Osteopathic manipulative treatment involves the use of a physician's hands to diagnose, treat, and prevent illness or injury. See http://www.osteopathic.org/index.cfm?PageID=ost_omt. An osteopathic physician moves a patient's muscles and joints using techniques including stretching, gentle pressure and resistance. Id. Rooted in a belief that all parts of the body work together and influence one another, the applied treatment is intended to assist in easing pain, promoting healing, and increasing mobility in a patient. Id. A D.O. receives special training that focuses on the nervous system and the musculoskeletal system (muscles and bones). Id.

The certifying body for doctors of osteopathic medicine is the American Osteopathic Association (AOA). Tr. at 34. Dr. Tenpenny explained that the titles of the specialties for which AOA certification is available have changed and her certification in osteopathic manipulative medicine is now equivalent to a certification in neuromuscular medicine. Id. There are currently eighteen areas of approved specialties for which the AOA can grant certification.[19] See http://www.osteopathic.org/index.cfm?PageID=ado_cert. The certification requirements vary by specialty. Id.

At hearing, Dr. Tenpenny described her training in diagnosing neurological conditions as the type of training that one receives as an emergency medicine physician. Tr. at 35. She testified that as part of her practice, she performs magnetic resonance imaging (MRI). Id. She added that as a fully trained and qualified physician, she is trained to read and assess medical literature. Id.

Dr. Tenpenny acknowledged that she is neither a neurologist nor an immunologist. Tr. at 34. She does not have any experience treating patients with the neurological conditions of transverse myelitis (TM) or chronic inflammatory demyelinating

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[19] The areas in which a doctor of osteopathic medicine can become board certified are: (1) Anesthesiology; (2) Dermatology; (3) Emergency Medicine; (4) Family Practice; (5) Internal Medicine; (6) Neurology and Psychiatry; (7) Neuromuskuloskeletal Medicine; (8) Nuclear Medicine; (9) Obstetrics and Gynecology; (10) Ophthalmology and Otolaryngology; (11) Orthopedic Surgery; (12) Pathology; (13) Pediatrics; (14) Physical Medicine and Rehabilitation; (15) Preventive Medicine; (16) Proctology; (17) Radiology; and (18) Surgery. http://www.osteopathic.org/index.cfm?PageID=ado_cert.

polyneuropathy (CIDP). Id. at 36. By her own admission, Dr. Tenpenny lacks the experience or training to testify as an expert in neurology or neuroimmunology. In addition to Dr. Tenpenny's own admission concerning the limitations of her expertise, petitioner's counsel limited the scope of Dr. Tenpenny's testimony. Tr. at 37 (stating "we are not holding her out to be a neurologist. She was one of Michael Shaw's treating physicians. . . .")

Nonetheless, it is Dr. Tenpenny's view that petitioner suffers from the neurological condition of TM or CIDP. She explained that her knowledge about TM and CIDP comes from reading conventional medical journals, understanding the material, asking questions, and coming to conclusions. Id. at 36.

Dr. Tenpenny further explained that she has strong concerns about vaccines, a subject on which she speaks and writes. See id. at 76. The focus of her revenue-generating speeches and writing has been the "problems that [she has] with some of the vaccination policies of a one-size-fits-all type of treatment program." Id. at 32-33, 78-79. She began her "personal investigation into vaccines in September, 2000[,]" and since that time, she has invested more than 7,500 hours "researching the often-overlooked association between vaccines and vaccine injuries as they affect individual persons." Pet.'s Ex. 45 at 4. She estimates that about 25 percent of her practice is devoted to the treatment of patients she believes have been vaccine-injured. Tr. at 33. Although she does not consider herself to be opposed to vaccines, she has compared vaccines to bio-terrorism. Id. at 33, 77.

As articulated at the hearing, petitioner did not offer Dr. Tenpenny as an expert in neurology. Id. at 37. Instead, petitioner offered Dr. Tenpenny as one of petitioner's treating physicians who had reviewed his medical records and the opinions of petitioner's other treating physicians and had developed her own opinion as to the cause of Mr. Shaw's injury. Id. Dr. Tenpenny possesses the skills, training, and experience of a doctor of osteopathic medicine. The undersigned accepted Dr. Tenpenny's testimony as a treating physician with expertise in manipulative treatment.

B. Opinion of Petitioner's Testifying Medical Witness, Dr. Tenpenny

Dr. Tenpenny personally treated petitioner on May 20, 2003 and June 3, 2003, nearly two and one half years after petitioner had filed his vaccine claim. Compare Tr. at 82-84 with Pet.'s Ex. 61[20]; see also Pet.'s Ex. 61. Although she was one of petitioner's

[20] Dr. Tenpenny testified at hearing that she treated petitioner in November 2003, but in the later filed statement of record unavailability, she corrected the dates of service for petitioner.

treating physicians, she did not submit any records of treatment in this case. Tr. at 83. She explained that the records of petitioner's treatment were lost in a fire in her office in July of 2006. Id.; Pet.'s Ex. 61.

Information about Dr. Tenpenny's treatment of petitioner may be gleaned, however, from other filed medical records. It appears from a reference in the medical records filed as Petitioner's Exhibit 25 that Dr. Tenpenny suspected and treated petitioner for mercury toxicity, a condition that he did not have. See Pet.'s Ex. 25 at 1. In the notes of interest from an "[e]ncounter [d]ate" of April 29, 2003, Dr. Harold Buttram of the Woodlands Healing Research Center wrote:

> Patient did see Dr. Tenpenny late last year into early this year, who put him th[r]ough allergy elimination (Bioset). However, when tested for Hg [(mercury)], he did not react. Last February he did have 6 of his "bad" Hg amalgams [(mercury-containing dental filings)] removed. Next he did a 500 mg oral dmsa urine challenge test [(mercury chelation)], which was in normal limits. The test did leave him "very sick" for about 3 weeks. He does plan to contact Dr. Tenpenny again but is not sure which way to go, as mercury does not appear to be his problem.

Pet.'s Ex. 25 at 1.

Dr. Tenpenny states that she formed her opinion that petitioner's injury is a vaccine-induced one based on her medical training, her treatment of petitioner, "an extensive, unbiased review of the literature," and a review of petitioner's medical records which include the opinions of other treating physicians. See Pet.'s Ex. 45 at 4; Tr. at 35-36, 38. It is Dr. Tenpenny's view that petitioner "has a vaccine-induced neuropathy . . . [a]ssociated with the hepatitis-B vaccine" he received. Tr. at 40. She asserts that this diagnosis is consistent with the diagnoses–suggested by several physicians who have examined petitioner–of "acute generalized neuropathic pain of unknown etiology and vaccine-induced neuroimmune dysfunction associated with hepatitis b vaccine." Pet.'s Ex. 45 at 4.

Dr. Tenpenny elaborated on her view concerning the nature of petitioner's injury, stating that "Mr. Shaw's initial presentation after receiving the hepatitis b vaccine strongly resembles acute transverse myelitis, [(TM)]. In addition, his condition also suggests an unusual form of CIDP, [(chronic inflammatory demyelinating polyneuropathy)]." Id. Noting that the four classic features of TM include: (1) arm and

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Compare Tr. at 82-84 with Pet.'s Ex. 61.

leg weakness, (2) severe pain, (3) sensory alteration, and (4) bowel and bladder dysfunction, Dr. Tenpenny observed that petitioner presented with these symptoms during the progression of his illness. Id.; Tr. at 45; see also Pet.'s Ex. 45A at 1 (NINDS Fact Sheet-TM[21]). Dr. Tenpenny further observed that "there is a possibility that [petitioner] had an unusual case of . . . CIDP[,] . . . a neurological disorder characterized by progressive weakness and impaired sensory function in the legs and arms." Pet.'s Ex. 45 at 5. CIDP "often presents with symptoms[, similar to petitioner's,] that include tingling or numbness (beginning in the toes and fingers), weakness of the arms and legs, fatigue, and abnormal sensations." Id. (footnote omitted); see also Pet.'s Ex. 45B at 1 (NINDS Fact Sheet-CIDP).[22] Dr. Tenpenny acknowledged during cross-examination that although she believed that petitioner's symptoms coincided with the symptoms associated with the conditions of TM or CIDP, she was not a neurologist and her opinion was not a diagnostic one. Tr. at 62.

Noting that most physicians who reviewed petitioner's case concluded that all of his diagnostic studies were normal, Dr. Tenpenny pointed out that there are reports in the medical literature that a patient with normal diagnostic studies can present with clinical symptoms of TM or CIDP. See Pet.'s Ex. 45 at 5-8; see also Tr. at 57 ("[M]any of the articles that I submitted into the record clearly document that patients can have neuropathic pain, transverse myelitis, Guillain-Barre, [and] CIDP, without having any objective findings."). Among the articles that petitioner's counsel filed in support of this proposition include the 2006 Said review article, filed as Petitioner's Exhibit 60. The author of the 2006 Said review article observed:

> In practice, the diagnosis of CIDP rests mainly on demonstration of an asymmetrical demyelinating process on electrophysiological studies. . . . [T]he [four defined] electrophysiologic criteria for CIDP . . . . which are mainly aimed at defining homogeneous groups of patients for research purposes, are fulfilled by only 50–60% of patients with typical clinical features of CIDP. . . . Thus, it is essential to interpret electrophysiological data in a clinical context and . . . even totally normal conduction studies should not be exclude the diagnosis of CIDP.

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[21] National Institute of Neurological Disorders and Stroke: Transverse Myelitis Fact Sheet. See http://www.ninds.nih.gov/disorders/transversemyelitis/detail_transversemyelitis.htm.

[22] National Institute of Neurological Disorders and Stroke: Transverse Myelitis Fact Sheet. See http://www.ninds.nih.gov/disorders/cidp/cidp.htm.

Pet.'s Ex. 60 at 295 (2006 Said review article) (emphasis added).[23]

Dr. Tenpenny further pointed to test results for petitioner that she asserted were "subtle signs . . . present early in his disease." Pet.'s Ex. 45 at 7. Specifically, she pointed to the mildly elevated protein level detected in petitioner's first spinal tap on December 6, 1999, six months after the alleged onset of his symptoms.[24] Id. Detection of an elevated protein level in cerebrospinal fluid (obtained by spinal tap) may be indicative of an infectious or inflammatory process. See Mosby's Manual of Diagnostic and Laboratory Tests at 681. Dr. Tenpenny also pointed to laboratory tests that showed a mildly elevated erythrocyte sedimentation (SED) rate for petitioner in March 2000, nearly nine months after petitioner first experienced the tingling and numbness in his toe that ascended to his knee and eventually developed in all of his extremities. See Pet.'s Ex. 45 at 8.[25] An erythrocyte sedimentation rate is a measure of the rate at which red blood cells settle in saline solution or plasma over time. Although the test is not specific for or diagnostic of any particular disease, it may be indicative of an inflammatory or infectious disease because such diseases increase the protein content of plasma and thus, produce higher SED rates. See Mosby's Manual of Diagnostic and Laboratory Tests at 233. Dr. Tenpenny admitted, however, that the laboratory results to which she pointed were not diagnostic of either TM or CIDP and petitioner's test results were generally normal.[26] Tr. at 65.

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[23] G. Said, Chronic inflammatory demyelinating polyneuropathy: A Review, Neuromuscular Disorders 16: 293–303 (2006).

[24] The normal range for protein was 15-45 mg/dL. See Pet.'s Ex. 45 at 7; see Pet.'s Ex. 2 at 48. Petitioner's protein level was 52. See Pet.'s Ex. 45 at 7; see Pet.'s Ex. 2 at 48.

[25] The normal range for a SED rate is less than 20. See Pet.'s Ex. 45 at 8; Pet.'s Ex. 6 at 6. Petitioner's SED rate was 31. Id. But compare this laboratory result to the SED rate which was well within the normal limits, was measured six months earlier (on September 8, 1999), and was noted by respondent's expert in his report. See Pet.'s Ex. 1 at 62 (reflecting a measured SED rate of 3 and a reference range of 0-15 mm/HG).

[26] Of concern to Dr. Tenpenny was the lack of imaging of petitioner's thoracic region (a thoracic MRI) among petitioner's test results. See Tr. at 67. She noted that the thoracic region of the spinal cord is the most common site of involvement in TM cases. Id. (citing K. H. Choi, et al., Idiopathic Transverse Myelitis: MR Characteristics. AJNR Am. J. Neuroradiol. 17(6):1151, 1156 (Jun-Jul 1996), which was filed as Pet.'s Ex. 58 and is referred to as the 1996 Choi article). Dr. Tenpenny testified that since her review of petitioner's medical records in 2003, she has recommended that petitioner obtain a thoracic MRI. Tr. at 86. Although she could have ordered one herself, she has not done so. Id.

In addition to her consideration of petitioner's symptoms and his laboratory test results, Dr. Tenpenny considered the post-marketing reports of adverse reactions to the hepatitis B vaccine that are listed on the package insert for Energix B, the type of hepatitis B vaccine that petitioner received. See Pet.'s Ex. 45 at 9; Pet.'s Ex. 45J at 8-9 (package insert); Tr. at 41. Among the post-marketing reports of adverse reactions in the nervous system are: (1) migraine; (2) fainting (or syncope);[27] (3) slight or incomplete paralysis (or paresis);[28] (4) peripheral nervous system affected by abnormally decreased sensitivity to touch (neuropathy including hypoesthesia);[29] (5) abnormal touch sensation (or paresthesia);[30] and (6) transverse myelitis. Pet.'s Ex. 45J at 8-9. Dr. Tenpenny pointed out that petitioner experienced some of the adverse reactions identified on the package insert for the hepatitis B vaccine that he received. Tr. at 41, 43. Dr. Tenpenny agreed, during questioning on cross-examination, that the listed post-marketing adverse events on the package insert for hepatitis B vaccine were "just reports that have been made and were not actually studies that have been done to establish a causal association."[31] Tr. at 71-72.

Dr. Tenpenny noted that petitioner received the hepatitis B vaccine in question approximately one month after his first hepatitis B vaccine. Tr. at 38. Relying on petitioner's later-given testimony rather than on the contemporaneous medical records, Dr. Tenpenny testified that "[w]ithin 48 hours[, he] began to experience severe numbness, burning, and as he described it, excruciating pain in his left big toe." Id. The symptoms claimed to have developed within forty-eight hours of the second hepatitis B vaccination "progressed rather rapidly, within 10 days or so from the time of the onset of symptoms." Id. at 48. Based on the "time line involved" between petitioner's vaccination and the onset of petitioner's symptoms, it is Dr. Tenpenny's opinion that the hepatitis B vaccine is causally associated with the sustained injury. Tr. at 40, 48.

Dr. Tenpenny added, "There are a lot of reports in the medical literature of patients

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[27] See Dorland's at 1807.

[28] See Dorland's at 1371.

[29] See Dorland's at 894, 1257.

[30] See Dorland's at 1371.

[31] Dr. Tenpenny explained that post-marketing reports of adverse events are gathered in an effort to determine whether "a vaccine is causing a problem" that was not detected during the research process. Tr. at 80. The reports also are helpful in identifying particular vaccine lots that are causing problems. Id. Such vaccine lots are known as "hot lots." Id.

who have had the hepatitis[]B vaccine and developed neuropathies, and one of the working theories on that is the principle of molecular mimicry." Tr. at 40-41. Dr. Tenpenny described the process of molecular mimicry:

> [W]hen you receive a vaccine and you develop an antibody[,] . . . the purpose of the antibody is to seek out a virus or bacteria that may have infected the body. The antibody is looking for a particular amino acid sequence on the side of that virus or bacteria. For the sake of clari[t]y, let's call that amino acid sequence ABC. So [the developed] antibody's looking around for amino acid sequence ABC, and if you're not infected with the virus or bacteria it has nothing to attach to, to neutralize.
>
> So then it finds that same amino acid sequence of ABC on a nerve, a myelin sheath, a pancreas, a variety of different parts of the body, and attac[hes] itself to that. The [antibody to] hepatitis-B surface antigen has been shown to attach to the myelin sheath [instead of the sought after viral surface material]. . . . [T]his [is the] principle of molecular mimicry.

Tr. at 42. As support for her theory of hepatitis B vaccine-induced demyelination, Dr. Tenpenny pointed to the 2001 Karaali-Savrun article,[32] filed as Petitioner's Exhibit 45L. See Tr. at 45-46. As Dr. Tenpenny correctly described at hearing, the article reports four cases of acute myelitis that developed within three months after the administration of the recombinant form of hepatitis B vaccine, which is the form of vaccine that petitioner received in this case. Pet.'s Ex. 45L at 711 (2001 Karaali-Savrun article); Tr. at 45-46. The authors of the article wrote that the case reports "suggest[] a possible relation between vaccination and demyelination[, and] [i]t can be speculated to be [either] an autoimmune cross[] reaction between a protein of the hepatitis B vaccine and the nervous system (molecular mimicry) or a possible reactivation of a dormant [wild hepatitis B] virus." Pet.'s Ex. 45L at 714 (internal citation omitted). The authors added that a "[p]robable causal link between hepatitis B vaccination and myelitis is suggested by: the temporal association between events, the previous reports of myelitis following the hepatitis B vaccine and no clinical or laboratory evidence suggestive of other underlying disorders." Id.

Dr. Tenpenny described another theory "regard[ing] . . . how the hepatitis-B vaccine can cause these types of nervous system . . . problems." Tr. at 42-43; see also Pet.'s Ex. 45 at 10. She termed the theory that she addressed as "an autoimmune

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[32] F. Karaali-Savrun et. al, Hepatitis B Vaccine Related-Myelitis? Eur. J. Neur. 8:711-715 (2001).

inflammatory reaction." Tr. at 43. She explained that it was her belief that such reaction involves "a cytokine pathway" that allows the body to react to itself and become increasingly inflamed "along different types of sheaths in the body." Id. "[T]he myelin sheath is broken down around the nerves [and no longer] conduct[s] the nerve transmissions as well." Id. Inflammation and pain result. Id.

In support of this particular theory of vaccine-related causation, Dr. Tenpenny pointed to the 1996 Choi article, filed as Petitioner's Exhibit 58,[33] in which the authors stated that "[t]ransverse myelitis . . . is diagnosed when both halves of the [spinal] cord are involved in an inflammatory process. The syndrome . . . is known to be associated with various viral infections, vaccinations, autoimmune diseases, and carcinomas, although most cases are idiopathic [(of unknown causes)]." Pet.'s Ex. 58 at 1151 (1996 Choi article) (footnotes omitted); see also Tr. at 45. Dr. Tenpenny explained that the involvement of the spinal cord in TM leads to bilateral motor and autonomic dysfunction that may manifest as "urinary retention, . . . weakness of the arms and legs, [and] pain." Tr. at 45. Dr. Tenpenny noted that petitioner has exhibited such symptoms during the progression of his illness. Id.

Impressed by the timing of the onset of petitioner's symptoms following his second hepatitis B vaccine, Dr. Tenpenny testified that the medical literature informs that autoimmune reaction after vaccination "tend to appear somewhere between one and 30 days." Tr. at 56-57. The 48 hours between petitioner's vaccination and the onset of his toe tingling symptom falls within that medically recognized time frame. Id. at 57.

The temporal association between the vaccine administration and the onset of symptoms, the absence of clinical or laboratory evidence of another cause, the support in the medical literature for an association between hepatitis B vaccine and TM, and legal precedent in the vaccine program for an association between hepatitis B vaccine and CIDP are among the factors that persuade Dr. Tenpenny that petitioner's disability is vaccine-induced. Pet.'s Ex. 45 at 10. Dr. Tenpenny conceded during her testimony that although she pointed to legal cases finding that the sustained injuries of TM and CIDP were compensable under the Vaccine Act, doctors do not typically rely on legal precedent in forming medical opinions. Tr. at 70. While Dr. Tenpenny declined to consider the impact of petitioner's prior traumatic injuries on his current condition, she did attribute some of his symptoms, specifically, his dizziness and depression, to the medications he has begun to take since the onset of his symptoms in June of 1999. Id. at 74-76.

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[33] See, supra, footnote 24.

Additional factors that persuade Dr. Tenpenny that petitioner's condition is vaccine-related are notations in petitioner's medical records by some of his treating doctors that either associate or potentially associate the received hepatitis B vaccine with the condition that petitioner has. See Tr. at 51- 56. The conclusion by a number of petitioner's other physicians that the cause of his problems was uncertain or, as suggested by one doctor, was possibly psychological did not dissuade Dr. Tenpenny from her opinion.[34] See Tr. at 66-68.

Convinced that petitioner's condition is causally related to the second hepatitis B vaccine that he received and of the opinion that hepatitis B surface antigen "could be" still in petitioner's body, Dr. Tenpenny addressed petitioner's lack of measurable hepatitis B titers when tested more than two years after the vaccination at issue, in September 2001. Tr. at 60-61, 73. In the report prepared by respondent's expert, Dr. Leist, the lack of a measurable hepatitis B surface antigen titer was identified as evidence that petitioner did not mount a significant immune response to the vaccine and thus could not have suffered a vaccine-related injury. Respondent's Exhibit (Resp.'s Ex.) A at 10-11. Dr. Tenpenny asserted that petitioner's lack of a measurable hepatitis B surface antigen titer more than two years after his vaccination did not "rule[] out the fact that Michael was injured by the vaccine at the time it was given." Tr. at 61. Dr. Tenpenny explained that according to the Centers for Disease Control (CDC), patients can be nonresponders or low responders to any type of vaccine, and in particular, the hepatitis B vaccine for several reasons, including: (1) genetic factors; (2) failure to receive the full vaccination series, the last of which is typically the biggest booster for the development of antibodies; or (3) the rapid decline of titer levels in a patient within the first year after the immunization. Tr. at 60-61; see also Principles of Vaccination: Immunology and Vaccine-Preventable Disease at 5, available at http://www2a.cdc.gov/nip/isd/immtoolkit/content/products/pinkbook.pdf (stating, "Inactivated vaccines[, such as hepatitis B] always require multiple doses. In general, the first dose does not produce protective immunity, but only "primes" the immune system. A protective immune response develops after the second or third dose. . . . Antibody titers against inactivated antigens . . . diminish with time.").

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[34] On April 30, 2003, Richard Ivins, Ph.D., a licensed psychologist evaluated petitioner in connection with his worker's compensation claim. See Pet.'s Ex. 22 at 1, 9. Dr. Ivins noted that there was some evidence of symptom exaggeration as reflected in the results of one of the tests administered to petitioner. Id. at 7. Dr. Ivins opined that "it would appear that the present difficulties are more psychological than neuropsychological in nature. There is no doubt that Mr. Shaw is experiencing some rather significant emotional difficulties, and this may well answer most of the questions with regard to his problems." Id. at 9.

C. Opinion of Respondent's Expert Witness, Dr. Leist

To address the opinion offered by Dr. Tenpenny, respondent offered the opinion and testimony of Dr. Leist, who serves as Chief of the Division of Neuroimmunology and Director of the Comprehensive Multiple Sclerosis (MS) Center at Thomas Jefferson University in Philadelphia, Pennsylvania. Resp.'s Ex. B at 1. Having obtained a doctorate in biochemistry from the University of Zurich in 1985 and a medical degree from the University of Miami in 1993, Dr. Leist has received postgraduate training in the areas of pathology, microbiology, immunology and neurology. Id. Describing himself as a bench-trained immunologist "with strong interests in general immunology and viral immunology," he has focused, through his training, on diseases that are immunological in nature and affect the nervous system. Tr. at 89. Board-certified by the American Board of Psychiatry and Neurology, he sees approximately 30 to 40 cases of TM every year. Id. at 91. At the time of the hearing, he also had about 60 patients carrying a diagnosis of CIDP that he was seeing in concert with the neuromuscular group at the university. Id. at 92. The undersigned accepted Dr. Leist as an expert in the areas of immunology and neuroimmunology. Id.

Dr. Leist opined that "there is no evidence that [petitioner] has suffered a neurological injury as a part of this vaccination." Tr. at 93; see also Resp.'s Ex. A at 12 (expressing "opinion that Mr. Shaw did not experience an injury [as] a consequence of the hepatitis B or for that matter any other vaccination whether listed in the Vaccine Injury Table or not contained therein").

Addressing the theory of vaccine causation advanced by Dr. Tenpenny, Dr. Leist described the distinctions between the two conditions of TM and CIDP. See Tr. at 93-100. He explained that TM is a condition involving "inflammation in the spinal cord." Id. at 93. The spinal cord is:

> a tract of nerve fibers that bring information from the brain to the periphery, make the muscles move, . . . and brings information back from the periphery to the brain in several layers . . . to tell us what the environment is like, . . . [and] where our limbs are, it gives us information regarding the location.
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> So the spinal cord is very topographically oriented. Certain functions are contained in certain tracts. And if this tract is interrupted then there is an associated symptom associated with this interruption or the dysfunction. [The] spinal cord is not a tube where the information flows in a certain random order; there are locations in the spinal cord and there are

> therefore well-recognized syndromes when the spinal cord is injured at the certain level in a certain way.

Id. at 93-94. It is this topographical orientation of the nervous system that permits neurologists to determine, based on the constellation of symptoms that the person has, where a lesion is likely to be. Id. at 94. In other words, where the lesion is located along the axis of the spinal cord determines what sensory level is affected and what symptoms develop; sensory abnormalities occur below the affected sensory level. See id. at 95; see also id. at 119 ("[W]here the lesion is matters very significantly with respect to . . . the clinical presentation."). If, for example, a lesion occurred in the thoracic region at level four (denoted as T-4), sensory abnormalities would be expected to appear "below the nipple line" on the chest. Id. at 96. And if the lesion occurred "at about T-10, . . . close to the end of the spinal cord," then sensory abnormalities would be expected to appear below the belly button. Id. The expected disturbances in function "are in [a descending] anatomical order." Id. Accordingly, thoracic lesions generally do not produce the symptom of confusion because thoracic lesions do not affect the brain. Id. The appearance of confusion as a symptom must be explained by a cause other than a thoracic lesion. Id.

Dr. Leist added that the size of the lesion determines the extent of the injury and affects how a person is going to present clinically. Id. at 119. For example, when a patient presents with bilateral sensory disturbances or bilateral weakness, "the lesion has to be a certain size." Id. at 121. When a patient's presentation suggests that a larger lesion exists, the lesion is "less likely" to elude detection on imaging. Id. Dr. Leist testified that the initial presentation of Mr. Shaw's symptoms in July 1999 "were not consistent" with TM. Id. at 97.

Dr. Leist explained that CIDP "affects the peripheral nervous system, and refers to a certain course of an inflammatory injury to the peripheral nervous system." Id. at 95. The condition does not result from an injury to the spinal cord, but rather from an injury to the nerve root out in the peripheral nerves. Id. at 96-97. The diagnostic criteria for CIDP include a finding of "neurological dysfunction attributable to the peripheral nerve[s] . . . demonst[rab]le in more than one group of peripheral nerves, [and] lasting for more than a month." Id. at 97-98. The condition "is an ongoing disorder that . . . very often . . . has periods of quiescence and periods of recurrence." Id. at 99. Although petitioner's symptoms may have been suggestive of potential CIDP by July 1999, a month after the onset of petitioner's symptoms, Dr. Leist testified that subsequent neurological examinations "did not provide evidence of clear peripheral involvement." Id. at 98.

In addition to expressing his own professional doubt that petitioner suffered from

either TM or CIDP, Dr. Leist noted that petitioner's treating neurologists and other treating physicians did not treat him as though he had either TM or CIDP. Tr. at 98-100. The available medical records do not reflect that he received the type of treatments that those conditions warrant. Id. at 100. Observing that petitioner "was seen in good [medical] centers, at good places," Dr. Leist opined, "I would assume that if [the treaters] would have had reasonable clinical suspicions that [petitioner suffered from one of the conditions suggested by Dr. Tenpenny,] they would have offered him these treatments." Id.

Dr. Leist addressed the lack of objective evidence to support petitioner's "difficult clinical constellation" of symptoms. See Tr. at 100-102. The conducted MRIs were within normal limits; "no lesions within the brain or the [spinal] cord . . . [were] imaged, visualized." Id. at 101. The "electrophysiologic testing to look for a dysfunction of the peripheral nerves, namely . . . whether . . . the nerve fibers[] were dying[,]" did not show the delays in nerve conduction that would support a finding of a "demyelinating neuropathy." Id. And the results of the conducted biopsies "were not in line with a neuropathy or a demyelinating neuropathy." Id. at 102. Noting that the purpose of such tests is to support a diagnosis, Dr. Leist testified that it is "very close to 100 percent" that a disease process that has eluded detection over time after a number of subsequent evaluations does not exist. Id. at 101, 103-104. He further testified that "[it is] certainly not in the center of what [he] know[s]" professionally to have a patient with a severely disabling case of either TM or CIDP but without any objective findings from MRIs, electrophysiological studies, and biopsies. Id. at 104.

Dr. Leist criticized Dr. Tenpenny's reliance on excerpted sentences from medical publications to support her opinion that petitioner may have suffered from neurological conditions that could not be established by any of the clinical examinations that petitioner had. Id. at 115. Dr. Leist described the analytical process employed by clinicians, explaining that "as clinicians[, we] . . . have a certain hierarchy of evidence that we require." Id. First in the hierarchy of considered evidence is the clinical exam. See id. When an exam reveals that particular aspects of a condition are missing, the question of whether the patient has the condition arises, and any consulted medical literature must be viewed in proper context. See id.

In addition to the lack of objective evidence that petitioner suffered a neurological injury, Dr. Leist discussed the shortcomings in Dr. Tenpenny's theory of causation for this particular petitioner. Dr. Leist stated that if, as petitioner has alleged, the suffered injury in this case is an ongoing, progressively worsening condition associated with the hepatitis B vaccine, then "the injurious mechanism needs to be present when we look." Tr. at 104-105. Dr. Leist explained:

> [T]he only way that [the hepatitis B vaccine] could cause [injury] is by inducing a cross-reactive immune response, [which is] an immune response that is . . . self-reactive. In order to do this, there needs to be an immune response.
>
> [To] . . . test for that, . . . we can do a hepatitis-B surface antigen antibody test and see whether that's present. . . . [A]t the time when Mr. Shaw had progression of his injury, . . . the hepatitis-B surface antigen antibody wasn't present.

Id. at 105. Because "hepatitis-B surface antigen contained within the vaccine gets taken up [by particular cells], is processed, presented, and then induces the immune response[,]" the hepatitis B surface antigen protein found in the vaccine is degraded and cannot persist in "long-lasting circulation . . . in the body." Id. at 106-107. Dr. Leist clarified that contrary to the suggestion by Dr. Tenpenny that hepatitis B surface antigen "could be" in petitioner's body years after the hepatitis B vaccination at issue, only those persons with an active or a chronic hepatitis-B infection will be found to have hepatitis-B surface antigen floating over time within their bodies. Id. Absent any evidence that petitioner in this case has an active hepatitis B infection, it would be unlikely that vaccine-related hepatitis B surface antigen would be present years later. See id. Dr. Leist found no evidence in the record "of an ongoing immune response to the hepatitis-B [vaccine]." Id. at 130. Addressing Dr. Tenpenny's suggestion in her testimony that immunological nonresponders and low responders to vaccines could sustain injury nonetheless from an administered vaccine, Dr. Leist testified that in his view, petitioner's lack of a measurable immunological response in titers to the administered hepatitis B vaccine, when considered with petitioner's negative findings on his neurological exams, his MRIs, and his electrophysiologic testing, substantially diminishes the likelihood that petitioner has suffered a vaccine-related, immunologically-induced injury. See id. at 140-143.

Dr. Leist did offer other possible explanations for petitioner's symptoms. Pointing first to petitioner's various medications, Dr. Leist stated that petitioner's chronic opioid use, as part of his prescriptive therapy, "cannot be set aside" given petitioner's "mood and psychosomatic and psychiatric presentation" as well as the record evidence of efforts to manage petitioner's dependence on such medications. Id. at 108-109. Dr. Leist observed that in his experience at the MS clinic that he oversees, "about 25 percent of [the] patients that [he] see[s] use marijuana as part of their self-associated treatment for MS or MS symptoms, not by [his] advice, obviously. . . . But they do it. And we deal on a regular basis with mood alterations, psychiatric side effects, marital problems, and other problems as a consequence of chronic marijuana use." Id. at 109. In Dr. Leist's view, the mood alterations, personality changes, and cognitive impairment experienced by

petitioner were consistent with chronic marijuana use. Id.

Dr. Leist then addressed petitioner's history of multiple traumatic injuries, including fractures in both legs, a fractured hip, and fractured hands. Id. at 109-110; see also Pet.'s Ex. 4 at 11; Pet.'s Ex. 8 at 17; Pet.'s Ex. 25 at 39; Pet.'s Ex. 32 at 44. Such traumas can lead to entrapment neuropathies by restricting the free movement of nerves in an area when the broken bone calcifies or heals itself. Tr. at 99, 110-111. The calcification process occurs over time and the healing patient can subsequently experience pain in the previously injured areas. See id. at 111-113.

Petitioner's records also include references to facial lacerations and concussive head injuries that petitioner sustained as well as dental work that petitioner required as a result of his sports-related accidents. Id. at 110; see also Pet.'s Ex. 2 at 4. The long-term effects of such earlier injuries may begin to become apparent as the body ages. See Tr. at 112-113; see also Resp.'s Ex. A2 (2006 Omalu article[35]) (discussing the impact of repetitive traumatic brain injury on psychiatric functioning of football players). Dr. Leist noted that quantifying the period of time between the precipitating injury and the resulting entrapment neuropathy or pain syndrome is "difficult." Tr. at 111.

Dr. Leist made clear during cross-examination that although the Institute of Medicine[36] has said it is "possible to put a mechanism together by which hepatitis-B could cause an injury"–a statement that could be construed as support for the general theory of hepatitis B vaccine-related causation, consideration must be given "in an individual case" to whether there is sufficient evidence to put together such a causal sequence. Id. at 116-117; see also 2002 IOM Report.[37] Based on the dearth of objective evidence of "any clear neurological abnormality" in petitioner's voluminous records of

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[35] B. I. Omalu et al., Chronic Traumatic Encephalopathy in a National Football League Player: Part II, Neurosurgery 59: 1086-1093 (2006).

[36] Congress created the National Academy of Sciences by An Act of Incorporation in 1863 to advise the federal government on scientific and technical matters. See An Act to Incorporate the National Academy of Sciences, ch. 111, 12 Stat. 806 (1863), codified as amended, 36 U.S.C. § 150303 (1998). Under the charter of the National Academy of Sciences, the Institute of Medicine (IOM) was established in 1970 to serve as an advisor to the nation on health issues. See www.iom.edu (last visited on 2/1/09). When enacting the Vaccine Act in 1986, Congress further charged the IOM with conducting studies to explore whether any causal relationships might exist between vaccines and injuries. See 42 U.S.C. § 300aa-1 note.

[37] Immunization Safety Review: Hepatitis B Vaccine and Demyelinating Neurological Disorders (K. Stratton et al., eds., The National Academies Press 2002).

treatment over time, Dr. Leist is not persuaded that petitioner has suffered a vaccine-related injury. Id. at 123-125.

When questioned about a letter dated May 7, 2007 from by Dr. Lomen-Hoerth, one of petitioner's treating neurologists, noting that petitioner clinically "appears to have a progressive small-fiber neuropathy, with documentation on skin biopsy suggestive of an early small-fiber neuropathy," Dr. Leist indicated that the letter posed two particular problems for petitioner's vaccine-injury claim. See Tr. at 126-128 (discussing Pet.'s Ex. 54 at 2). First, the characterization of petitioner's injury as merely "suggestive of an early small-fiber neuropathy" in 2007 underscored the medical uncertainty that persisted--eight years after the onset of petitioner's symptoms--concerning the nature of petitioner's injury. Id. at 127 (emphasis added). Second, the diagnostic impression of an emerging small fiber neuropathy undercuts petitioner's demyelination theory of causation because small nerve fibers are not myelinated. Id. While the Institute of Medicine has examined whether hepatitis B vaccine can cause injury by provoking demyelination, there is no evidence--of which Dr. Leist is aware--that hepatitis B vaccine can lead to direct injury of nerve fibers and thereby cause injury "independent of demyelination." Id. Dr. Leist asserted that absent a demyelinating injury, petitioner "would have to suggest a completely different mechanism of how [the hepatitis B] vaccine [caused] injury." Id.

Dr. Leist points out that while some of petitioner's treating doctors "entertained the possibility" that he had suffered a hepatitis B vaccine-induced injury in the absence of a better understanding of petitioner's chronic pain syndrome, other examining physicians suggested that petitioner may have suffered from a conversion disorder. Id. at 128-129. As described by Dr. Leist, a conversion disorder "is . . . a psychiatric diagnosis . . . where there is no actual physical injury occurring but the patient responds with a psychiatric illness that manifests itself as clinical symptoms." Id. at 129. Dr. Leist distinguished a conversion disorder from a malingering syndrome, explaining that a malingering syndrome has a "volitional component in it." Id. By contrast, a patient with a conversion disorder, "unbeknownst to himself, develops a symptom complex that can be very debilitating[] and is very real." Id.

Dr. Leist was emphatic during his testimony that although he presented alternative explanations for petitioner's symptoms, his opinion that petitioner's injury is not a vaccine-related one was based, not on the alternative explanations he had considered but rather "on the hard facts" of findings on examination and test results–facts on which he relies daily in his practice as a neurologist evaluating presented injuries. Id. at 145.

D. Evaluating the Testimony of the Parties' Witnesses

Before turning to address the merits of the opinions offered by the parties' witnesses, the undersigned turns to address the relative credibility and persuasiveness of the witnesses' testimony.

As recently observed by the court in Broekelschen v. Secretary of Health and Human Services, evaluating the testimony of the experts "in the context of the entire record" to determine which expert is more persuasive "is the job of the special master." 2009 WL 2569734, at *8 (Fed. Cl. Aug. 4, 2009) (emphasis added) (citing De Bazan v. Sec'y of Health & Human Servs., 539 F.3d 1347, 1353 n.4 (Fed. Cir. 2008) ("[T]he special master admitted and weighed both parties' evidence but simply decided that the government's evidence was more persuasive."). The court in Broekelschen further observed that the Federal Circuit's recent decision in Andreu "did not alter the causation standard or the standard of review applicable in Vaccine Act proceedings." 2009 WL 2569734, at *8 (Fed. Cl. Aug. 4, 2009) (citing Andreu v. Sec'y of Health & Human Servs, 569 F.3d 1367, 1379 (Fed. Cir. 2009). As the court expressed in Broekelschen, once a special master has considered the relevant evidence of record, has drawn plausible inferences from that evidence, and has articulated a rational basis for his (or her) findings, the special master's findings merit deference.

In this case, Dr. Tenpenny testified not as an expert but as one of petitioner's treating doctors. Although she conceded that she lacks the training to testify as an expert in either neurology or neuroimmunology, Tr. at 34, she nonetheless offered her view that petitioner's symptoms were suggestive of two different neurological conditions, specifically either TM or CIDP. Admitting that her opinion concerning the nature of petitioner's injury was not a diagnostic one, Dr. Tenpenny acknowledged that she had no experience treating patients with either TM or CIDP. See Tr. at 36. She explained that she formed her opinion concerning the nature of petitioner's injury and concerning the causal relationship between the hepatitis B vaccine that petitioner received and his injury based on her review of petitioner's medical records and her research of the medical literature. And in further support of her opinion, she supplied copies of vaccine cases in which petitioners who had either TM or CIDP and had received a hepatitis B vaccine were able to prove that their claims were compensable under the Vaccine Program.

The difficulty with Dr. Tenpenny's offered medical opinion in this case is that by her own admission she lacks the professional experience and training to address petitioner's particular injury. Dr. Tenpenny did not treat petitioner for the injury she alleges that he now suffers. Rather, she treated him for what she suspected was mercury toxicity, a condition that, contrary to Dr. Tenpenny's belief, petitioner did not have. Dr. Tenpenny's assertion here that her medical training qualifies her to offer the opinion that she has in this case misses the mark. An offered expert or medical opinion in support of a

vaccine claim is more persuasive when the offered opinion is well-informed and is rooted in soundly explained medicine or science. See 42 U.S.C. § 300aa-13(a) (requiring a medical or expert opinion in support of a petitioner's vaccine claim); Knudsen, 35 F.3d at 548 (requiring, as support for a claimant's theory of causation, a sound and reliable medical or scientific explanation). Moreover, the value that a specialist brings to an evaluation of particular medical problems cannot be ignored. In the view of the undersigned, there can be no serious dispute that an opinion, evaluation, or diagnosis of a heart problem given by a competent cardiologist may be found to be more persuasive than an opinion or evaluation of a heart problem given by a competent podiatrist–even if the podiatrist has studied medical literature pertaining to heart problems.

Although Dr. Tenpenny states that her opinion is not a diagnostic one, she offers an opinion concerning the nature of petitioner's injury that none of petitioner's many treating physicians, including numerous examining neurologists, has advanced. Not only has Dr. Tenpenny attributed petitioner's injury to neurological conditions that are at variance with the consensus diagnostic condition that petitioner's many treating doctors have put forward, she has done so without objective examination findings and test results that support her view of petitioner's condition. Dr. Tenpenny's opinion about the nature of petitioner's injury is essential to her theory of vaccine causation in this case. But, her opinion is not based on relevant medical experience or specialized training that she possesses or on petitioner's own medical record as a whole.

In contrast, as a neuroimmunologist, Dr. Leist possesses both professional experience and specialized training to address neurological conditions of the type that Dr. Tenpenny asserts petitioner has. Dr. Leist's patient load includes both patients with TM and patients with CIDP. See Tr. at 91-92. Dr. Leist specifically addressed in his testimony both the clinical presentation expected for the two neurological conditions and the objective exam findings and test results that would support a finding–if not at the time that petitioner's symptoms first appeared, then certainly eight years after symptom onset—that petitioner had one of the two neurological conditions. Although Dr. Leist was of the opinion, based on petitioner's test results and exam findings over the course of time, that petitioner's injury was not a neurological one, he testified that even if he were to accept the consensus diagnostic opinion of a small fiber polyneuropathy found in petitioner's medical records, the theory of causation that Dr. Tenpenny has proposed—specifically, vaccine-induced demyelination either through molecular mimicry or an autoimmune inflammatory reaction–cannot account for petitioner's alleged injury because the small nerve fibers suspected to be affected by some of petitioner's treating doctors do not have myelin sheaths. Without myelin sheaths surrounding the affected small nerve fibers, petitioner's theory of demyelination cannot be sustained.

Having considered the testimony of the parties' witnesses carefully, the undersigned finds the testimony of Dr. Leist to be more persuasive than the testimony of Dr. Tenpenny. Informed by both specialized training and his clinical experience, Dr. Leist offered an opinion that fully contemplated the course of petitioner's injury, the various diagnoses that petitioner has received, and the results of the objective testing that petitioner has received. By contrast, Dr. Tenpenny offered an opinion that petitioner has one of two neurological conditions that: (1) by her own admission, Dr. Tenpenny has neither the training nor experience to diagnose or treat; (2) none of petitioner's other treating doctors, including neurologists, has identified; and (3) none of the performed testing has confirmed. It appears that Dr. Tenpenny's opinion was prepared to support a finding of vaccine compensation on grounds similar to those put forth in the vaccine cases attached to her written opinion. Upon close examination, however, the basis for her offered opinion is not consistent with the medical facts of petitioner's particular case.

E. Evaluating Petitioner's Claim under the Althen Prongs

As stated earlier, petitioner must prove causation by showing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing the vaccination was the reason for the injury; and (3) a proximate temporal relationship between the vaccination and the injury. Althen, 418 F.3d at 1278. The undersigned addresses each of the prongs of the Althen standard in turn. For ease of discussion, the undersigned addresses the first and the third prongs of the Althen before turning to the second prong.

1. Petitioner's Offered Medical Theory

Petitioner must offer a medical theory causally connecting the vaccination and the injury. Althen, 418 F.3d at 1278.

Impressed by both the constellation of petitioner's symptoms and the timing of the onset of petitioner's symptoms, Dr. Tenpenny proposed a theory of causation based on her view that petitioner's symptoms were consistent with the symptoms that present in cases of either TM or CIDP. She asserted that petitioner had suffered a hepatitis B vaccine-induced demyelination. See Tr. at 40-46. She explained that the destruction of the myelin sheath surrounding petitioner's nerve fibers was the cause of his neurological problems. See id. at 42-43.

She further explained that the hepatitis B vaccine can cause demyelination by one of two biological mechanisms. First, she stated, through a process of molecular mimicry, "hepatitis-B surface antigen [antibody] has been shown to attach to myelin sheath" in the

body rather than to the intended viral target. See id. Such attachment can lead to demyelination. See id. at 45-46. Alternatively, she stated, the hepatitis B vaccine can provoke "an autoimmune inflammatory reaction" by triggering a cytokine response that leads to increasing inflammation and then to destruction of the myelin sheath around the nerve fibers. Id. at 42-43.

As support for her theory, she pointed to case reports in the medical literature that "suggest[] a possible relation between [hepatitis B] vaccination and demyelination." See Pet.'s Ex. 45L at 711 (2001 Karaali-Savrun article); Pet.'s Ex. 45 M at 99 (2005 Girard article).[38]

Respondent's expert did not challenge petitioner's theory of causation as a possible general theory of causation. Rather, on cross-examination, Dr. Leist acknowledged that the Institute of Medicine has recognized the possibility that a biological mechanism exists by which hepatitis B vaccine could cause injury. See Tr. at 116-117. But, Dr. Leist cautioned that an individual case must be examined to determine whether there is sufficient evidence to support such a causal sequence.

Having proposed a medical theory causally connecting his hepatitis B vaccination to an injury that petitioner's medical witness alleged that he has, petitioner has satisfied the first prong of the Althen standard.

Because the timing of onset of petitioner's symptoms was striking to Dr. Tenpenny and helped inform her theory of causation, the undersigned turns next to address the third prong of the Althen standard before reaching the second prong.

2. The Temporal Relationship between the Vaccination and the Injury

Petitioner must show more than a proximate temporal relationship between the vaccination and the injury to satisfy the burden of showing actual causation. Althen, 418 F.3d at 1278; see also Grant v. Secretary of Health and Human Services, 956 F.2d 1144, 1148 (Fed. Cir. 1992). Specifically, petitioner must demonstrate that his first symptoms of toe numbness and tingling occurred in a time frame that would be consistent with an immune-mediated disorder caused by the hepatitis B vaccine at issue.

Here, petitioner asserted in his affidavit and at hearing that his symptoms began

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[38] M. Girard, Autoimmune Hazards of Hepatitis B Vaccine, Autoimmunity Reviews 4: 96-100 (2005).

two days after he received his second hepatitis B vaccine. The contemporaneous medical records, however, indicate that petitioner's symptoms began six days after the receipt of his second hepatitis B vaccination. As addressed earlier in this ruling, the undersigned need not resolve the issue of when petitioner's first symptoms occurred because the two-day period of onset alleged by petitioner in his later-prepared affidavit and at hearing as well as the six-day period of onset reflected in the contemporaneous medical records fall within the time frame that petitioner's witness, Dr. Tenpenny, asserts is proper for an autoimmune injury. According to Dr. Tenpenny, that time frame is between one and thirty days. Tr. at 56-57. Respondent's expert, Dr. Leist, did not rebut this testimony by Dr. Tenpenny. Because symptoms of petitioner's injury occurred within an appropriate medical time frame for an immune-mediated injury, petitioner has satisfied the third prong of the Althen standard.

The undersigned turns now to address the logic of petitioner's proposed sequence of cause and effect.

3. The Sequence of Cause and Effect

The Federal Circuit has observed that an offered medical theory is persuasive when accompanied 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[.]'" Althen, 418 F.3d at 1278 (quoting Grant, 956 F.2d at 1148). Here, because the asserted medical theory is premised upon medical conditions that the record fails to show that petitioner has, the undersigned finds that petitioner has not established a logical sequence of cause and effect between the received vaccine and his actual injury.

Petitioner's medical records reflect different views about the precise nature of his injury. What is consistently reported is that the condition involves a chronic pain syndrome and is a progressive one. See Pet.'s Ex. 5 at 28; Pet.'s Ex. 9 at 107; Pet.'s Ex 13 at 165; Pet.'s Ex. 53 at 1. There is some agreement among petitioner's various treating doctors that some of the symptoms that he has experienced may be attributable to the medications he has taken in connection with his condition. See Pet.'s Ex. 13 at 5; Pet.'s Ex. 53 at 2. A careful review of petitioner's records indicates that to the extent that petitioner's injury may be a neurological one, the consensus diagnosis is that petitioner suffers from a small fiber neuropathy. See Pet.'s Ex. 4; Pet.'s Ex. 9 at 107; Pet.'s Ex. 53 at 1; Pet.'s Ex. 54 at 2.

In contradistinction to the many assessments and impressions described in

petitioner's medical records, Dr. Tenpenny, as petitioner's designated medical witness, has offered an opinion regarding the nature of petitioner's injury that stands alone in this case. She opined that petitioner suffers either from TM or CIDP. In reaching her opinion, she considered the similarity between petitioner's symptoms and the symptoms of the conditions TM and CIDP. Although the results of petitioner's diagnostic studies and tests were interpreted as normal, Dr. Tenpenny pointed to two test results, obtained respectively six months and nine months after the alleged first appearance of petitioner's symptoms, as evidence of "subtle signs" of his disease. See Pet.'s Ex. 45 at 7. Although the test results, specifically an elevated protein level in petitioner's cerebrospinal fluid and a mildly elevated SED rate, may be suggestive of either an infectious or an inflammatory process, neither test result is diagnostic of either TM or CIDP. On the contrary, the common diagnostic indicators–a spinal lesion for TM or evidence of demyelination for CIDP–are absent in petitioner's case. Not only were the common diagnostic indicators missing when petitioner's symptoms first developed, but the indicators have failed to become apparent over the nearly ten-year course of examinations that petitioner has received since the onset of his condition.

In the absence of objective test results to support her assertions about petitioner's condition, Dr. Tenpenny relied on various medical articles stating that patients affected by certain neurological conditions may produce certain normal test results. But, Dr. Leist persuasively challenged Dr. Tenpenny's position on the ground that the disease process of TM or CIDP–that is associated with expected disturbances in function–is unlikely to elude detection over a pronounced length of time during which the patient has undergone extensive testing and has had a number of good medical examinations. Moreover, Dr. Leist observed that other than an early and unsuccessful trial of prednisone, see Pet.'s Ex. 1 at 29, petitioner has not received the type of treatments that typically are prescribed in cases of TM or CIDP.

Petitioner's medical witness has asserted that petitioner has suffered an injury of either TM or CIDP. But, the weight of the record evidence does not support a finding that petitioner has either of the two conditions, and by her own admission, Dr. Tenpenny has no experience treating patients with either TM or CIDP. See Tr. at 35. The logic of the proposed sequence of cause and effect is diminished by the lack of evidence that petitioner has the injury that informs Dr. Tenpenny's theory of causation.

And further compromising petitioner's claim is the inapplicability of the biological mechanism of demyelination proposed by petitioner's medical witness, Dr. Tenpenny, where petitioner's injury is most consistently viewed in the record as a small fiber neuropathy. As respondent's expert pointed out, and petitioner's witness did not rebut, small nerve fibers lack the myelin sheaths that would be harmed by the proposed

demyelination process.

Although it is true that a number of physicians that examined petitioner after the onset of his symptoms noted a possible causal relationship between the vaccination and petitioner's neuropathic symptoms,[39] the examining doctors suggested that either an inflammatory or autoimmune-type process may have been triggered by the received vaccination. It is not clear from the records, however, whether the biological mechanism contemplated by these various treaters involved demyelination, the particular process suggested by Dr. Tenpenny.

Moreover, as Dr. Dresser, one of petitioner's treating neurologists observed in early 2002 (more than one year and a half after the onset of petitioner's symptoms), the early diagnostic impressions by examining neurologists that petitioner had suffered a sensory neuropathy as a response to his vaccinations were modified over time after extensive testing failed to provide objective findings of petitioner's alleged injury. See Pet.'s Ex. 18 at 45. Indeed, petitioner's own treaters found the nature of his injury to be "very complicated" and difficult to define. See id. at 47. But, at no point does the record reflect that petitioner's treating doctors entertained a diagnosis of either TM or CIDP, the conditions suggested by Dr. Tenpenny.

Without more from the treating doctors than the summarily expressed concerns about a possible causal association between petitioner's hepatitis B vaccination and his injury and without an explanation from petitioner's offered medical witness that provides a causal link between petitioner's hepatitis B vaccination and the injury for which the record supports a finding, the undersigned cannot credit the sequence of cause and effect proposed by Dr. Tenpenny as logical.[40] Accordingly, as presented, petitioner's claim must fail.

IV. Conclusion

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[39] These physicians included : (1) Dr. Roberts, petitioner's primary care doctor; (2) Dr. Villanueva, an infectious disease specialist; (3) Dr. Lomen-Hoerth, a neurologist; (4) Dr. Martin, a rheumatologist; (5) Dr. Chiu, a neurologist; and (6) Dr. Dresser, a neurologist.

[40] The undersigned observes here, and will address in further detail upon submission of petitioner's request for attorneys' fees and costs, that although presented as a treating doctor, Dr. Tenpenny effectively offered an expert opinion without the requisite qualifications to do so. On this ground, the reasonableness of the requested fees for Dr. Tenpenny, when submitted, will be closely examined.

For the foregoing reasons, the undersigned finds that petitioner has failed to establish a logical sequence of cause and effect in this case as now presented and thereby, has failed to satisfy his evidentiary burden showing that the received vaccination at issue brought about his injury. The extensive record before the undersigned does not support a finding that petitioner suffered the injury contemplated in the causation theory offered by petitioner's medical witness. Although a number of petitioner's treating doctors considered the possibility that petitioner experienced a vaccine-related neuropathic reaction, the medical witness that petitioner offered failed to address or to explore further those ponderings in her opinion. Rather, petitioner's medical witness, who by her own admission is neither specially trained nor experienced in neurology, advanced a theory of causation that her research supported even though the facts of petitioner's specific case did not. In the view of the undersigned, Dr. Tenpenny's own limitations as a medical witness adversely limited the presentation of petitioner's case. And, as presented in this circumstance, petitioner's theory is unavailing. The Clerk of the Court is directed to dismiss petitioner's claim and to enter judgment for respondent.[41]

IT IS SO ORDERED.

s/ Patricia E. Campbell-Smith
Patricia E. Campbell-Smith
Special Master

[41] Pursuant to Vaccine Rule 11(a), entry of judgment is expedited by the parties' joint filing of notice renouncing the right to seek review.