NOTE: This disposition is nonprecedential.

United States Court of Appeals for the Federal Circuit

PITEY MORGAN,
Petitioner-Appellant

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,
Respondent-Appellee

2020-2107

Appeal from the United States Court of Federal Claims in No. 1:15-vv-01137-RTH, Judge Ryan T. Holte.

Decided: March 24, 2021

SYLVIA CHIN-CAPLAN, Law Office of Sylvia Chin-Caplan, LLC, Boston, MA, for petitioner-appellant. Also represented by TIMOTHY MASON.

ZOE WADE, Torts Branch, Civil Division, United States Department of Justice, Washington, DC, for respondent-appellee. Also represented by JEFFREY B. CLARK, C. SALVATORE D'ALESSIO, HEATHER LYNN PEARLMAN, CATHARINE E. REEVES.

Before DYK, BRYSON, and HUGHES, *Circuit Judges*.

BRYSON, *Circuit Judge*.

Appellant Pitey Morgan filed a petition for compensation under the National Vaccine Injury Compensation Program, 42 U.S.C. §§ 300aa-10–300aa-34 ("Vaccine Act"), claiming that an influenza vaccination resulted in serious neurological injury. The chief special master in the Vaccine Program's Office of Special Masters denied his claim for compensation, and the Court of Federal Claims sustained that decision. We affirm.

I

A

Mr. Morgan has an extensive medical history from well before his influenza vaccination in 2012. *See Morgan v. Sec'y of Health & Hum. Servs.*, No. 15-1137V, 2019 WL 7498665, at *1–7 (Fed. Cl. Spec. Mstr. Dec. 4, 2019) ("*Special Master's Decision*"). The evidence showed that Mr. Morgan had numerous preexisting conditions, including persistent lower back pain, lower extremity radiculopathy, multi-level degenerative disc disease, lumbar spondylosis, and prostatitis. Nonetheless, the special master agreed with Mr. Morgan that his neurological injury did not predate his vaccination. *Id.* at *17.

Mr. Morgan received a flu vaccination on October 16, 2012. Following his vaccination, Mr. Morgan sought treatment from numerous physicians for medical issues. The reports from those physicians reveal that the assessments of Mr. Morgan's illness shifted over time as his symptoms matured and as the treating physicians received a more complete picture of his medical condition.

The day after his vaccination, Mr. Morgan saw a urologist, Dr. Arthur Golin, complaining of urination issues. *Id.* at *3. Mr. Morgan told Dr. Golin that his urination

issues had begun during the previous year but had accelerated during the past two and a half months. Mr. Morgan also reported weakness and numbness in his lower extremities. Dr. Golin assessed his condition as urinary retention with a possible neurologic component.

Six days later, Mr. Morgan saw Dr. Scott Greenwald, complaining of lower back pain. Mr. Morgan reported that the pain was radiating down both of his legs and that he was experiencing numbness in his calves. Dr. Greenwald treated Mr. Morgan with a lumbar steroid injection.

The next day, Mr. Morgan was taken to an emergency room after losing strength in, and the ability to ambulate, both of his legs. Dr. Christopher Hummel assessed possible cauda equina syndrome and epidural hematoma in light of Mr. Morgan's recent steroid injection. Dr. Hummel ordered an MRI of Mr. Morgan's spinal cord.

Later that day, Mr. Morgan was transferred to a neighboring hospital where he was examined by Dr. Christopher Marquart. A physical examination revealed that Mr. Morgan had poor sensory reception corresponding to the T12 and L1 vertebrae. *Id.* at *4. Upon reviewing Mr. Morgan's most recent MRI results, Dr. Marquart observed evidence of nerve root clumping at the conus (i.e., the base of the spinal cord), leading him to suspect that Mr. Morgan was experiencing transverse myelitis ("TM")[1] or some other acute, neuro-inflammatory process. Dr. Marquart admitted Mr. Morgan to the intensive care unit to ensure that "he [did] not have any type of ascending paralysis with the recent flu vaccination." *Special Master's Decision* at *4.

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[1] Myelitis is inflammation of the spinal cord. *Stedman's Medical Dictionary* 1268 (28th ed. 2006). Transverse myelitis is inflammation across the entire thickness of only one or two segments of the spinal cord. *Id.*

Dr. Roni Devlin, an infectious disease specialist, examined Mr. Morgan the following day and ordered another MRI examination. That MRI revealed swelling in the spinal cord from the T8 vertebra to the lower tip of the spinal cord. Dr. Devlin assessed myelitis of indeterminate etiology. He noted that "[c]ase reports of myelitis following vaccination have certainly been reported, but rarely." *Id.*

Two days later, Mr. Morgan was evaluated by Dr. Larry Wahl, who noted that Mr. Morgan's difficulties with his lower extremities seemed to reach a critical point the day after the October 23 steroid injection. Dr. Wahl reached a differential diagnosis of a viral infection, arachnoiditis, and TM, but he expressed skepticism about TM given the nature of the swelling in Mr. Morgan's spinal cord.

Mr. Morgan was discharged from the hospital on October 29, 2012. By that time, he had recovered the ability to stand, bear weight, and walk short distances with a walker. However, he continued to experience numbness and tingling in his lower extremities. During a follow-up examination on November 15, 2012, Dr. Marquart reiterated his belief that Mr. Morgan had probably experienced myelitis as a "reaction to his flu vaccine for lack of a better explanation." *Id.* A third MRI showed improvement in the appearance of the spinal cord.

On December 13, 2012, Mr. Morgan was examined by Dr. Douglas Gelb at the University of Michigan Neurology Clinic. Dr. Gelb proposed that Mr. Morgan had suffered from either an isolated episode of TM or the first instance of a recurring, central nervous system disease, such as multiple sclerosis or neuromyelitis optica ("NMO").[2]

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[2] Neuromyelitis optica is characterized by the demyelination of the optic nerve and the spinal cord. *Dorland's Medical Dictionary* 1249 (33rd ed. 2020). Demyelination

Special Master's Decision at *5. Dr. Gelb ordered an NMO antibodies test, which returned a negative result but with the qualification that a negative result did not necessarily preclude a diagnosis of NMO.

Mr. Morgan's condition improved from late 2012 into early 2013. He recovered strength in his lower extremities and did not experience any new symptoms.

In early 2013, however, Mr. Morgan's condition once again began to deteriorate. An examination by Dr. Wahl revealed decreased strength in both of Mr. Morgan's legs. Dr. Wahl ordered an MRI, and the results showed expanding lesions in the thoracic section of the spinal cord, the appearance of which was suggestive of TM. By June 2013, Mr. Morgan had lost further strength in his lower extremities and could no longer stand on his own. On July 23, 2013, Dr. Ivan Landon classified Mr. Morgan as paraplegic and concluded that Mr. Morgan had "suffered at least one, maybe two, relapses" and "was likely suffering from a polyphasic TM." *Id.*

From July 2013 through June 2014, Mr. Morgan saw occasional improvements in his condition while completing a treatment and rehabilitation program. *Id.* at *6. Despite those occasional improvements, Dr. Landon observed that Mr. Morgan's condition was generally deteriorating as he continued to experience relapses. By June 2014, Mr. Morgan was restricted to a wheelchair and began complaining of issues with his upper extremities.

On August 15, 2014, Mr. Morgan saw Dr. Gelb again. An examination revealed that Mr. Morgan's lower

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occurs when myelin, the protective coating on nerve cells, is damaged. *Id.* at 480. Symptoms of neuromyelitis optica often include changes in vision, flaccid paralysis of the extremities, and sensory and genitourinary disturbances. *Id.* at 1249.

extremities were completely immobile. Dr. Gelb was unsure whether Mr. Morgan's clinical deterioration "was due to [a] new episode of spinal cord inflammation, or simply some systemic illness exacerbating his deficits from his initial episode." *Id.* Dr. Gelb judged that the former was more likely because of the severity of Mr. Morgan's new symptoms. Dr. Gelb ordered MRIs of Mr. Morgan's spine and brain, as well as another NMO antibodies test. The antibodies test was negative. The spinal MRI revealed a loss of volume starting at the T8 vertebra and extending down to the conus, likely caused by prior inflammation. The spinal MRI also showed signal changes and abnormal enhancements at the T2 vertebra that were indicative of spinal inflammation. The brain MRI revealed "nonspecific small areas of nonenhancing T2 signal prolongation in predominantly left supratentorial white matter," possibly as a result of prior inflammation at the T2 vertebra. *Id.*

On November 26, 2014, Dr. Robert Pace of the University of Michigan Multiple Sclerosis Clinic further evaluated Mr. Morgan's MRIs. Regarding the brain MRI, Dr. Pace noted that the scans did not reveal patterns that were suggestive of multiple sclerosis. "However, there is T2 hyperintensity in the fourth ventricle surrounding the cerebral aqueduct. This is of unclear significance, but can be seen in [NMO] spectrum . . . ." *Id.* Based on his review of the laboratory tests and imaging studies, Dr. Pace diagnosed Mr. Morgan with "longitudinal myelitis due to [NMO], sero-negative." *Id.* at *7.

In an August 15, 2015, visit to Dr. Pace, Mr. Morgan reported persistent paralysis in his lower extremities and numbness starting at, and extending below, his middle back. Dr. Pace ordered additional MRIs. The brain MRI revealed that the "signal hyperintensities located [near the ventricles] of the brain [had been] stable since January." *Id.* The spinal MRI revealed no abnormalities. Dr. Pace reported a differential diagnosis of relapsing-remitting

multiple sclerosis, NMO, and flaccid paralysis of the lower extremities.

On April 20, 2016, Mr. Morgan visited Dr. Pace for a third time. Mr. Morgan was still confined to a wheelchair but had not developed any new or worsening symptoms. Following an examination, Dr. Pace once again diagnosed Mr. Morgan's condition as "most likely seronegative [NMO]." *Id.*

In April 2017, Dr. Pace examined Mr. Morgan for a fourth and final time. The examination revealed positive progress with Mr. Morgan's lower extremities. An MRI showed no evidence of new or enhanced lesions on the spinal cord. Dr. Pace's differential diagnosis listed NMO, acute TM, paralytic syndrome, and spinal stenosis of the cervical region.

B

Mr. Morgan filed a petition seeking compensation under the Vaccine Act. He alleged that he had developed longitudinally extensive transverse myelitis ("LETM")[3] caused by the flu vaccine he received in 2012. At a January 2019 hearing, the special master heard testimony from both sides' expert witnesses.

Mr. Morgan's expert, Dr. Carlo Tornatore, testified that Mr. Morgan had developed remitting and relapsing LETM, not NMO or NMO spectrum disorder ("NMOSD").[4]

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[3] Longitudinally extensive transverse myelitis is characterized by bilateral spinal cord inflammation (i.e., transverse myelitis) that extends vertically through three or more vertebral segments. D. Karussis et al., *The Spectrum of Post-Vaccination Inflammatory CNS Demyelinating Syndromes*, Autoimmunity Reviews 1, 6 (2013).

[4] The term neuromyelitis optica spectrum disorder was first introduced in 2007 to cover patient groups with

Special Master's Decision at *8. Dr. Tornatore described TM/LETM as a syndrome in which an immune-mediated process causes inflammation of the spinal cord, resulting in scarring and neural injury. That inflammatory process, according to Dr. Tornatore, is known to cause symptoms that align with Mr. Morgan's symptoms, including weakness in limbs, sensory alterations, and autonomic dysfunction.

Dr. Tornatore stated that Mr. Morgan did not meet the diagnostic criteria for NMOSD. Because Mr. Morgan tested negative for NMO-related antibodies, Dr. Tornatore testified, he needed to exhibit an additional clinical characteristic besides acute myelitis to justify a diagnosis of NMOSD, but his medical records did not reveal such a characteristic. *Id.* at *9. Dr. Tornatore added that in his view Mr. Morgan did not fit the typical demographic for NMOSD and that, although the remitting-relapsing nature of Mr. Morgan's disease did not fit the typical TM case, TM could be polyphasic in certain individuals.

As for causation, Dr. Tornatore testified that the close connection in time between Mr. Morgan's vaccination and the onset of his symptoms meant that Mr. Morgan's vaccination was more likely than not the cause of his LETM. *Id.* at *7. Dr. Tornatore proposed molecular mimicry as the medical theory for causation. *Id.* at *9. In support of that

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various clinical features and AQP4-IgG antibody test results. *See* Wingerchuk et al., *International Consensus Diagnostic Criteria for Neuromyelitis Optica Spectrum Disorders*, 85 Neurology 177, 178 (2015). In 2013, a panel of experts decided that the term "NMO would be subsumed into the single descriptive term NMOSD because the clinical behavior, immunopathogenesis, and treatment of patients who have NMOSD are not demonstrably different than for those with NMO and patients with incomplete forms of NMO frequently later fulfill NMO criteria." *Id.*

theory, Dr. Tornatore cited medical literature discussing molecular mimicry and its role in the development of TM following vaccinations. *See, e.g.*, N. Nakamura et al., *Neurologic Complications Associated with Influenza Vaccination: Two Adult Cases*, 42 Internal Med. 191, 193–94 (2003). Dr. Tornatore also relied on statements from Mr. Morgan's treating physicians indicating a possible connection between Mr. Morgan's vaccination and the onset of his disease. Dr. Tornatore did not, however, point to any evidence suggesting a connection between the influenza vaccine and NMOSD.

The government's expert, Dr. Subramaniam Sriram, disagreed with Dr. Tornatore and testified that in his opinion Mr. Morgan's condition was NMOSD featuring relapsing LETM, not merely LETM.[5] *Special Master's Decision* at *10. Regarding the NMOSD diagnostic criteria, Dr. Sriram agreed with Dr. Tornatore that Mr. Morgan was seronegative and thus needed to exhibit a second clinical characteristic in addition to acute myelitis in order to justify a diagnosis of NMOSD under the NMOSD diagnostic criteria. However, Dr. Sriram asserted that the additional characteristic was present. He pointed to Dr. Pace's analysis of the two brain MRIs, which supported the existence of an area postrema brain lesion. He also pointed to the MRI ordered by Dr. Wahl, which showed expansion of myelitis along the spinal cord, thus evincing dissemination in space. *Id.* at *11. Dr. Sriram also noted that the NMOSD diagnostic criteria were "guidelines" for treating

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[5] The government asserted, and the special master agreed, that LETM can be an acute condition or a feature of a chronic disorder, such as NMOSD or multiple sclerosis, both of which result from hyperactivity of the immune system directed to the central nervous system. *See Special Master's Decision* at *16. Dr. Tornatore's testimony is consistent with that conclusion. *See* J.A. 107–08.

physicians and did not have to be applied rigidly in order to reach a correct diagnosis.

Dr. Sriram testified that TM is generally considered a monophasic disease, not a polyphasic disease, especially when connected to an infection or vaccination event, as alleged in this case. On the other hand, NMOSD is typically considered a chronic condition, with 60 to 70 percent of patients suffering relapses, according to Dr. Sriram. In sum, Dr. Sriram concluded from the totality of Mr. Morgan's medical history, including the remitting and relapsing nature of his disease, that the proper diagnosis was NMOSD with clinically relapsing LETM.

The special master issued a decision denying Mr. Morgan's claim for compensation. *Special Master's Decision* at *20. As a preliminary matter, the special master agreed with Mr. Morgan that his neurologic condition did not predate his flu vaccination. *Id.* at *17. The special master found, however, that the record best supported a diagnosis of NMOSD, not LETM. *Id.* at *18. The special master then reviewed Mr. Morgan's showing on causation regarding the flu vaccine and NMOSD. *Id.* at *19. The special master concluded that Mr. Morgan failed to carry his burden with respect to the first two prongs of the causation test set out in *Althen v. Secretary of Health & Hum. Servs.*, 418 F.3d 1274 (Fed. Cir. 2005).

Mr. Morgan moved for review of the special master's decision, but the Court of Federal Claims affirmed. *Morgan v. Sec'y of Health & Hum. Servs.*, 148 Fed. Cl. 454, 477 (2020).

Mr. Morgan appeals to this court. We have jurisdiction pursuant to 42 U.S.C. § 300aa-12(f).

II

On appeal, Mr. Morgan challenges the special master's conclusion that the evidence best supports a diagnosis of

NMOSD, and he argues that the special master's findings on causation were tainted by that erroneous conclusion.

A petitioner seeking compensation under the Vaccine Act for an injury not listed in the Vaccine Injury Table (*see* 42 U.S.C. § 300aa-14) must prove by a preponderance of the evidence that the vaccine was the cause in fact of the injury. *Broekelschen v. Sec'y of Health & Hum. Servs.*, 618 F.3d 1339, 1341 (Fed. Cir. 2010); *see* 42 U.S.C. §§ 300aa-13(a)(1)(A) and 300aa-11(c)(1)(C)(ii). To prove causation in fact, the petitioner must establish by a preponderance of the evidence "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *De Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008) (quoting the "*Althen* test" from 418 F.3d at 1278). In cases in which the nature of the claimant's injury is in dispute, a fundamental first step in the causation analysis is to determine the nature of the injury based on a preponderance of the evidence. *See Lombardi v. Sec'y of Health & Hum. Servs.*, 656 F.3d 1343, 1352 (Fed. Cir. 2011).

In Vaccine Act cases, we review a decision by the Court of Federal Claims de novo, applying the same standard of review as that court applies in reviewing a decision of a special master. *Broekelschen*, 618 F.3d at 1345. Although we review legal determinations without deference, we review the special master's factual findings under the arbitrary and capricious standard. *Milik v. Sec'y of Health & Hum. Servs.*, 822 F.3d 1367, 1375 (Fed. Cir. 2016). "The arbitrary and capricious standard is difficult for an appellant to satisfy with respect to any issue, but particularly with respect to an issue that turns on the weighing of evidence by the trier of fact." *Id.* (internal quotation marks omitted).

A

Mr. Morgan argues that the special master's diagnosis of NMOSD was arbitrary and capricious because it relied on factual findings that had no basis in the evidence of record. More specifically, Mr. Morgan argues that the special master erred in finding that Mr. Morgan's condition satisfied the diagnostic criteria for NMOSD, seronegative type.

The NMOSD diagnostic criteria for seronegative adult patients are as follows: First, the patient must exhibit two or more core clinical characteristics along with the corresponding MRI components. Second, at least one of the core clinical characteristics must be optic neuritis, TM, or area postrema clinical syndrome. Third, the two required core clinical characteristics must occur across different neuroanatomic regions, i.e., they must exhibit "dissemination in space." Wingerchuk et al., *International Consensus Diagnostic Criteria for Neuromyelitis Optica Spectrum Disorders*, 85 Neurology 177, 179 (2015); *see also* Weinshenker et al., *Neuromyelitis Spectrum Disorders*, 92 Mayo Clinic Proc. 663, 666 (2017). It is undisputed that Mr. Morgan's injury satisfied the clinical characteristic of TM and its MRI component. The characteristic in dispute is the area postrema syndrome and its corresponding MRI component requiring a lesion in a specific area of the brain. *See* Wingerchuk et al., *supra*, at 179.

The special master found that Mr. Morgan's medical history "demonstrated brain lesions in the area postrema region of the brain" and that the "mere existence of an area postrema lesion supported a diagnosis of NMOSD by itself." *Special Master's Decision* at *18. That finding was clearly erroneous, Mr. Morgan contends, because the NMOSD diagnostic criteria require area postrema *syndrome*, and Dr. Sriram admitted that Mr. Morgan did not have any of the clinical symptoms evincing that syndrome, namely hiccups, nausea, or vomiting. According to Mr. Morgan, the special master's finding was also clearly

erroneous because the official interpretation of the brain MRIs in Mr. Morgan's medical records did not indicate a lesion in the area postrema. Finally, beyond the dispute over the area postrema characteristic, Mr. Morgan contends that the special master erred in concluding that the April 2013 MRI showing the expansion of myelitis along the spinal cord satisfied the requirement of dissemination in space. That was error, Mr. Morgan argues, because dissemination in space requires two or more clinical characteristics that affect different neuroanatomic regions, not merely different segments of the spinal cord.

Even assuming Mr. Morgan is correct in arguing that his injury did not satisfy the technical requirements of the NMOSD diagnostic criteria, the special master's conclusion that Mr. Morgan was suffering from NMOSD is still supported by ample evidence, for several reasons.

First, the remitting and relapsing nature of Mr. Morgan's condition strongly supports a diagnosis of NMOSD as opposed to LETM. Dr. Sriram explained that TM can be a stand-alone condition or a feature of NMOSD. He testified that TM, manifesting on its own, is typically a monophasic event, while "60 to 70 percent" of patients with NMOSD will relapse. J.A. 156. He also explained that physicians will reconsider an initial diagnosis of TM if a relapse occurs.

While Dr. Tornatore testified that in his opinion an individual with TM can experience relapses and that Mr. Morgan had LETM, *see* J.A. 116 and 125, the special master was entitled to credit Dr. Sriram's testimony over Dr. Tornatore's conflicting testimony and to conclude that the overall course of Mr. Morgan's symptoms fit best with a diagnosis of NMOSD. *See Special Master's Decision* at *18–19. The special master's decision in that regard is not an outlier. Other special masters have reached the same conclusion on similar facts. *See, e.g.*, *Doles v. Sec'y of Health & Hum. Servs.*, No. 17-642V, 2021 WL 750416, at *16 (Fed.

Cl. Spec. Mstr. Feb. 1, 2021) (noting a "distinction between acute demyelinating injuries such as transverse myelitis and chronic, relapsing demyelinating injuries such as multiple sclerosis"); *Wei-Ti Chen v. Sec'y of Health & Hum. Servs.*, No. 16-634V, 2019 WL 2121208, at *19 (Fed. Cl. Spec. Mstr. Apr. 19, 2019) (collecting cases); *see also Crosby v. Sec'y of Health & Hum. Servs.*, No. 08-799V, 2012 WL 13036266, at *5 n.7 (Fed. Cl. Spec. Mstr. June 20, 2012) (citing various medical journals that describe TM as "acute" and "generally monophasic").

Second, there is significant evidence in Mr. Morgan's medical history other than acute myelitis that is suggestive of NMOSD. In particular, Dr. Sriram pointed to the expansion of myelitis vertically along the spinal cord and a brain abnormality in an area most commonly associated with NMOSD. The MRI ordered by Dr. Wahl showed that myelitis had expanded along Mr. Morgan's spinal cord over a period of roughly seven months. That expansion was very relevant to an NMOSD diagnosis, Dr. Sriram testified, regardless of whether it fit within the technical conditions of the NMOSD diagnostic criteria. In addition, Dr. Pace's analysis of Mr. Morgan's brain MRI noted signal hyperintensities located near the ventricles of the brain. Dr. Pace described those hypersensitivities as having "unclear significance," but noted that they "can be seen in [NMO] spectrum." J.A. 269. Regarding Dr. Pace's notes, Dr. Sriram testified that there are very few diseases that produce an abnormality near the fourth ventricle of the brain, that NMOSD is one of those diseases, and that such an abnormality would be something treating physicians would "pay attention to" with respect to an NMOSD diagnosis. J.A. 176–77.

Mr. Morgan argues that his medical history does not reflect the presence of all the symptoms normally associated with NMOSD, particularly those symptoms associated with area postrema syndrome. As Dr. Sriram testified and the special master found, however, the symptoms

reported by the treating physicians were strongly suggestive of NMOSD rather than LETM. Mr. Morgan thoroughly explored the ways in which his symptoms departed from the classic symptoms of NMOSD, both on cross-examination of Dr. Sriram and in his briefing to the special master. Notwithstanding the absence of some symptoms generally associated with NMOSD, the special master concluded that the evidence summarized by both experts supported Dr. Sriram's proposed diagnosis of NMOSD better than Dr. Tornatore's proposed diagnosis of LETM. We are not inclined to second-guess that weighing of the evidence.

Third, the diagnoses and assessments of Mr. Morgan's condition by his treating physicians, on balance, favor a diagnosis of NMOSD. There were 13 such diagnoses and assessments between the day after Mr. Morgan's vaccination and the spring of 2017. Six of those diagnoses and assessments suggested stand-alone TM/LETM, while five suggested NMOSD. Importantly, however, four of the five suggesting NMOSD were provided after all of Mr. Morgan's relapses, which occurred in the spring of 2013 and thereafter. On the other hand, only two of the six suggesting TM were provided after the first relapse in the spring of 2013.

Based on that record, the special master concluded that "[t]reaters initially, and rationally, interpreted [Mr. Morgan's] symptoms and test results (like MRIs) as supportive of LETM. . . . But over time, [Mr. Morgan] began experiencing a progressive course of symptoms that suggested a relapse, and certainly resulted in more severe symptoms that impacted his ambulation. . . . Thereafter, other evidence (as extensively referenced above) undermined the initial conclusion about the possible nature of [Mr. Morgan's] injury," and the more likely diagnosis became NMOSD. *Special Master's Decision* at *18. The special master's conclusion was not unreasonable.

Finally, contrary to Mr. Morgan's suggestion, the evidence did not show that the NMOSD diagnostic criteria are

the definitive metric for diagnosing NMOSD, to the exclusion of all other evidence in the record. The primary purpose of the NMOSD diagnostic criteria was to differentiate NMOSD from multiple sclerosis, because treatments for either of those two diseases are known to have detrimental effects on patients suffering from the other disease. *See* Weinshenker et al., *supra*, at 666. The purpose was not to distinguish NMOSD from relapsing LETM. *See id.*[6]

In contrast to the purpose for which the NMOSD diagnostic criteria were created, the special master's focus was not to distinguish between NMOSD and multiple sclerosis, but to determine whether Mr. Morgan suffered from the alleged injury, LETM. For that purpose, the NMOSD diagnostic criteria served as a relevant data point but were not dispositive. The diagnostic criteria themselves recognize that in the case of a seronegative patient experiencing recurring myelitis, "NMOSD cannot be excluded." Weinshenker et al., *supra*, at 666–67. Thus, even if Mr. Morgan's injury did not fully satisfy the NMOSD diagnostic criteria, the special master's diagnosis of NMOSD is well supported by the evidence of record, including the remitting-relapsing nature of Mr. Morgan's condition, the MRIs showing features unique to NMOSD, the sum of the treating physicians' diagnoses, and expert testimony from Dr. Sriram. For those reasons, we hold that, based on the record as a

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[6] Dr. Sriram testified that although Mr. Morgan's brain abnormality persuaded him that NMOSD was the correct diagnosis, the existence of that brain abnormality was not pertinent to the proper treatment—"[Mr. Morgan was] going to be treated similarly" with or without the brain lesion. J.A. 177.

whole, the special master's diagnosis was not arbitrary or capricious. *See* 42 U.S.C. § 300a-13(a)(1).[7]

B

Mr. Morgan argues that the special master's findings on causation were erroneous because they relied upon the special master's improper conclusion that Mr. Morgan suffered from NMOSD. Because we reject Mr. Morgan's argument that the special master's finding regarding NMOSD was arbitrary and capricious, we review the special master's causation findings with respect to whether Mr. Morgan's vaccination caused his NMOSD.

In his causation analysis, the special master first noted that Mr. Morgan's theory of molecular mimicry was an accepted scientific theory for explaining how a vaccine could cause TM. *Special Master's Decision* at *19. Despite the acceptability of molecular mimicry, the special master found an absence of evidence in the record and a lack of authority in prior Vaccine Act decisions supporting molecular mimicry as a viable mechanism for causing NMOSD and, more generally, supporting the hypothesis that a flu vaccine could cause NMOSD. *Id.* at *19–20. For those reasons, the special master concluded that Mr. Morgan had not satisfied the first two prongs of the *Althen* test.

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[7] Mr. Morgan asserts that the government abandoned its theory that the NMOSD diagnostic criteria were satisfied, and that the special master erred by "credit[ing] an argument that [the government] clearly elected to waive." Appellant's Opening Br. 21. We reject that argument. The special master was entitled to weigh the totality of the evidence, regardless of the position taken by the government as to particular pieces of evidence. The question before us is whether the evidence as a whole was sufficient to support the special master's conclusion, and we hold that it was.

We discern no error in the special master's causation analysis. To carry his burden on causation, Mr. Morgan needed to provide a reputable medical or scientific explanation pertaining to his alleged vaccine injury, although that explanation needed only to be "legally probable, not medically or scientifically certain." *Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1322 (Fed. Cir. 2010) (citation omitted). Mr. Morgan failed to show how his flu vaccination could have caused his NMOSD through the mechanism of molecular mimicry. The most Mr. Morgan offered on that question was a reference listing various infectious agents that could play a role in triggering NMOSD. *See* S. Kim et al., *Differential Diagnosis of Neuromyelitis Optica Spectrum Disorders*, 10 Therapeutic Advances in Neurological Disorders 265, 279 (2017). That list of infectious agents does not, however, include the influenza virus. Additionally, while Dr. Tornatore testified regarding the possible relationship between molecular mimicry and NMOSD, his testimony did not implicate the flu vaccine. *See* J.A. 104–07.

Given the lack of evidence supporting a connection between the flu vaccine and NMOSD, it is clear that Mr. Morgan has not established, by a preponderance of the evidence, that his flu vaccination was causally connected to his NMOSD through the medical theory of molecular mimicry. Nor has he established by a preponderance of the evidence that there was a logical sequence of cause and effect showing that his vaccination was the reason for his remitting-relapsing NMOSD.

In sum, as in *Broekelschen*, 618 F.3d 1339 (Fed. Cir. 2010), the causation inquiry in this case largely turns on which injury the claimant has suffered. *See id.* at 1346. Because Mr. Morgan was found to have suffered from NMOSD, and because Mr. Morgan has not provided sufficient evidence to support a legally probable connection between his vaccination and his NMOSD, we hold that the

special master's findings on causation were not arbitrary or capricious.

We therefore uphold the judgment of the Court of Federal Claims.

AFFIRMED