# In the United States Court of Federal Claims

**OFFICE OF SPECIAL MASTERS**

Filed: January 7, 2026

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| RONDI JOHNSON, administrator of the ESTATE OF JAMES CHRISTIAN JOHNSON, | * | |
| Petitioner, | * | No. 18-1749V |
| v. | * | Special Master Young |
| SECRETARY OF HEALTH AND HUMAN SERVICES, | * | |
| Respondent. | * | |
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*Leigh Finfer*, Muller Brazil, LLP, Dresher, PA, for Petitioner.
*Alyssa M. Petroff*, U.S. Department of Justice, Washington, DC, for Respondent.

## FACT RULING[1]

On November 9, 2018, Rondi Johnson ("Petitioner") filed a petition pursuant to the National Vaccine Injury Compensation Program ("the Program")[2] as the administrator of the estate of her brother, James Christian Johnson. Pet., ECF No. 1. Petitioner alleged that Mr. Johnson suffered from acute motor and sensory neuropathy ("AMSAN"),[3] a rare form of Guillain-Barré Syndrome ("GBS"),[4] as a result of an influenza ("flu") vaccine Mr. Johnson received on November

[1] Because this Ruling contains a reasoned explanation for the action taken in this case, it must be made publicly accessible and will be posted on the United States Court of Federal Claims' website, and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the Ruling will be available to anyone with access to the internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, I agree that the identified material fits within this definition, I will redact such material from public access.

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

[3] Motor neuropathy is a "neuropathy or polyneuropathy involving only motor nerves." *Motor Neuropathy*, DORLAND'S MED. DICTIONARY ONLINE, https://www.dorlandsonline.com/dorland/definition?id=92695 (hereinafter, "DORLAND'S"); Sensory neuropathy is a "neuropathy or polyneuropathy of sensory nerves." *Sensory Neuropathy*, DORLAND'S.

[4] GBS is a "rapidly progressive ascending motor neuron paralysis of unknown etiology, frequently seen after enteric or respiratory infection. . . . It begins with paresthesias of the feet, followed by flaccid paralysis

12, 2015.[5] Am. Pet. at 1, ECF No. 35. Respondent contested Petitioner's diagnosis, arguing that Mr. Johnson's treating physicians did not agree on a diagnosis and that Mr. Johnson displayed several signs of a central nervous system ("CNS") disease and not AMSAN or GBS. Resp't's Rep. at 11, ECF No. 20.

A careful analysis and weighing of all the evidence presented in this case in accordance with the applicable legal standards[6] reveals that Petitioner has failed to provide preponderant evidence that Mr. Johnson suffered from AMSAN or another form of GBS.

## I. Procedural History

Petitioner filed her petition and medical records on November 9, 2018. Pet., Pet'r's Exs 1–2, 4, 7–9, ECF No. 1. Petitioner filed additional medical records between November 21, 2018, and August 5, 2019. Pet'r's Exs. 3, 5–6, 10, ECF No. 7; Pet'r's Ex. 11, ECF No. 8; Pet'r's Ex. 12, ECF No. 17. Respondent filed his Rule 4(c) Report, opposing compensation, on March 3, 2020. Resp't's Rep. Specifically, Respondent argued that Mr. Johnson's diagnosis was "unclear in this case, as his treating physicians postulated his diagnoses to be various demyelinating diseases of the central nervous system." *Id.* at 11.

Petitioner filed an expert report from Lawrence Steinman, M.D., along with his CV and supporting medical literature on April 27, 2022. Pet'r's Exs. 13–14, 16–20, 22–34, ECF No. 37. Petitioner filed additional medical literature on May 7, 2022. Pet'r's Exs. 15, 21, ECF No. 38. Respondent filed responsive expert reports from You-Wen He, M.D., Ph.D, and Michael Wilson, M.D., the experts' CVs, and supporting medical literature on October 11, 2022. Resp't's Ex. A, Tabs 1–6, Resp't's Exs. B–D, ECF No. 42. Petitioner filed a supplemental report from Dr. Steinman and supporting literature on May 22, 2023. Pet'r's Exs. 35–37, ECF No. 45. Respondent filed a supplemental report from Dr. He and supporting literature on August 14, 2023. Resp't's Ex. E, Tabs 1–6, ECF No. 47.

On February 8, 2024, I scheduled a hearing for October 21–22, 2025. Order, ECF No. 49. Petitioner filed a pre-hearing brief and supporting literature on June 20, 2025. Pet'r's Exs. 38–42, ECF No. 52; Pet'r's Br., ECF No. 53. Respondent filed his pre-hearing brief on August 20, 2025. Resp't's Br., ECF No. 54. On September 4, 2025, I held a status conference with the parties where they agreed to a factual ruling regarding Petitioner's diagnosis due to the sudden unavailability of Respondent's expert, Dr. Wilson, for the upcoming hearing. *See* Order, ECF No. 55. Petitioner

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of the entire lower limbs, ascending to the trunk, upper limbs, and face; other characteristics include slight fever, bulbar palsy, absent or lessened tendon reflexes, and increased protein in the cerebrospinal fluid without a corresponding increase in cells." *Gullain-Barré Syndrome*, DORLAND'S.

[5] Petitioner originally alleged Mr. Johnson suffered from axonal sensory motor neuropathy in her first petition, and then clarified Mr. Johnson's alleged injury in her amended petition. *See* Pet. at 1; *see also* Am. Pet. at 1.

[6] While I have reviewed all of the information filed in this case, only those filings and records that are most relevant to the decision will be discussed. *Moriarty v. Sec'y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision.") (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.*, 527 F. App'x 875, 884 (Fed. Cir. 2013) ("Finding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered.").

filed her motion for a fact ruling on October 6, 2025, and Respondent filed his response and additional medical literature on November 6, 2025. Pet'r's Mot., ECF No. 56; Resp't's Exs. C, Tabs 1–9, ECF No. 57; Resp't's Resp., ECF No. 58. Petitioner did not file a reply.

## II. Factual History

### A. Pre-Vaccination Medical History

Mr. Johnson was 59 years old at the time of his flu vaccination on November 12, 2015. Pet'r's Ex. 1 at 1. His pre-vaccination medical history was significant for hay fever/sinusitis, prior mononucleosis and measles infections, lactose and gluten intolerance, and an allergy to penicillin. Pet'r's Ex. 4 at 55. Mr. Johnson was treated for a sleep disorder from 2014 to 2015. *Id.* at 4. He also received treatment for suspected Lyme disease five to six years prior to vaccination. *Id.*

### B. Vaccination and Post-Vaccination Medical History

Mr. Johnson received the flu vaccine on November 12, 2015. Pet'r's Ex. 1 at 1. His first post-vaccine medical appointment was on November 30, 2015, at the emergency department ("ED") at Oakwood Heritage Hospital. Pet'r's Ex. 2 at 15. Mr. Johnson presented to Dr. Gaiti Bakhsh with a two-day history of difficulty urinating, difficulty moving his eyes after waking up, dizziness, and blurriness in his right eye. *Id.* at 15, 17. On examination, Dr. Bakhsh noted supra-pubic tenderness and abdominal pain, and Mr. Johnson's bloodwork revealed abnormalities in his glucose and mean platelet volume. *Id.* at 15, 17. He was offered an ophthalmology consultation and a computed tomography ("CT") scan but denied both. *Id.* at 17. Mr. Johnson had a foley catheter installed and was discharged the same day. *Id.*

On December 3, 2015, Mr. Johnson was admitted to St. Joseph Hospital Orange with complains of blurry vision for one week, urinary retention for three to four days, and worsening lower extremity weakness. Pet'r's Ex. 3-3 at 800. Dr. Colleen Rivers opined about the possibility of GBS, but noted "normal reflexes and urinary retention make this less likely." *Id.* She also considered a primary spinal cord lesion; "however, no back pain and blurry vision make this less likely." *Id.* Dr. Rivers ordered a head CT and scheduled a consult with neurology. *Id.*

Mr. Johnson was next examined by neurologist Mayank Pathak, who noted complaints of eye pain, loss of vision in his right eye, and waxing/waning weakness in both lower limbs. Pet'r's Ex. 3-3 at 810. Dr. Pathak recorded that Mr. Johnson received the flu shot "mid-November," and that his symptoms began on November 25, 2015. *Id.* Mr. Johnson further reported that following his November 30, 2015 ED visit, his leg weakness became so bad "he was unable to ambulate." *Id.* On examination, Dr. Pathak observed his pupils were moderately dilated and "extremely sluggish to light reaction," with the right side being worse than the left. *Id.* at 811. Dr. Pathak also noted mild weakness in Mr. Johnson's right arm and 0/5 strength and profound sensory loss in both lower limbs in addition to hypotonic muscle tone. *Id.* He exhibited +2 deep tendon reflexes at both biceps and triceps, 1 at both knees and ankles, and had silent plantar responses bilaterally. *Id.* A CT scan of Mr. Johnson's head revealed "a slightly complex-appearing hypodense lesion . . . over [two centimeters] in diameter" on the left side of his brain. *Id.* Dr. Pathak assessed Mr. Johnson as having "signs and symptoms referable both to brain/optic nerve and to the spinal cord,"

and opined that he may have "a significant myelopathy[7] of undetermined cause." *Id.* She further assessed Mr. Johnson to have a mild encephalopathy,[8] and opined a CNS disease such as "Devic's/neuromyelitis optica [("NMO")],[9] multiple sclerosis [("MS")],[10] or acute disseminated encephalomyelitis [("ADEM")][11] should be considered." *Id.* Dr. Pathak specifically noted that "[t]he syndrome is least consistent with peripheral polyneuropathy." *Id.*

Mr. Johnson underwent magnetic resonance imaging ("MRI") of his brain on December 4, 2015, which revealed a large demyelinating lesion on the left periventricular white matter of the frontal lobe with inflammatory changes and possible infarcation. Pet'r's Ex. 3-3 at 819. A spinal MRI taken the same day "showed extensive patchy and confluent areas of demyelination in the thoracic cord, extending to lumbar segments, and perhaps slightly in [the] cervical [spine]." *Id.* Dr. James Roh reviewed Mr. Johnson's imaging, and opined he had a CNS "demyelinating disease such as might be caused by [NMO], [MS], or [ADEM]." *Id.* at 820. By December 9, 2015, Mr. Johnson had completed five days of high-dose intravenous ("IV") Solumedrol, and by December 11, 2015, he had completed a five-day course of plasmapheresis. *Id.* Due to improvements in his vision and stabilization of his sensory levels on his torso, Mr. Johnson was discharged on December 11, 2015, to Fountain Care Skilled Nursing Facility ("FCSNF"), with diagnoses of "demyelinating disease of [the CNS]" and urinary retention. *Id.* at 792. He continued to have sensory defects in his torso and vision problems. *Id.*

Mr. Johnson remained at FCSNF from December 11, 2015, to December 21, 2015, where he underwent physical and occupational therapy. Pet'r's Ex. 11 at 37–67. On December 21, 2015, Mr. Johnson was transported to the St. Joseph Hospital ED due to decreased responsiveness, inability to communicate verbally, and lethargy. Pet'r's Ex. 3-3 at 770. A head CT revealed evidence of a previous infarction, but no acute injury. *Id.* at 772. He was diagnosed with dehydration and a urinary tract infection and discharged the same day. *Id.* at 777.

Mr. Johnson returned to the St. Joseph ED two days later on December 23, 2015, for worsening neurological symptoms. Pet'r's Ex. 3-3 at 463. His attending physician, Dr. Jason Caberto, noted that he was "visibly tired and week," and that his blood work showed an elevated C-reactive protein level of 53.1. *Id.* Petitioner reported that Mr. Johnson had begun experiencing these symptoms following his flu shot and that he was currently non-verbal. *Id.* On examination, Dr. Caberto noted that Mr. Johnson had "no sensation[,] no ability to move lower extremities,"

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[7] A myelopathy is "any of various functional disturbances or pathologic changes in the spinal cord, often referring to nonspecific lesions in contrast to the inflammatory lesions of myelitis." *Myelopathy*, *Dorland's*.
[8] Encephalopathy is "any degenerative disease of the brain." *Encephalopathy*, *Dorland's*.
[9] Neuromyelitis optica is the "combined, but not usually clinically simultaneous, demyelination of the optic nerve and the spinal cord; it is marked by diminution of vision and possibly blindness, flaccid paralysis of the extremities, and sensory and genitourinary disturbances." *Neuromyelitis Optica*, *Dorland's*.
[10] MS is "a disease in which there are foci of demyelination throughout the white matter of the central nervous system, sometimes extending into the gray matter; symptoms usually include weakness, incoordination, paresthesias, speech disturbances, and visual complaints. The course of the disease is usually prolonged, so that the term *multiple* also refers to remissions and relapses that occur over a period of many years." *Multiple Sclerosis*, *Dorland's* (emphasis in original).
[11] Acute disseminated encephalomyelitis is "an acute or subacute encephalomyelitis or myelitis characterized by perivascular lymphocyte and mononuclear cell infiltration and demyelination." *Acute Disseminated Encephalomyelitis*, *Dorland's*.

and he was unable to use his right arm. *Id.* at 469. Petitioner reported vision problems in Mr. Johnson's right eye and that he had not been eating. *Id.* Dr. Caberto assessed Mr. Johnson with "subacute paraplegia status [following] a flu shot" and admitted him the same day for neurological and gastrointestinal observation. *Id.* His differential diagnosis included MS, NMO, and ADEM. *Id.* at 474.

An MRI of Mr. Johnson's brain taken the same day revealed his brain lesion had increased in diameter, as well as "multiple areas of T2 signal change involving the right cerebral peduncle, bilateral pons/tectal nuclei region and [] the left cerebellar peduncle." Pet'r's Ex. 3-1 at 20. The MRI was reviewed by neurologist Peter Piampiano, who opined the changes were related to MS or another demyelinating condition. *Id.* Mr. Johnson also underwent an MRI of his cervical and thoracic spine, which revealed T2 signal abnormalities "concerning for acute inflammatory myelitis," as well as ADEM. *Id.* at 22–25. He was seen by neurologist Lars Anker later that day as a consultation for a potential brain biopsy. Pet'r's Ex. 3-3 at 488. Dr. Anker noted that Mr. Johnson's findings were consistent with a demyelinating condition, however, Mr. Johnson "ha[d] not responded to the usual treatment for such a condition," noting that his brain lesion had increased in size. *Id.* at 489. A biopsy was ordered to rule out a lymphoma. *Id.*

Mr. Johnson was also seen by neurologist Amir Shokrae the same day, who found Mr. Johnson to have severely reduced strength in all extremities and 1+ deep tendon reflexes throughout his body. Pet'r's Ex. 3-3 at 494. Dr. Shokrae's impression was a "[s]evere process of demyelinating disease of [the CNS] as well as thoracic spinal cord, which started following a flu shot in November 2015." *Id.* Dr. Shokrae restarted Mr. Johnson's Solumedrol and started him on a five-day course of intravenous immunoglobulin ("IVIG"). *Id.* The following day, on December 24, 2015, Mr. Johnson had a PEG tube installed to increase his caloric intake. Pet'r's Ex. 3-3 at 490.

Mr. Johnson underwent a brain biopsy on December 29, 2015, and a sample was also sent to the Mayo Clinic for consultation. Pet'r's Ex. 3-1 at 41. Mr. Johnson's treater at St. Joseph observed that "[a]lthough the features suggest a demyelinating lesion, they are not typical of primary demyelination. Although there is no evidence of lymphoma in these biopsies, a lymphoma which has regressed secondary to steroid treatment cannot be entirely excluded." *Id.* Dr. Joseph Parisi, who reviewed Mr. Johnson's biopsy at the Mayo Clinic, noted the lesion was not "typical of primary demyelination." *Id.* at 43. "The myelin loss appear[ed] to be incomplete and not demarcated from the surrounding parenchyma." *Id.* He noted the presence of axonal swelling "suggesting the presence of an axonal injury component, not typical of demyelination." *Id.* He also observed the lack of evidence of lymphoma, but echoed Mr. Johnson's treaters at St. Joseph by noting that a vanishing lymphoma could not be ruled out. *Id.* A repeat CT scan of Mr. Johnson's brain conducted on December 31, 2015, revealed the hypodense area in the left parietal lobe had increased from one-half centimeter to one centimeter in size. *Id.* at 10.

On January 5, 2016, Mr. Johnson was seen by neurologist Neelantha De Silva for a neurology follow-up. Pet'r's Ex. 3-1 at 222. Dr. De Silva opined that Mr. Johnson was suffering from a presumably "aggressive form of MS given biopsy results." *Id.* He recommended Mr. Johnson begin Tysabri infusions as an aggressive form of treatment. *Id.* Mr. Johnson was discharged on January 8, 2016, with a plan to start Tysabri infusions. Pet'r's Ex. 5-1 at 30.

Mr. Johnson returned the following day on January 9, 2016, with severe sepsis, and was treated with antibiotics until he was discharged on January 13, 2016. Pet'r's Ex. 3-1 at 150; Pet'r's Ex. 11 at 1. He returned again for sepsis on January 14, 2016, and was treated with antibiotics and IVIG from January 15, 2016, to January 18, 2016. Pet'r's Ex. 3-1 at 47–69, 80. His Tysabri infusions were deferred until his active infection ceased. *Id.* at 69. Mr. Johnson was discharged on January 19, 2016, with diagnoses of severe sepsis, a demyelinating disease of the CNS, functional quadriplegia, and neurogenic bladder, with a plan to start Tysabri infusions. *Id.* at 47.

On January 20, 2016, Mr. Johnson was admitted to the ED at Orange County Global Medical Center after being unresponsive for 30 minutes. Pet'r's Ex. 6 at 31. It was noted that he had been experiencing "worsening mental status and general weakness" prior to his unresponsiveness. *Id.* at 34. A CT scan revealed a small intracranial hemorrhage, and he was admitted to the intensive care unit for further observation. *Id.* at 45.

A consult with neurologist Clyde Holstein on January 22, 2016, described Mr. Johnson's condition as follows:

> He [] had MRI scans of his head and spine and he ha[d] evidence of multiple white matter lesions in the spine as well as [one] or [two] large lesions in the brain . . . . There [was] a small amount of hemorrhagic area at the biopsy site. . . . The biopsy may have been interpreted by the Mayo Clinic as showing some atypical demyelination and they even raise[d] the possibility this might represent the disappearing lymphoma. The patient [] received multiple courses of IV corticosteroids; he said that [] helped him. He also [] had IVIG and plasmapheresis. However, generally he [was] failing and [was] now really totally quadriplegic, unable to swallow and c[ould] only whisper. . . . He [] had a repeat MRI scan of the head; it show[ed] the area of biopsy with a small amount of hemorrhage and edema around it. . . . There [were] other white matter lesions of the brain. Initial blood work here [was] unremarkable.

Pet'r's Ex. 6 at 59. Dr. Holstein noted Mr. Johnson's prognosis was "rather poor at this time" and started him on Solumedrol, aquaporin 4 antibody, and serum immunoelectrophoresis. *Id.* at 60. Mr. Johnson was discharged on February 15, 2016, into hospice care, with diagnoses of MS, neurogenic bladder, quadriplegia, and sepsis. *Id.* at 4. Hospital course noted a diagnosis of "progressive demyelinating MS at St. Joe's hospital via brain biopsy." *Id.*

Mr. Johnson was transported to the Arizona Mayo Clinic on February 24, 2016, to seek a second opinion. He was seen by Dr. Shari Brand, who noted that Mr. Johnson was paralyzed from the waist down and had a decreased ability to move his upper limbs. Pet'r's Ex. 7 at 4. Dr. Brand recorded that "no antibody was noted in his blood for [NMO]," and that he had not yet started any treatment for his MS diagnosis. *Id.* Mr. Johnson had undergone steroid and plasmapheresis therapies without any effect. *Id.* He was seen by neurologist Erika Driver-Dunckley on February 25, 2016, who admitted him the same day. *Id.* at 19. In her patient notes, Dr. Driver-Dunckley recorded that Mr. Johnson's symptoms first began following his November 2015 flu vaccine. *Id.* "At first he was told he had [GBS], later was told he had some aggressive form of MS or perhaps

NMO." *Id.* Dr. Driver-Dunckley also noted that she had none of Mr. Johnson's prior imaging and testing to review, and Mr. Johnson had no sensation from his chest downward. *Id.* She noted that Mr. Johnson had received IV steroids, plasmapheresis, and IVIG, which he reported did not help. *Id.* Dr. Driver-Dunckley ordered an electromyography ("EMG") study and SEPs testing, as well as an MRI of his brain and CT of his lower spine. *Id.*

Because the Mayo Clinic did not have access to Mr. Johnson's prior testing, EMG, MRI, and CT scans were completed on February 25, 2016. Pet'r's Ex. 7 at 38, 51, 63. His EMG revealed "evidence of diffuse denervating process that most likely localize[d] to anterior horn cells or their motor roots, with probable superimposed critical illness neuropathy." Pet'r's Ex. 7 at 38. His MRI and revealed multiple lesions "suggestive of demyelination, particularly near the root entry zone of the left trigeminal nerve." *Id.* at 51. Neurologist Erin Okazaki reviewed Mr. Johnson's CT scan and observed "a long segment of abnormal T2 hyperintensity" from the C2 to C8 vertebra and considered "[ADEM], [NMO], and infection/inflammatory myelitis" as differential diagnoses. *Id.* at 52, 63. She noted Mr. Johnson's imaging and EMG were "concerning for demyelinating disease in the CNS" with extensive spinal cord lesions, "as well as a peripheral process involving motor nerve/AHC." *Id.* at 64. She further opined that "his presentation may be a sequelae of (a previously acute), now subacute transverse myelitis [("TM")]and a[] superimposed sensory motor axonal neuropathy in the setting of vaccination." *Id.* On February 27, 2016, Mr. Johnson was seen for increased difficulty breathing and diaphoresis. *Id.* at 80. He was assessed with neuromuscular respiratory failure and bulbar weakness. *Id.*

Mr. Johnson was seen by Physician's Assistant ("PA") Andrew Walker on March 1, 2016, for progressive respiratory failure. Pet'r's Ex. 7 at 126. PA Walker noted that Mr. Johnson was originally diagnosed with GBS in California, but "this was revised to an aggressive form of [MS]." *Id.* He continued by noting Mr. Johnson's imaging at the Mayo Clinic showed "widespread extensive spinal cord abnormalities." *Id.* His assessment was sepsis possibly secondary to a urinary tract infection and "[a]cute hypoxic respiratory failure possibly secondary to CNS demyelination disease/[TM] with superimposed neuropathy." *Id.* at 127. At a neurology consultation on March 10, 2016, Mr. Johnson started Cytoxan "using the Partners MS protocol," and his last dose was administered on March 18, 2016. *Id.* at 242, 339.

On the night of March 20, 2016, the rapid response intervention team was called due to Mr. Johnson's difficulty breathing, which continued through March 22, 2016. Pet'r's Ex. 373, 413. Mr. Johnson passed away on March 25, 2016, from respiratory failure. *Id.* at 503. His principal diagnosis was "fulminant idiopathic inflammatory demyelinating disease." *Id.* His death certificate attributed his cause of death to respiratory failure due to "demyelination disease, not otherwise specified," and listed the flu vaccine as a cause. Pet'r's Ex. 2 at 3.

## III. Affidavit of Petitioner

On November 21, 2018, Petitioner filed a brief affidavit. Pet'r's Ex. 10. She stated that she was Mr. Johnson's sister, and that he had received the flu vaccine on November 12, 2015. *Id.* at ¶¶ 1–2. She further stated that Mr. Johnson had "suffered post-[flu] vaccination sensory motor axonal neuropathy." *Id.* at ¶ 3.

## IV. Experts[12]

### A. Expert Qualifications

#### 1. Petitioner's Expert, Dr. Lawrence Steinman, M.D.

Dr. Steinman is a board-certified neurologist in California who has practiced since 1973. Pet'r's Ex. 13 at 2. He received his M.D. from Harvard University and completed his surgical internship and pediatric and neurology residencies at Stanford University Hospital. Pet'r's Ex. 14 at 1. He also completed a National Institutes of Health Fellowship in Neuroimmunology. Pet'r's Ex. 13 at 2. He currently serves as a Professor of Neurology and Pediatrics at Stanford University Medical Center and previously served as Chair of the Immunology Program. *Id.* "Over the past 42 years as a Professor of Neurology at Stanford University [he has] cared for hundreds of adults and children with various forms of neuroinflammatory diseases" including ADEM, GBS, TM, NMO, and MS. *Id.* He has published several times in the field of molecular mimicry and neurology, and "[h]undreds of these articles deal with neuroinflammatory reactions in the central and peripheral nervous system." *Id.* In the last five years Dr. Steinman has consulted "on hundreds of patients with [TM], [NMO], inflammatory neuropathy and [MS]." Pet'r's Ex. 13 at 2. He has also led two successful Phase 3 trials for treatment of MS and has received various awards and committee assignments for his work. *Id.* at 2–5.

#### 2. Respondent's Expert, Dr. Michael Wilson, M.D.

Dr. Wilson is a board-certified neurologist with subspeciality training in neuro-infectious diseases and neuroimmunology. Resp't's Ex. C at 1. He received his M.D. from the University of California, San Francisco ("UCSF") School of Medicine. Resp't's Ex. D at 1. He completed his internship in internal medicine and residency in neurology at Massachusetts General Hospital, as well as his clinical fellowship in neuro-infectious diseases. *Id.* He currently serves as an Associate Professor of Neurology at UCSF and is the Director of the UCSF Center for Encephalitis and Meningitis, and is also a member of the Multiple Sclerosis and Neuroinflammation Center. Resp't's Ex. C at 1. In his clinical practice he diagnoses and treats "patients with a variety of neuroinflammatory disorders ranging from autoimmune diseases like [MS] and [NMO] to infectious causes of meningitis, myelitis, and encephalitis. *Id.* He has published several times in the field of neurology and neuroinflammatory diseases. *Id.* at 1–2.

### B. Expert Reports

#### 1. Reports of Dr. Steinman

Although Dr. Steinman focused primarily on causation in his reports, he addressed Mr. Johnson's diagnosis throughout. *See generally* Pet'r's Ex. 13. In his initial report, Dr. Steinman acknowledged that Mr. Johnson's diagnoses included "diseases of both the central and peripheral nervous systems including [MS], [TM], [ADEM], and axonal sensory motor neuropathy." *Id.* at 10. Dr. Steinman based his theory on the concept of molecular mimicry by arguing that the flu

[12] Respondent's expert, Dr. He, did not opine on Mr. Johnson's diagnosis, and thus his reports are not discussed in this Ruling. *See generally* Resp't's Ex. A at 4–5.

vaccine has "extensive molecular mimicry with two proteins in the Node of Ranvier in both the central and peripheral nervous system." These proteins were "associated with central nervous system diseases like [MS] as well as inflammatory peripheral axonal sensorimotor neuropathies." *Id.*

Dr. Steinman also found it "remarkable that [Mr. Johnson] was treated with Tysabri for [MS]," as Tysabri "is the first monoclonal antibody approved for treatment of MS." *Id.* Dr. Steinman focused on Mr. Johnson's [MS] differential diagnosis in his report, referencing several articles and proteins linked to the development of MS. *See* Pet'r's Ex. 13 at 16 (referencing self-authored articles[13] showing "that a molecular mimic between EBNA-1 and a protein called GlialCAM[] triggers [MS]"). He used these articles, in conjunction with a BLAST report of contactin-1 and contactin-2 proteins against the flu vaccine, to show the vaccine contained homologies with sequences of five or more identifies in runs of 12 amino acids and therefore could be a trigger for MS. *Id.* at 25. (explaining that "an epitope with [five] identical amino acids out of 12 was described as a trigger for MS" for the Epstein Barr virus). He also noted the "very strong homology" shared by contactin-1, contactin-2, and GlialCAM, and argued that the molecular mimicry between those proteins and the H1N1 portion of Mr. Johnson's vaccine "may explain the neuroinflammation seen in MS and in other neuroinflammatory conditions of the central and peripheral nervous system." *Id.* at 29. When specifically addressing the issue of diagnosis, Dr. Steinman stated that Mr. Johnson's medical record contained "strong references to . . . [MS], ADEM, and inflammatory sensorimotor neuropathy, all diagnosed by neurologists at leading medical institutions including the Mayo Clinic." *Id.* at 33. He later took note of Mr. Johnson's prior mononucleosis diagnosis, arguing that "[t]he infection with EBV may have set the necessary initial condition for the development of extensive neuroinflammation including a diagnosis of MS." *Id.* at 34.

In his supplemental report, Dr. Steinman clarified Mr. Johnson's diagnosis as a "[w]idespread [n]euroinflammatory [d]isease of [b]oth the [c]entral and the [p]eripheral [n]ervous [s]ystems." Pet'r's Ex. 35 at 7. He continued by arguing that contactin-1 and contactin-2 are not only linked to the development of chronic inflammatory demyelinating polyneuropathy ("CIDP"), but also GBS and AMAN. *Id.* (citing Pet'r's Ex. 36).[14] Dr. Steinman also referenced an article he authored examining the relationship between GlialCAM and MS, explaining:

> GlialCAM and paranodal proteins share similar structures[.] GlialCAM is a member of the immunoglobulin supergene family, one of the four major types of adhesion molecules. . . . The paranodal proteins, contactin, contactin-associated protein and neurofascin are members of this family and share significant homology with GlialCAM. . . . [C]ontactin-1 has a 24 per cent identity with GlialCAM. Explorations of cross-reactive antibodies to EBNA-1 with these paranodal proteins in inflammatory neuropathy are indicated. Infections with EBV account for about

[13] Tobias v. Lanz et al., *Clonally Expanded B Cells in Multiple Sclerosis Bind EBV EBNA1 and GlialCAM*, 603 NATURE 321 (2022); William H. Robinson & Lawrence Steinman, *Epstein-Barr Virus and Multiple Sclerosis*, 375 SCIENCE 264 (2022); Kjetil Bjornevik et al., *Longitudinal Analysis Reveals High Prevalence of Epstein-Barr Virus Associated With Multiple Sclerosis*, 375 SCIENCE 296 (2022).

[14] Jérôme J. Devaux et al., *Nodal Proteins Are Target Antigens in Guillain-Barré Syndrome*, 17 J. PERIPHERAL NERVOUS SYS. 62 (2012).

> 10% of GBS cases. Antibodies to paranodal proteins are found in MS and in both [GBS] and chronic inflammatory polyneuropathy."

*Id.* at 8 (citing Pet'r's Ex. 37).[15]

#### 2. Report of Dr. Wilson

In Dr. Wilson's report, he agreed with Dr. Steinman and Mr. Johnson's treating physicians that Mr. Johnson "developed a fulminant CNS syndrome in November 2015 that was likely demyelinating," though he acknowledged Mr. Johnson's brain biopsy could not rule out a malignant lymphoma. Resp't's Ex. C at 4. He also pointed to Mr. Johnson's failure to respond to repeated rounds of aggressive immunomodulatory therapy as evidence that it was less likely Mr. Johnson had "a 'typical' form of relapsing remitting MS, or even the more aggressive demyelinating disease, NMO." *Id.* He also acknowledged that "the Mayo Clinic pathologist noted that the biopsy findings were not typical for primary demyelination given the changes consistent with axonal injury." *Id.*

Dr. Wilson also stated that the evidence for a primary peripheral neuropathy was unclear. Resp't's Ex. C at 4. He explained that Mr. Johnson's symptoms of "vision loss, urinary retention, and paraplegia progressing to quadriplegia, were all consistent with the extensive lesion burden seen on the brain and spine MRIs (i.e., CNS injuries), and a concomitant peripheral nerve injury[] is not necessary to explain this presentation." *Id.* Although Mr. Johnson exhibited decreased tone and absent reflexes consistent with a peripheral nerve injury, Dr. Wilson argued that "it is well recognized that patients with severe spinal cord injury" can present with these symptoms. *Id.* Dr. Wilson also took note of Mr. Johnson's EMG findings, which found the most likely localization for the pathology to be in the anterior horn cells in the spinal cord and that Mr. Johnson's changes "could be consistent with a critical illness neuromyopathy. . . rather than part of the presenting disease process." *Id.* Dr. Wilson also emphasized that Mr. Johnson's EMG "showed no evidence of temporal dispersion or conduction block, hallmarks of a peripheral demyelinating process." *Id.* at 5. Dr. Wilson continued by explaining Mr. Johnson lacked the cerebrospinal fluid ("CSF") markers present in "the vast majority of MS patients" in either of his CSF exams. *Id.* Dr. Wilson therefore concluded that Mr. Johnson likely suffered from "a Marburg variant of MS[,] . . . ADEM or aquaporin-4 antibody negative NMO." *Id.*

## V. Parties' Arguments

### A. Petitioner's Brief

In her brief, Petitioner discussed the diagnostic criteria and characteristics for AMSAN, the expert witnesses' conclusions regarding Mr. Johnson's diagnosis, and the different diagnoses posited by Mr. Johnson's treating physicians in the medical record. Petitioner acknowledged that Mr. Johnson's "treating physicians grappled with diagnosis;" however, she argued "despite any potential indications of a CNS demyelinating disease process like MS, NMO, or TM, [Mr.

[15] Tobias V. Lanz et al., *Roadmap for Understanding Mechanisms on How Epstein-Barr Virus Triggers Multiple Sclerosis and for Translating These Discoveries in Clinical Trials*, 12 CLINICAL TRANSLATIONAL IMMUNOLOGY 1438 (2023).

Johnson] did not have those conditions but did have AMSAN." Pet'r's Br. at 12. Petitioner noted that Mr. Johnson's CSF cytology revealed elevated protein levels (92 mg/dL) and a white blood cell count of 22 cells/uL on December 4, 2015, and argued this is supportive of a GBS diagnosis. *Id.* at 18; Pet'r's Ex. 5 at 179, 199. She similarly cited to Mr. Johnson's February 25, 2016 EMG, which revealed "low amplitude motor responses with prolonged compound muscle action potential durations and no evidence of temporal dispersion or conduction block," as being supportive of a GBS diagnosis. Pet'r's Br. at 18; Pet'r's Ex. 7 at 38. Petitioner took issue with Dr. Wilson's skepticism of Mr. Johnson's EMG results, as it was conducted three months after onset, and cited to Dimachkie & Barohn[16] to argue that conduction blocks typically seen in AMAN may disappear within two-to-five weeks. Pet'r's Br. at 19 (citing Pet'r's Ex. 39 at 6). "Therefore, if the timing of [Mr. Johnson's] electrodiagnostic testing is limiting for diagnostic purposes as Dr. Wilson suggests, it is reasonable to conclude that such testing performed earlier in [Mr. Johnson's] disease course could actually have further supported an axonal neuropathy." *Id.*

Petitioner conceded that Mr. Johnson's testing "revealed evidence of CNS involvement," including several brain and spinal lesions. Pet'r's Br. at 19. However, Petitioner cited to a case study by Okumura et al.,[17] which detailed the clinical course of a GBS patient with CNS lesions, to show that CNS involvement does not rule out a diagnosis of GBS. *Id.* (citing Pet'r's Ex. 40 at 1). The report noted that although GBS is "a predominately motor neuropathy with few sensory features," CNS manifestations had been reported in GBS cases of both adults and children. Pet'r's Ex. 40 at 1. The report also noted that in such cases, the patient typically developed CNS related symptoms, such as reduced consciousness, seizure, or brain stem impairment. *Id.* at 2.

Petitioner next addressed Mr. Johnson's symptoms and clinical progression. She argued that Mr. Johnson's weakness of both lower limbs in late November 2015, decreased deep tendon reflexes at both knees and ankles and silent plantar responses bilaterally on December 5, 2015, and a failure to respond to aggressive immunomodulatory therapies throughout his treatment satisfies criterion A-D of the Vaccine Table qualifications and aids to interpretation ("QIAs") of AMSAN. Pet'r's Br. at 19–20; *see also* 42 C.F.R. § 100.3 14(c)(15)(iv). Petitioner continued by arguing that Mr. Johnson's illness followed a monophasic course, as "there is no indication that [his] disease involved two or more separate episodes of inflammation or symptoms." *Id.* at 20. She explained that Mr. Johnson "experienced a clinical plateau with stabilization at the nadir of symptoms, which was confounded by other conditions and sequelae that lead to the deterioration of his health," noting that his condition did not improve and then subsequently worsened as would be expected in a bi- or polyphasic illness. *Id.*

Petitioner also pointed to the opinions of Mr. Johnson's treating physicians for support of a GBS diagnosis, particularly that of Dr. Rivers on December 3, 2015, where she initially considered GBS upon Mr. Johnson's initial admission to the hospital, but noted this was less likely due to his urinary retention. Pet'r's Br. at 21. Petitioner cited to Damachkie & Barohn to explain this, which found that approximately 5% of GBS cases can mimic a spinal cord lesion and can include symptoms of urinary retention. *Id.* (citing Pet'r's Ex. 39 at 2). Petitioner also pointed to

[16] Mazen D. Dimachkie & Richard J. Barohn, *Guillain-Barré Syndrome and Variants*, 31 NEUROLOGIC CLINICS 491 (2013).

[17] A. Okumura et al., *Guillain-Barré Syndrome Associated with Central Nervous System Lesions*, 86 ARCHIVES OF DISEASE IN CHILDHOOD 304 (2002).

the report of Dr. Driver-Dunckley on February 25, 2016, who initially suspected acute TM with an acute motor axonal neuropathy and later amended her diagnosis to subacute TM and a superimposed sensory motor axonal neuropathy in the setting of vaccination. *Id.* at 21–22 (citing Pet'r's Ex. 7 at 19). Petitioner also noted that Dr. Driver-Dunckley did not consider MS as a viable diagnosis. *Id.* at 22.

Petitioner further argued that Mr. Johnson's symptoms began no later than 18 days after vaccination and therefore falls within the Table criteria for flu vaccine-induced GBS. Pet'r's Br. at 24; 42 C.F.R. § 100.3 (a)(XIV)(D). She concluded by reviewing each QIA for GBS, arguing that (1) Mr. Johnson experienced weakness of both lower limbs and absent reflexes on December 5, 2015; (2) Mr. Johnson's condition slowly deteriorated over time with no response to treatment or relapse, being unable to ambulate by December 5, 2015, and that his presenting symptoms never improved or resolved; (3) that Mr. Johnson's nadir of weakness was at his December 5, 2015 hospital visit, with an approximate onset date of November 25, 2015, making the interval between onset and nadir 10 days; (4) that Mr. Johnson never reached a clinical plateau and stabilization of his symptoms before his death; and (5) that there was no more likely alternative diagnosis, as the notes made by Mr. Johnson's treating physicians were "merely suspected differential diagnoses." Pet'r's Br. at 24–27. With regard to the final QIA, Petitioner argued that Mr. Johnson's various diagnoses relating to a CNS disease, MS, NMO, or TM were not clear diagnoses, and that his principal diagnosis upon death was "fulminant idiopathic inflammatory demyelinating disease." *Id.* at 27 (citing Pet'r's Ex. 7 at 503). Petitioner dismissed Dr. Wilson's consideration of the Marburg variant of MS as it is "poorly understood." *Id.* at 28. She also cited to Thompson et al.[18] to argue that a diagnosis of MS should be approached cautiously when CSF oligoclonal bands are not detected, as in the case of Mr. Johnson. *Id.* (citing Resp't's Ex. C, Tab 4 at 5).

**B. Respondent's Brief**

Respondent began with Mr. Johnson's ultimate diagnosis, noting that "nearly every treating physician identified that [Mr. Johnson] had a disease of the [CNS,]" and arguing that CNS and brain involvement, as shown on testing, preclude a diagnosis of GBS. Resp't's Br. at 12. Respondent continued that at both of Mr. Johnson's admissions to St. Joseph's, at Orange County Global Medical Center, and at the Mayo Clinic, each treating physician identified Mr. Johnson as having a disease of the CNS. *See id.* Respondent also argued that it is significant that Mr. Johnson's treaters postulated his diagnosis to be a disease of the CNS, such as MS, NMO, or TM, and not GBS, a disease of the peripheral nervous system. *Id.* at 13. Further, Respondent noted that both Petitioner's expert, Dr. Steinman, and Respondent's expert, Dr. Wilson, opined that Mr. Johnson was suffering from some sort of disease effecting the CNS. *Id.*; *see also* Pet'r's Ex. 13 at 10; Pet'r's Ex. 35 at 7; Resp't's Ex. C at 4.

Respondent also argued that Mr. Johnson was never diagnosed with GBS or AMSAN at any point in his treatments. Resp't's Br. at 14. He noted that the only reference to GBS was by a provider early in Mr. Johnson's clinical course, and it was eventually dropped from Mr. Johnson's differential diagnosis. *Id.* Respondent also pointed to Mr. Johnson's IVIG treatments in late December 2015 and January 2016 and Cytoxan treatment in March 2016 to show that his

[18] Alan J. Thompson et al., *Diagnosis of Multiple Sclerosis: 2017 Revisions of the McDonald Criteria*, 17 LANCET NEUROLOGY 162 (2018).

symptoms mildly improved before deteriorating again after each treatment. *Id.* "These treatment related functions are simply not consistent with GBS after nine weeks of symptom onset." *Id.* Respondent also noted that Dr. Steinman did not opine Mr. Johnson had AMSAN in his reports as his original report referred only to two references of AMSAN in the medical record. Dr. Steinman later clarified his opinion on diagnosis to be a widespread neuroinflammatory disease of both the central and peripheral nervous systems in his second report. *Id.* at 14–15.

Finally, Respondent also pointed to Dr. Wilson's argument regarding Mr. Johnson's EMG/NCS testing, which found "the anterior horn cells, which are housed in the spinal cord, were the most likely localization of pathology." Resp't's Br. at 16. Respondent argued that this was consistent with a critical illness neuromyopathy, not a disease process, as opined by Dr. Larson at the Mayo Clinic. *Id.* at 15 (citing Pet'r's Ex. 7 at 124). Respondent further argued that the lack of temporal dispersion and conduction block coupled with extensive imaging revealing lesions on the brain and spine were inconsistent with a diagnosis of GBS. *Id.*

Turning to the nadir of Mr. Johnson's symptoms, Respondent argued that it was outside the Table timeframe. Resp't's Br. at 17. He noted that although Petitioner argued December 5, 2015, was the nadir of Mr. Johnson's symptoms, Mr. Johnson's symptoms continued to worsen after this date. *Id.* at 17–18. Respondent noted that Mr. Johnson's symptoms progressed until he lost all function in both upper limbs, along with gradual ascending loss of sensation and fluctuation of his vision and speech on January 5, 2016. *Id.* at 18. Therefore, according to Respondent, Mr. Johnson did not reach the nadir of his symptoms until 41 days from onset at the earliest, outside the 28-day requirement for the Table. *Id.* at 19.

Respondent then argued that Mr. Johnson never reached a clinical plateau that eventually stabilized as required in a flu-GBS Table claim. Resp't's Br. at 19. He contested Petitioner's assertion that Mr. Johnson reached a plateau because "he experienced a prolonged deterioration of health and sequelae" as contradictory for failing to consider the entire clinical picture. *Id.* Respondent pointed to the fluctuation of Mr. Johnson's symptoms in January 2016 and March 2016 to show that "his condition deteriorated over time with mild improvements before deteriorating again." *Id.* at 19–22. Respondent also argued that by Petitioner's own admission, Mr. Johnson did not have a monophasic illness pattern, highlighting language from Petitioner's brief that reads "[his] weakness did not ever improve and then subsequently worsened, as in a bi- or polyphasic illness pattern." *Id.* at 22 (quoting Pet'r's Br. at 20).

## VI. Legal Standards

To receive compensation under the Vaccine Act, a petitioner must demonstrate either that: (1) the petitioner suffered a "Table injury" by receiving a covered vaccine and subsequently developing a listed injury within the time frame prescribed by the Vaccine Injury Table set forth at 42 U.S.C. § 300aa-14, as modified by 42 C.F.R. § 100.3; or (2) the petitioner suffered an "off-Table injury," one not listed on the Table, as a result of his receiving a covered vaccine. *See* 42 U.S.C. §§ 300aa-11(c)(1)(C); *Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1319–20 (Fed. Cir. 2006).

Special masters, as finders of fact, "are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence." *Moberly*, 592 F.3d at 1326. The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. § 11(c)(2). The special master is required to consider "all [] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as "the results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." § 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Hum. Servs*., 3 F.3d 415, 417 (Fed. Cir. 1993). Pursuant to Vaccine Act § 13(a)(1)(A), a petitioner must prove her claim by a preponderance of the evidence. A special master must consider the record as a whole but is not bound by any diagnosis, conclusion, judgment, test result, report, or summary concerning the nature, causation, and aggravation of the petitioner's injury or illness that is contained in a medical record. § 13(b)(1).

In Program cases, contemporaneous medical records and the opinions of treating physicians are favored. *Capizzano*, 440 F.3d at 1326 (citing *Althen v. Sec'y of Health & Hum. Servs.*, 418 F.3d 1274, 1280 (Fed. Cir. 2005)). This is because "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.'" *Id.* In addition, "[m]edical records, in general, warrant consideration as trustworthy evidence." *Cucuras v. Sec'y of Health & Hum. Servs.*, 933 F.2d 1525, 1528 (Fed. Cir. 1993). Indeed, contemporaneous medical records are ordinarily to be given significant weight due to the fact that "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." *Id.* Contemporaneous medical records are also generally found to be afforded greater weight than contradictory oral testimony. *United States v. U.S. Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.").

However, there is no "presumption that medical records are accurate and complete as to all of the patient's physical conditions." *Kirby v. Sec'y of Health & Hum. Servs.*, 997 F.3d 1378, 1383 (Fed. Cir. 2021) (finding that a special master must consider the context of a medical encounter before concluding that it constitutes evidence regarding the absence of a condition.). While a special master must consider these opinions and records, they are not "binding on the special master or court." § 13(b)(1). Rather, when "evaluating the weight to be afforded to any such . . . [evidence], the special master . . . shall consider the entire record." *Id.*

In determining the accuracy and completeness of medical records, special masters will consider various explanations for inconsistencies between contemporaneously created medical records and later given testimony. The Court of Federal Claims has identified four such explanations for explaining inconsistencies: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of

symptoms that did not exist. *La Londe v. Sec'y of Health & Hum. Servs.*, 110 Fed. Cl. 184, 203 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014).

## VII. Discussion

On December 3, 2015, 22 days after vaccination, Mr. Johnson presented to Dr. Rivers at St. Joseph's Orange Hospital with complaints of blurry vision, urinary retention, and worsening lower extremity weakness. In addition to considering a primary spinal cord lesion, Dr. Rivers opined that Mr. Johnson's symptoms may be from GBS; however, she noted that his urinary retention made this diagnosis less likely. This was the first mention of GBS in Mr. Johnson's medical record and the only instance of a provider directly implicating GBS in Mr. Johnson's clinical course. Mr. Johnson's providers consistently considered a disease process involving the CNS throughout the course of his treatment, including when AMSAN briefly entered as a differential diagnosis on February 25, 2016, only to be later dropped. I find that these contemporaneous records provide preponderant evidence that Mr. Johnson did not suffer from GBS or AMSAN.

Petitioner's argument that Mr. Johnson suffered from AMSAN rests on two key contentions: (1) that Mr. Johnson's treating physicians suspected he suffered from GBS or AMSAN; and (2) that Mr. Johnson meets the QIAs required for a Table GBS claim. As discussed below, I find that these reasons and the evidence provided to support them do not sufficiently explain the inconsistencies between the contemporaneously created medical records and the later provided arguments in this case.

### A. The Opinions of Mr. Johnson's Treating Physicians

Petitioner relies heavily on three pieces of evidence in the medical record to support the claim that Mr. Johnson's physicians believed he suffered from GBS: 1) Mr. Johnson's initial progress notes on December 3, 2015, where Dr. Rivers opined about the possibility of GBS but noted the diagnosis to be less likely due to his urinary retention; 2) the assessment of possible AMSAN with subacute TM following MRI, CT, and EMG testing by Dr. Okazaki on February 25, 2016; and 3) a consultation note from a PA on March 1, 2016, noting that Mr. Johnson had been initially diagnosed with GBS by his California doctors, but this diagnosis had later been revised to a form of aggressive MS. However, these records, even together, within the entirety of Mr. Johnson's treatment course, do not provide persuasive evidence that his providers ever seriously considered GBS or AMSAN as a possibility.

First, Petitioner points to Mr. Johnson's December 3, 2015 progress note from Dr. Rivers to show his providers suspected GBS. However, this note was created before any specific testing was performed on Mr. Johnson, and such testing was explicitly ordered by Dr. Rivers to further investigate Mr. Johnson's symptoms. *See* Pet'r's Ex. 3-3 at 800 (Dr. Rivers ordering a head CT and neurology consult to further examine Mr. Johnson's symptoms). Further, Dr. Rivers herself cautioned against a GBS diagnosis, stating that "urinary retention makes this less likely." *Id.* It is also notable that at the subsequent neurology consultation ordered by Dr. Rivers, neurologist Dr. Pathak reviewed Mr. Johnson's imaging and opined that he had a disease of the CNS, such as MS or NMO. *Id.* at 810. Dr. Pathak also opined that his presentation was "least consistent with

peripheral polyneuropathy," the same disease category as GBS and AMSAN. *Id.* Additionally, from Mr. Johnson's original hospitalization on December 3, 2015, to his treatment at the Mayo Clinic, his physicians consistently identified his disease to be related to the CNS. Pet'r's Ex. 3-1 at 47, 53, 59, 62, 69, 127, 131, 133, 142, 199, 203, 207, 215, 222, 231, 244, 260, 279 (noting differential diagnoses of MS, NMO, and ADEM); Pet'r's Ex. 3-3 at 472, 493, 632, 640, 648, 657, 665, 792, 807, 810, 816, 819, 823, 827, 830, 834, 838, 841, 845, 847, 851, 854, 856, 862, 864 (noting differential diagnoses of NMO, MS, and disseminated encephalomyelitis); Pet'r's Ex. 6 at 4, 55, 59, 63, 220, 225, 238, 243, 247, 255, 264, 273, 285, 289, 293, 297, 306, 310 (noting a diagnosis of MS); Pet'r's Ex. 7 at 61, 85, 101, 109, 126, 154, 168, 179, 196, 204, 212, 219, 231, 241, 270, 277, 298, 312, 327, 339, 361, 390, 413, 429, 493, 510 (noting concern for "demyelinating disease in the CNS [with longitudinally] extensive spinal cord lesions as well as a peripheral process involving motor nerve/[anterior horn cells]").

Second, Petitioner relies on part of Dr. Driver-Dunckley's differential diagnosis from Mr. Johnson's initial intake at the Mayo Clinic where she opined that "acute [TM] with likely an acute motor axonal neuropathy and perhaps even encephalomyelitis" were a possibility. Pet'r's Ex. 7 at 19. However, this evidence is not persuasive for two reasons. First, in the same note relied on here by Petitioner, Dr. Driver-Dunckley stated that she suspected an encephalomyeloneuropathy (a CNS disease) is the most likely diagnosis, not AMSAN or GBS (a peripheral nervous system disease). *Id.* Notably, this assessment was made solely on Mr. Johnson's then physical presentation and his own accounting of his medical history, as the Mayo Clinic did not have access to his prior imaging or testing upon his arrival. *See id.* (requesting that Mr. Johnson's sister obtain all medical records and bring them prior to imaging). Upon the completion of this testing, Dr. Okazaki opined that the results were concerning for a demyelinating disease in the CNS in addition to a "peripheral process involving motor nerve/[anterior horn cells]." *Id.* at 64. The ultimate impression was "sequelae of (a previously acute), now subacute [TM], central demyelinating disease, and a[] superimposed sensory motor axonal neuropathy in the setting of vaccination." *Id.* at 85. This remained Mr. Johnson's differential diagnosis until his death on March 20, 2016, and at no point was GBS considered. Further, although the Mayo Clinic requested Mr. Johnson's records, they never received them before his death, and instead, relied on Mr. Johnson's self-reported diagnosis of previous GBS to frame his diagnosis. *See id.* at 110 (noting on February 28, 2016, that a request for outside records had been placed to obtain actual disks).

Third, Petitioner relies on the note from PA Walker on March 1, 2016, which stated that Mr. Johnson "was initially diagnosed with [GBS] in California but this was revised to be an aggressive form of [MS]." Pet'r's Ex. 7 at 126. Similar to the notes relied on by Petitioner above, this note does not reference present GBS and relies on Mr. Johnson's self-reported history, as the Mayo Clinic still had not received his prior hospitalization records. Further, it is clear that Mr. Johnson's physicians did not believe him to have a primary GBS or AMSAN diagnosis, as he began treatment on the MS drug Cytoxan due to elevated albumin and IgG on his MS panel. *Id.* at 242. Petitioner's argument that Mr. Johnson suffered from GBS and not any form of a CNS disease is further undercut by her own expert's opinions, as Dr. Steinman first appeared to focus his theory on a primary diagnosis of MS before explicitly stating he believed Mr. Johnson suffered from both diseases in the CNS and peripheral nervous system.

Accordingly, the opinions of Mr. Johnson's treating physicians relied on by Petitioner does not show by preponderant evidence that he suffered from GBS or AMSAN.

**B. Table QIA Criteria for GBS**

Petitioner further argues that Mr. Johnson meets the Table QIA for a GBS AMSAN diagnosis and thus should succeed on her claim. According to the Table QIA, a diagnosis of GBS AMSAN requires a showing of the following:

(A) Bilateral flaccid limb weakness and decreased or absent deep tendon reflexes in weak limbs;

(B) A monophasic illness pattern;

(C) An interval between onset and nadir of weakness between 12 hours and 28 days;

(D) Subsequent clinical plateau (the clinical plateau leads to either stabilization at the nadir of symptoms, or subsequent improvement without significant relapse; however, death may occur without a clinical plateau); and,

(E) The absence of an identified more likely alternative diagnosis.

42 C.F.R. § 100.3(c)(15)(ii). As discussed below, I do not find preponderant evidence that Petitioner has satisfied all five of the Table QIA criteria for a GBS AMSAN diagnosis.

**1. Criterion A: Bilateral flaccid limb weakness and decreased or absent deep tendon reflexes in weak limbs**

Petitioners seeking to establish a Table GBS claim must show they suffered bilateral flaccid limb weakness and decreased or absent deep tendon reflexes in weak limbs. *See* 42 C.F.R. § 100.3(c)(15)(ii)(A). Petitioner alleges that Mr. Johnson suffered from bilateral flaccid limb weakness and absent deep tendon reflexes early in his clinical progression. Respondent does not contest this criterion. Indeed, during Mr. Johnson's December 3, 2015 hospitalization, Dr. Pathak noted reduced deep tendon reflexes and silent plantar responses bilaterally. Pet'r's Ex. 3-3 at 811. Mr. Johnson's lower limb weakness continued and eventually progressed into quadriplegia prior to his death. *See generally* Pet'r's Ex. 7. Accordingly, I find preponderant evidence that Petitioner has satisfied the first criterion.

**2. Criterion B: Monophasic illness pattern**

The Table QIAs for GBS also require petitioners to establish that their illness followed a monophasic pattern. While treatment related fluctuations can occur within nine weeks of onset, recurrence of symptoms after this timeframe would not be consistent with GBS. 42 C.F.R. § 100.3(c)(15)(i). Here, Petitioner alleges that Mr. Johnson's illness progressed in a monophasic pattern, noting that his condition "slowly deteriorated over time, with no significant response to treatment and no significant relapse." Pet'r's Br. at 26. Petitioner described Mr. Johnson's later

presenting symptoms as "complicated by subsequent infections and what could be described as ongoing GBS sequelae." *Id.* Respondent contests this by quoting Petitioner's brief that Mr. Johnson's "weakness did not ever improve and then subsequently worsened, as in a bi- or polyphasic illness pattern," as an admission that Petitioner did not have a monophasic illness pattern," Resp't's Br. at 21 (quoting Pet'r's Br. at 20). However, Respondent's argument on this matter is rooted in a misreading of Petitioner's brief and is taken out of context. Petitioner's brief, in its entirety, reads as follows:

> Decedent experienced a clinical plateau with stabilization at the nadir of symptoms, which was confounded by other conditions and sequelae that lead to the deterioration of his health. His weakness did not ever improve and then subsequently worsened, as in a bi- or polyphasic illness pattern.

Pet'r's Br. at 20. Here, it is clear that Petitioner is not making an admission that Mr. Johnson suffered from a bi- or polyphasic illness but rather is directly contesting this point. However, despite Respondent's insufficient argument on this point, the record reflects that Mr. Johnson did not suffer a monophasic illness pattern.

According to the record, Mr. Johnson's symptoms began on approximately November 28, 2015. *See* Pet'r's Ex. 2 at 15 (noting a two-day history of right eye problems and dizziness on November 30, 2015). Nine weeks from this date would be January 30, 2016. Although Mr. Johnson did experience some periods of improvement following his December 2015 and January 2016 IVIG treatments, his symptoms continued to fluctuate through March 2016. *See* Pet'r's Ex. 7 at 127 (intact sensation in all extremities on March 1, 2016); *see also id.* at 162 (active voluntary movement in left arm, trace function in right arm, no function in lower extremities on March 2, 2016); *id.* at 212 (no sensation in bilateral lower extremities on March 7, 2016); *id.* at 312 (intact sensation in upper extremities and no sensation in lower extremities, increased movement in right arm on March 16, 2016). These fluctuations, well outside the nine weeks following onset allowed by the Table, are inconsistent with a monophasic illness pattern associated with GBS. Petitioner argues that Mr. Johnson's symptoms never improved or resolved; however, his increasing right arm movement throughout March 2016 and his contrasting worsening of his bilateral lower extremity sensation contradict this assertion. Accordingly, Petitioner has not established that Mr. Johnson meets the second criterion.

### 3. Criterion C: An interval between onset and nadir of weakness between 12 hours and 28 days

The Table requires that there be an interval between the onset and nadir of weakness between 12 hours and 28 days. *See* 42 C.F.R. § 100.3(c)(15)(ii)(C). Here, the record reflects that Mr. Johnson did not reach the nadir of his symptoms until well beyond the 28 days allowed by the Table. Although Petitioner is correct that Mr. Johnson's legs became completely immobilized on approximately December 5, 2015, she fails to consider Mr. Johnson's progressive upper extremity decline. Around January 5, 2016, Mr. Johnson was recorded as having reduced reflexes in both upper extremities, flaccid paralysis of the right upper extremity, and 1/5 distal and 3/5 proximal strength of the left upper extremity, and new onset of a right-sided facial droop. *See* Pet'r's Ex. 3-

3 at 222. This was approximately 39 days after onset, and well outside the range defined by the Table. Accordingly, Petitioner has not established that Mr. Johnson meets the third criterion.

**4. Criterion D: Subsequent clinical plateau**

The fourth Table QIA criterion requires that the injured person must reach a clinical plateau with stabilization at the nadir of symptoms. 42 C.F.R. § 100.3(c)(15)(ii)(D). However, the vaccinee may die before reaching their clinical plateau. 42 C.F.R. § 100.3(c)(15)(i). Here, Petitioner argues that Mr. Johnson passed away before reaching his clinical plateau, as "[t]here is no evidence of improvement or subsequent relapse – only prolonged deterioration of health and sequelae." Pet'r's Br. at 26. However, this is directly contradictory to Petitioner's above argument that Mr. Johnson reached the nadir of his symptoms on December 5, 2015. As discussed earlier, there is preponderant evidence that Mr. Johnson did not reach the nadir of his symptoms until January 5, 2016, and there was no stabilization at this point, as Mr. Johnson's symptoms continued to fluctuate until his death in March 2016. Accordingly, Petitioner has not established that Mr. Johnson meets the fourth criterion.

**5. Criterion E: The absence of a more likely alternative diagnosis**

Finally, the Table requires that no more likely alternative diagnosis be identified. 42 C.F.R. § 100.3(c)(15)(ii)(E). Evidence that is supportive of a GBS diagnosis, but not required, includes electrophysiologic findings consistent with GBS or an elevation of CSF protein with a total CSF white blood cell count below 50 cells per microliter. *Id.* at § 100.3(c)(15)(iv). Conversely, the ultimate diagnosis of any of the following conditions exclude the existence of a GBS Table claim: spinal cord infarct, anterior horn cell diseases, MS, and critical illness neuropathy. *Id.* at § 100.3(c)(15)(vi).

Here, the record reflects a litany of possible diagnoses of the CNS, many of which are directly exclusionary of a GBS Table claim. Mr. Johnson's treating physicians primarily focused on diseases of the CNS, including MS, NMO, and TM. Further, Mr. Johnson's EMG testing was directly unsupportive of a GBS diagnosis, instead showing results consistent with anterior horn cell disease or a superimposed critical illness neuropathy, both of which are exclusionary diagnoses for a GBS Table claim. *See* Pet'r's Ex. 7 at 38.

Petitioner points to elevated CSF protein with a CSF white blood cell count of 22 on December 4, 2015, and Mr. Johnson's February 25, 2015 NCS testing, which revealed low amplitude motor responses, as supportive evidence of a GBS AMSAN diagnosis. However, Mr. Johnson's same NCS testing revealed no evidence of temporal dispersion or conduction block. While the Dimachkie & Barohn article does show such findings may be consistent with an AMSAN diagnosis, it does not explain the persistent consideration of a CNS disease by Mr. Johnson's treating physicians, their explicit disregard of a possible peripheral pathology, and the clear imaging evidence of CNS involvement in the form of several lesions on both the brain and spine. It is also incredibly persuasive that Petitioner's own expert, Dr. Steinman, approached this case as though Mr. Johnson suffered from a disease of the CNS, which Petitioner now asserts Mr. Johnson did not have. Accordingly, I find that Petitioner has failed to establish that Mr. Johnson meets the fifth criterion.

## VIII. Conclusion

After a careful review of the record, Petitioner has failed to provide preponderant evidence that Mr. Johnson suffered from AMSAN, or any other type of GBS. Any subsequent action that Petitioner takes should be consistent with Petitioner's arguments that Mr. Johnson suffered from GBS; and therefore, a factual finding on diagnosis would be outcome determinative. Petitioner has thirty (30) days from the filing of this Ruling to file a status report indicating how she wishes to proceed.

**IT IS SO ORDERED.**

s/Herbrina D. S. Young
Herbrina D. S. Young
Special Master