In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: September 23, 2024

DONNA BAUER, *surviving spouse and heir-at-law of* WILLIAM BAUER, *deceased*,	*	No. 18-1451V
Petitioner,	*	Special Master Sanders
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

William P. Ronan, III, The Ronan Law Firm, Overland Park, KS, for Petitioner.
Bridget Corridon, United States Department of Justice, Washington, DC, for Respondent.

DECISION ON ENTITLEMENT[1]

On September 21, 2018, Donna Bauer ("Petitioner") filed a petition for compensation in the National Vaccine Injury Compensation Program ("the Program")[2] on behalf of her deceased spouse, William Bauer. Pet., ECF No. 1. Petitioner alleged that the influenza ("flu") vaccine that Mr. Bauer received on October 12, 2017, caused him to suffer from Guillain-Barré syndrome ("GBS")[3] and death. *Id.* at 1. On June 28, 2022, Petitioner filed an amended petition alleging in

[1] Because this Decision 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 Decision 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] GBS is a "rapidly progressive ascending motor neuron paralysis of unknown etiology, frequently seen after an enteric or respiratory infection. An autoimmune mechanism following viral infection has been postulated. It begins with paresthesias of the feet, followed by flaccid paralysis of the entire lower limbs, ascending to the trunk, upper limbs, and face." *Dorland's Illustrated Medical Dictionary* 1468 (33rd ed.

the alternative that Mr. Bauer suffered from conditions that were caused or significantly aggravated by the vaccination at issue. *See* Am. Pet. ¶¶ 37–39, ECF No. 49.

After carefully analyzing and weighing all the evidence and testimony presented in this case in accordance with the applicable legal standards,[4] I find that Petitioner has failed to provide preponderant evidence that the flu vaccine Mr. Bauer received on October 12, 2017, caused his death, caused him to suffer from GBS, or significantly aggravated his preexisting conditions. Accordingly, Petitioner is not entitled to compensation.

I. Procedural History

Petitioner filed her petition on September 21, 2018. Pet. Petitioner also filed her affidavit, an affidavit from Christopher Jarvis, M.D., Mr. Bauer's primary care provider ("PCP"), and Mr. Bauer's medical records. Pet'r's Exs. 1–9, ECF No. 1. Petitioner filed additional medical records and a statement of completion on November 29, 2018. Pet'r's Exs. 10–12, ECF Nos. 9–10.

On April 16, 2019, I ordered Petitioner to file an expert report. Scheduling Order at 2, ECF No. 15. Petitioner filed an expert report from Dr. Jarvis on June 17, 2019. Pet'r's Ex. 13, ECF No. 18-1. Respondent submitted an expert report from Brian Callaghan, M.D., Dr. Callaghan's curriculum vitae ("CV"), and medical literature on October 7, 2019. Resp't's Exs. A–D, ECF No. 23. On November 14, 2019, Petitioner filed a supplemental expert report from Dr. Jarvis and medical literature. Pet'r's Exs. 14–16, ECF No. 24. Respondent filed a supplemental expert report from Dr. Callaghan on December 30, 2019. Resp't's Ex. E, ECF No. 26-1. On January 27, 2020, Petitioner filed a status report stating that she did not wish to file an additional supplemental expert report. ECF No. 29 at 1.

On January 25, 2022, I scheduled an entitlement hearing for June 29–30, 2022. Hearing Order, ECF No. 32. Petitioner filed a prehearing brief on May 3, 2022, and Respondent filed his prehearing brief on June 8, 2022. Pet'r's Br., ECF No. 34; Resp't's Br., ECF No. 36. Petitioner filed medical literature and a reply brief on June 22, 2022. Pet'r's Exs. 17–23, ECF No. 39; Pet'r's Reply, ECF No. 42. Respondent filed an explanation of medical literature on the same date. Resp't's Ex. F, ECF No. 41-1.

On June 24, 2022, I held a status conference with the parties to discuss the upcoming entitlement hearing and issues raised in the parties' prehearing submissions. *See* Min. Entry, docketed June 24, 2022. On June 28, 2022, Respondent filed a notice of objection to memorialize an objection raised during the status conference. Resp't's Notice, ECF No. 45. Respondent

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2020) [hereinafter "*Dorland's*"]. A paresthesia is "an abnormal touch sensation, such as burning, prickling, or formication, often in the absence of an external stimulus." *Id.* at 1362.

[4] 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.").

objected to the possibility of Petitioner alleging a new injury or raising a significant aggravation claim in the days before the entitlement hearing. *See id.* at 4–7. Respondent requested the opportunity to submit additional expert evidence following the entitlement hearing. *Id.* at 6–7.

Petitioner filed additional medical literature and an amended petition on June 28, 2022. Pet'r's Exs. 24–27, ECF No. 46; Am. Pet. The entitlement hearing was held as scheduled on June 29, 2022. Min. Entry, docketed June 29, 2022. On July 20, 2022, I ordered Respondent to file a status report indicating whether he wished to file an additional expert report or how he wished to proceed. Scheduling Order, docketed July 20, 2022. On August 3, 2022, Respondent filed a status report stating that he had engaged an additional expert. ECF No. 54 at 1.

On October 14, 2022, Respondent filed an expert report from Derek Fine, M.D., Dr. Fine's CV, and medical literature. Resp't's Exs. G–H, ECF No. 55; Resp't's Exs. I–T, ECF No. 56. On October 26, 2022, I ordered Petitioner to file a supplemental expert report or a status report indicating that she did not intend to file a response by December 27, 2022. Scheduling Order, docketed Oct. 26, 2022. After Petitioner requested additional time to review the hearing transcript, I ordered her to file a status report stating that she did not wish to file an additional expert report or information regarding the name of her expert and how long the expert would need to complete a report. ECF No. 57; Scheduling Order at 1–2, ECF No. 58. On February 3, 2023, Petitioner filed a status report stating that she did not intend to file a supplemental expert report. ECF No. 59.

This matter is now ripe for consideration.

II. Factual Background

A. Medical Records

1. Pre-vaccination Medical Records

Mr. Bauer's pre-vaccination medical history is notable for chronic renal failure,[5] type 2 diabetes mellitus,[6] hypertension,[7] dyslipidemia,[8] obesity, metabolic syndrome[9], and coronary

[5] Chronic renal failure, also known as chronic kidney disease, is "gradual loss of kidney function, with progressively more severe renal insufficiency." *Dorland's* at 523.

[6] Diabetes mellitus is "a chronic syndrome of impaired carbohydrate, protein, and fat metabolism owing to insufficient secretion of insulin or to target tissue insulin resistance." *Dorland's* at 499. Type 2 diabetes mellitus is "characterized by peak age of onset between 50 and 60 years, gradual onset with few symptoms of metabolic disturbance (glycosuria and its consequences), and no need for exogenous insulin." *Id.* at 500.

[7] Hypertension is "high arterial blood pressure." *Dorland's* at 885.

[8] Dyslipidemia is "abnormality in, or abnormal amounts of, lipids and lipoproteins in the blood." *Dorland's* at 572.

[9] Metabolic syndrome is "a combination including at least three of the following: abdominal obesity, hypertriglyceridemia, low level of high-density lipoproteins, hypertension, and high fasting plasma glucose level." *Dorland's* at 1809.

artery disease.[10] Pet'r's Ex. 7 at 54, ECF No. 1-9. On September 5, 2013, he had an estimated glomerular filtration rate ("eGFR" or "GFR")[11] of 56 while the normal value is above 59.[12] *Id.* at 2. He also had a high creatinine[13] level of 1.30 (reference range = 0.60–1.20 mg/dL) and a high blood urea nitrogen ("BUN")[14] level of 32 (reference range = 6–20 mg/dL). *Id.* at 1. On May 25, 2016, Mr. Bauer had high creatinine level of 1.26 and an eGFR of 58. *Id.* at 8.

From December 1–4, 2016, Mr. Bauer was hospitalized for chest pain, an episode of shortness of breath, and highly elevated blood pressure. Pet'r's Ex. 8 at 6, 23, ECF No. 1-10. On December 2, 2016, Michael Eisenhauer, M.D., a cardiologist, performed a coronary angiogram,[15] left heart catheterization, and ventriculography.[16] *Id.* at 14. Dr. Eisenhauer recommended treatment for symptomatic relief only but noted that "nothing . . . will reduce future risk of [myocardial infarction]." *Id.* at 15. During his hospitalization, Mr. Bauer was found to have a low potassium level of 2.9 and assessed with hypokalemia.[17] *Id.* at 22. Mr. Bauer noted that he took a potassium supplement at home, but Dr. Eisenhauer increased the dosage in the hospital due to Mr. Bauer's low levels. *Id.* When the levels remained low, Dr. Eisenhauer prescribed IV supplementation. *Id.* On discharge, Mr. Bauer's listed medications included Lotrel, or amlodipine benazepril,[18] an

[10] Coronary artery disease is "atherosclerosis of the coronary arteries, which may cause angina pectoris, myocardial infarction, and sudden death." *Dorland's* at 524. Atherosclerosis involves "formation of deposits of yellowish plaques [] containing cholesterol, lipoid material, and lipophages in the intima and inner media of large and medium-sized arteries." *Id.* at 169. Angina pectoris is "a paroxysmal thoracic pain, often radiating to the arms, particularly the left, sometimes accompanied by a feeling of suffocation and impending death." *Id.* at 82. Myocardial infarction is "gross necrosis of the myocardium as a result of interruption of the blood supply to the area." *Id.* at 923.

[11] GFR is "the quantity of glomerular filtrate formed per unit time in all nephrons of both kidneys." *Dorland's* at 1570. Glomerular filtrate is "the ultrafiltrate of plasma that passes across the membranes of the renal corpuscles into the urinary space." *Id.* at 700. Nephrons are "the anatomic and functional unit[s] of the kidney." *Id.* at 1224. The renal glomerulus is "a globular tuft formed by capillaries in the kidney, the site of the filtration barrier between the blood and the kidney." *Id.* at 778.

[12] The medical record defines chronic kidney disease "as either kidney damage or a GFR less than 60ml/min that persists for at least [three] months. Stage 3 = 30–59 ml/min[.] Stage 4 = 15–29 ml/min[.] Stage 5 = <15 ml/min." Pet'r's Ex. 7 at 2.

[13] Creatinine is "the cyclic anhydride of creatine, produced as the final product of decomposition of phosphocreatine." *Dorland's* at 424.

[14] BUN, also known as urea nitrogen, is "the urea concentration of blood or serum in terms of nitrogen content." *Dorland's* at 1975.

[15] Angiography is "the radiographic visualization of blood vessels following introduction of contrast material." *Dorland's* at 83.

[16] Ventriculography is "radiography of a ventricle of the heart after injection of a contrast medium." *Dorland's* at 2017.

[17] Hypokalemia is "abnormally low potassium concentration in the blood." *Dorland's* at 891.

[18] Amlodipine besylate is "a calcium channel blocking agent used in the treatment of hypertension and chronic stable and vasospastic angina." *Dorland's* at 63. Benazepril hydrocholoride is "an angiotensin-converting enzyme inhibitor administered orally . . . for treatment of hypertension." *Id.* at 205.

angiotensin-converting enzyme ("ACE") -inhibitor[19] (10–20 mg once per day); atenolol,[20] a beta blocker[21] (50 mg once per day); and potassium supplements (20 mg three times per day) as well as other medications. *Id.* at 25–26. Pharmacy records indicate that Mr. Bauer was prescribed spironolactone[22] around December 3, 2016. *See* Pet'r's Ex. 10 at 169, ECF No. 9-2.

On December 19, 2016, Mr. Bauer followed up with John Joliff, M.D., his cardiologist, following his hospitalization. Pet'r's Ex. 4 at 11–12, ECF No. 1-6. Dr. Joliff listed Mr. Bauer's problems as "[s]evere hypertension [that was] difficult to control[,]" coronary disease, hyperlipidemia, diabetes, and recurrent angina. *Id.* at 11. Dr. Joliff listed Mr. Bauer's medications, which included spironolactone (25 mg twice per day), Lotrel, atenolol, and potassium. *Id.* at 11–12. However, Dr. Joliff noted that "[a]pparently we had on a list that he was taking atenolol at home, he was not." *Id.* at 11. Dr. Joliff attributed the change in Mr. Bauer's symptoms to his "poorly controlled blood pressure[,]" and Dr. Joliff prescribed a different beta blocker, carvedilol,[23] to address this. *Id.* at 12–13. Dr. Joliff also increased Mr. Bauer's dosage of hydralazine.[24] *Id.* at 11. Dr. Joliff and his office continued to list spironolactone, carvedilol, and Lotrel in Mr. Bauer's medication list on January 23, March 6, and July 17, 2017. *Id.* at 14–15, 17–18, 24.

On May 17, 2017, Mr. Bauer presented to a nurse practitioner at his endocrinologist's office. Pet'r's Ex. 7 at 63. He had a high creatinine level of 2.15 and an eGFR of 31. Pet'r's Ex. 7 at 12–13. He had a normal microalbumin[25] level of 10 mg/L. *Id.* at 13. His listed medications included Lotrel, potassium, and atenolol, but not spironolactone or carvedilol. *Id.* at 64.

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[19] ACE inhibitors are "competitive inhibitors of peptidyl-dipeptidase A (angiotensin-converting enzyme)[]" and are "used for treatment of hypertension, usually in conjunction with a diuretic." *Dorland's.* at 928.

[20] Atenolol is "a cardioselective β1-adrenergic blocking agent used in the treatment of hypertension and chronic angina pectoris and the prophylaxis and treatment of myocardial infarction and cardiac arrhythmias." *Dorland's.* at 169.

[21] Beta blockers, or beta-adrenergic blocking agents, are "agent[s] that induces adrenergic blockade at either β_1- or β_2-adrenergic receptors or at both." *Dorland's* at 38.

[22] Spironolactone is "a synthetic 17-spirolactone steroid and aldosterone antagonist that is a potassium-sparing diuretic; it blocks the aldosterone-dependent exchange of sodium and potassium in the distal renal tubule, which increases excretion of sodium and water and decreases excretion of potassium." *Dorland's* at 1722. Aldosterone is "the major mineralocorticoid secreted by the adrenal cortex; it promotes retention of sodium and bicarbonate, excretion of potassium and hydrogen ions, and secondary retention of water." *Id.* at 46. Aldosterone antagonists are "any of a group of compounds that block the action of aldosterone and function as potassium-sparing diuretics." *Id.* at 96. Potassium-sparing diuretics are "a class of drugs that block the exchange of sodium for potassium and hydrogen ions in the distal tubule, causing an increase in the excretion of sodium and chloride with a negligible increase in potassium excretion." *Id.* at 552.

[23] Carvedilol is "a beta-adrenergic blocking agent used in the treatment of essential hypertension and as an adjunct in the treatment of mild or moderate congestive heart failure." *Dorland's* at 296.

[24] Hydralazine is "a peripheral vasodilator used as an antihypertensive." *Dorland's* at 865.

[25] Albumin is "the major plasma protein, approximately 60 percent of the total, which is responsible for much of the plasma colloidal osmotic pressure and serves as a transport protein for large organic anions such as fatty acids, bilirubin, and many drugs." *Dorland's* at 44.

On May 18, 2017, Mr. Bauer presented to Dr. Jarvis "with complaints of fatigue and weakness." Pet'r's Ex. 4 at 21. He also complained of sleep apnea[26] spells and nocturia.[27] *Id.* Dr. Jarvis's assessment included benign prostatic hypertrophy[28] and obstructive sleep apnea[29] to be confirmed through a home sleep study. *Id.* Mr. Bauer presented to a sleep specialist on June 21, 2017, following his home sleep study. Pet'r's Ex. 11 at 29–30, ECF No. 9-3. Mr. Bauer reported leg cramps that woke him up during the night as well as snoring and apnea spells. *Id.* at 29. He reported waking up five to eight times per night due to snoring or needing to use the bathroom. *Id.* The sleep specialist assessed Mr. Bauer with obstructive sleep apnea. *Id.* at 30.

On July 17, 2017, a nurse practitioner at Dr. Joliff's office noted that Mr. Bauer was "doing well from a cardiac standpoint." Pet'r's Ex. 5 at 182. She noted that Mr. Bauer was "able to do everything he physically want[ed] to." *Id.* He reported a recent episode of lightheadedness and dizziness and some occasional fourth- and fifth-digit numbness during the previous four to six weeks, but his blood pressure was stable. *Id.* at 180–81. His listed medications included carvedilol (12.5 mg twice per day), Lotrel (10/20 mg daily), spironolactone (25 mg twice per day), and potassium (40 mEq three times per day). *Id.* at 181.

2. Vaccination and Post-Vaccination Medical Records

Mr. Bauer received the flu vaccine at issue on October 12, 2017. Pet'r's Ex. 3 at 10, ECF No. 1-5. Five days post vaccination, on October 17, 2017, Mr. Bauer presented to Dr. Jarvis "with complaints of difficulty walking." Pet'r's Ex. 4 at 27. Mr. Bauer reported "pain down his legs, tingling down his upper arms, and it just really started hitting him in the last couple of weeks." *Id.* He complained of tingling down his arms to the fourth and fifth digits as well as "difficulty going up stairs." *Id.* Mr. Bauer also reported that "[h]is legs just give out on him[,] and he tumbles." *Id.* Dr. Jarvis noted that Mr. Bauer had "no history of any diabetic neuropathy"[30] and that his reported symptoms "just occurred in the last few weeks." *Id.* Mr. Bauer's blood pressure was measured at 120/66. *Id.*

On exam, Mr. Bauer had diminished patellar[31] and Achilles reflexes[32] and weakness in his hamstrings. *Id.* Dr. Jarvis's assessment was "[t]ingling and paresthesias over C7 dermatomes[33] as

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[26] Sleep apnea refers to "transient periods of cessation of breathing during sleep." *Dorland's* at 115.
[27] Nocturia is "urinary frequency at night." *Dorland's* at 1261.
[28] Benign prostatic hypertrophy or hyperplasia is "age-associated enlargement of the prostate resulting from proliferation of both glandular and stromal elements." *Dorland's* at 882.
[29] Obstructive sleep apnea, or obstructive apnea, is "sleep apnea resulting from collapse or obstruction of the airway with the inhibition of muscle tone that occurs during REM sleep." *Dorland's* at 115.
[30] Diabetic neuropathy is "any of several clinical types of polyneuropathy seen with diabetes mellitus." *Dorland's* at 1251. Polyneuropathy, also known as peripheral neuropathy is "neuropathy of several peripheral nerves simultaneously." *Id.* at 1468. Neuropathy is "a functional disturbance or pathologic change in the peripheral nervous system." *Id.* at 1250.
[31] The patellar reflex is "contraction of the quadriceps and extension of the lower limb when the patellar ligament is tapped." *Dorland's* at 1589.
[32] The Achilles, or triceps surae, reflex is "plantar flexion of the foot caused by a twitchlike contraction of the triceps surae muscle." *Dorland's* at 1590.
[33] Dermatomes are "area[s] of skin supplied with afferent nerve fibers by a single spinal nerve." *Dorland's* at 491.

well as lower extremity weakness and falls, symmetric." *Id.* Dr. Jarvis noted concern for spinal stenosis[34] and ordered an electromyogram ("EMG")[35] and imaging of the cervical[36] and lumbar spine.[37] *Id.* On October 18, 2017, Mr. Bauer had magnetic resonance imaging ("MRI") of his cervical spine, which revealed "[c]ervical degenerative disc disease worse at C4-5 . . . ." Pet'r's Ex. 5 at 189, ECF No. 1-7. A lumbar spine MRI revealed "[s]ignificant lumbar degenerative disc and degenerative facet joint disease, worse at L4-5 and L5-S1 . . . ." *Id.* at 191–92.

At 1:36 PM on October 20, 2017, eight days post vaccination, Mr. Bauer presented to the ER at Coffey County Hospital with complaints of dyspnea,[38] respiratory distress, and motor loss. *Id.* at 304. Dr. Jarvis evaluated Mr. Bauer in the emergency room ("ER"), and Dr. Jarvis wrote that Mr. Bauer had a "flu shot [one and a half] weeks ago." *Id.* Dr. Jarvis noted that Mr. Bauer's symptoms "started with leg weakness [on the first] of the week[39] with a couple of falls." *Id.* at 305. He continued that Mr. Bauer presented "with acute worsening of symptoms" that began one hour prior and included arm and leg weakness and difficulty breathing. *Id.* Dr. Jarvis noted that the EMG had not yet been completed. *Id.* Petitioner reported that Mr. Bauer had severe generalized weakness in his face, arms, hands, legs, and feet. *Id.* She noted that he had had "fatigue, difficulty breathing[,] and weakness[]" as well as mild hand numbness and difficulty walking. *Id.* An electrocardiogram ("EKG") revealed an atrioventricular[40] block, and lab testing showed "[m]arked hyperkalemia"[41] with a potassium level of 10.8. *Id.* at 306. Mr. Bauer's BUN was 73, and his creatinine was 3.6. *Id.* His eGFR was 18. *Id.* at 248. His alanine transaminase ("ALT")[42] level was 29 (reference range = 12–78 U/L), and his aspartate transaminase ("AST")[43] level was 17 (reference range = 15–37 U/L). *Id.* at 247. His blood pressure at 1:44 PM measured at 186/101. *Id.* at 256. Mr. Bauer was intubated and ventilated, and his blood pressure decreased. *Id.* at 256, 306. Dr. Jarvis's clinical impression included "[c]ardiac arrest[44] secondary to asystole,"[45] "[s]evere

[34] Spinal stenosis is "narrowing of the vertebral canal, nerve root canals, or intervertebral foramina of the lumbar spine caused by encroachment of bone upon the space." *Dorland's* at 1740.
[35] Electromyography is "an electrodiagnostic technique for recording the extracellular activity (action potentials and evoked potentials) of skeletal muscles at rest, during voluntary contractions, and during electrical stimulation." *Dorland's* at 595.
[36] The cervical spine comprises the cervical vertebrae, which are "the upper seven vertebrae, constituting the skeleton of the neck." *Dorland's* at 1720, 2020.
[37] The lumbar spine comprises the lumbar vertebrae, which are "the five vertebrae between the thoracic vertebrae and the sacrum." *Dorland's* at 1720, 2020.
[38] Dyspnea is "breathlessness or shortness of breath." *Dorland's* at 576.
[39] The "first of the week" would have been Sunday, October 15, 2017, or Monday, October 16, 2017.
[40] Atrioventricular "pertain[s] to both an atrium and a ventricle of the heart." *Dorland's* at 172.
[41] Hyperkalemia is "abnormally high potassium concentration in the blood, most often due to defective renal excretion." *Dorland's* at 879.
[42] ALT is "an enzyme of the transferase class that catalyzes the reversible transfer of an amino group from alanine to α-ketoglutarate to form glutamate and pyruvate, with pyridoxal phosphate as a cofactor." *Dorland's* at 43.
[43] AST is "an enzyme of the transferase class that catalyzes the reversible transfer of an amino group from aspartate to α-ketoglutarate to form glutamate and oxaloacetate, with pyridoxal phosphate required as a cofactor." *Dorland's* at 161.
[44] Cardiac arrest is "sudden cessation of the pumping function of the heart, with disappearance of arterial blood pressure." *Dorland's* at 131.
[45] Asystole is "absence of a heartbeat." *Dorland's* at 167.

acute renal failure[but n]ot chronic renal failure," "[GBS] with respiratory compromise," and "[h]yperkalemia." *Id.* at 307.

Later that afternoon, Mr. Bauer was "noted to be in a wide complex bradycardia[46] without a pulse." *Id.* at 281. His providers unsuccessfully performed cardiopulmonary resuscitation,[47] and Mr. Bauer passed away. *Id.* at 264–65, 278–81. Dr. Jarvis signed Mr. Bauer's death certificate as the pronouncing and certifying physician. Pet'r's Ex. 2 at 1, ECF No. 1-4. The certificate lists "cardiopulmonary arrest" as the immediate cause of death and hyperkalemia, coronary artery disease, and "possible" GBS as underlying causes. *Id.*

B. Affidavits and Fact Testimony

1. Petitioner

Petitioner submitted an affidavit and testified at the entitlement hearing. Pet'r's Ex. 1, ECF No. 1-3; Tr. 12–44. During her testimony, Petitioner stated that Mr. Bauer "was fine[]" on October 12, 2017, the date of his vaccination, and that he went about his normal activities that day. Tr. 14:24–15:3. Petitioner did not recall anything unusual about Mr. Bauer's health that day or remember him reporting any symptoms. Tr. 15:13–16.

Discussing the date of October 13, 2017, Petitioner noted that Mr. Bauer "was fine in the morning[]" before visiting their granddaughter's school in Wichita, Kansas for Grandparents' Day and that he first reported leg cramps during their trip to the zoo that afternoon. Tr. 15:12–25. She testified that Mr. Bauer reported cramps and pain beginning around 3:00 to 4:00 PM. Tr. 18:8–14. Petitioner did not recall Mr. Bauer ever reporting similar leg cramping before this date. Tr. 18:17–21. Petitioner recalled that Mr. Bauer asked to leave the zoo after experiencing leg cramps. Pet'r's Ex. 1 ¶ 6. Petitioner recalled helping Mr. Bauer into the car before she drove them to their daughter's house. Tr. 19:6–13, 23–24. At their daughter's house, Mr. Bauer "just sat around because he had what he described as cramps in his legs." Pet'r's Ex. 1 ¶ 7. Petitioner stated that they tried to massage Mr. Bauer's legs and that the cramps did not bother him as much as long as he stayed seated. *Id.* They went to a restaurant that evening and, after dinner, Mr. Bauer quickly exited the restaurant because he did not "know if [his] legs would make it." Tr. 19:13–20.

Petitioner recalled that on October 14, 2017, Mr. Bauer reported continuing cramps in his legs after he woke up. Pet'r's Ex. 1 ¶ 8. Petitioner and Mr. Bauer returned to their home in Burlington, Kansas that day. *Id.* Petitioner stated that Mr. Bauer did not help load the car because "he was just not moving very well." Tr. 21:6–10. She noted that Mr. Bauer "kind of crawled up the stairs, scooted up their stairs[]" when they were leaving their daughter's house. Tr. 21:3–4. When they arrived home, Mr. Bauer helped unload the car before resting in a recliner for the rest of the day. Tr. 21:24–22:3.

Mr. Bauer still reported cramps in his legs on October 15, 2017, and Petitioner and Mr. Bauer tried using heating pads to relieve the cramps. *Id.* ¶ 9. Mr. Bauer continued to "just pretty

[46] Bradycardia is "slowness of the heartbeat." *Dorland's* at 241.
[47] Cardiopulmonary resuscitation is "the artificial substitution of heart and lung action . . . ." *Dorland's* at 1604.

much s[it] in the chair[]" on October 15, 2017. Tr. 22:25–23:4. Petitioner noted that "[i]f he got up, he was holding onto the furniture to walk to get a drink or go to the bathroom." Tr. 23:4–6.

Petitioner recalled that on Monday, October 16, 2017, Mr. Bauer made an appointment with Dr. Jarvis for October 17, 2017. Pet'r's Ex. 1 ¶ 10. Petitioner testified that on October 16, 2017, Mr. Bauer's symptoms continued, and he "was still walking gingerly[]" and "being careful when he moved around." Tr. 23:11–17, 26:16–18. Petitioner noted that her and Mr. Bauer's children had wanted him to go to the ER in Wichita before they returned home but that Mr. Bauer instead wanted to see Dr. Jarvis, his longtime PCP. Tr. 24:8–19.

She continued that on the morning of October 17, 2017, Mr. Bauer attended a work meeting in the morning. Pet'r's Ex. 1 ¶ 11. Petitioner recalled that Mr. Bauer "later told [her] that while at the meeting, he went to use the bathroom[]" and that "[w]hile in the bathroom, his legs went out from under him[,] and he ended up facedown on the floor." *Id.*

Petitioner noted that Mr. Bauer presented to Dr. Jarvis on the afternoon of October 17, 2017, and that Dr. Jarvis ordered an MRI and planned to arrange an EMG. *Id.* ¶ 12. Petitioner recalled taking Mr. Bauer to this appointment, and she noted that Mr. Bauer walked slowly from the car to the medical office and put his hand on her shoulder when walking. Tr. 28:10–14. Petitioner recounted the appointment with Dr. Jarvis during her testimony, and she did not remember Mr. Bauer telling Dr. Jarvis about his recent flu vaccination. Tr. 28:22–29:24.

Petitioner recalled that Mr. Bauer had his MRI on October 18, 2017, and "then he went home for the rest of the day because of the problems with his legs." Pet'r's Ex. 1 ¶ 13. Petitioner discussed taking Mr. Bauer to his MRI, and she noted that he was walking "[m]uch slower[]" than usual. Tr. 31:3–32:4. She noted that Mr. Bauer "told [her] he still had a lot of cramping in his legs." Pet'r's Ex. 1 ¶ 13. Petitioner stated that Mr. Bauer worked from home on October 19, 2017. *Id.* ¶ 14. Petitioner "could tell that he was having trouble navigating his way into [their] bathroom[]" when he "got up after midnight on October 19, 2017[,] to use the bathroom." *Id.*

On October 20, 2017, Petitioner took Mr. Bauer to his office, and she "had to help him get into his office because of the problem he was having with his legs." *Id.* ¶ 15. She recalled that Mr. Bauer called her at around 10:00 AM and reported that "he was extremely tired." *Id.* ¶ 16. Petitioner "went to his office to pick him up[,] and he could not stand up." *Id.* Mr. Bauer showed her his "horrible" handwriting from that morning. Tr. 34:2–6. Petitioner recalled that Mr. Bauer "did[not] have the best [handwriting] anyway, but this was really bad." Tr. 34:6–7. Petitioner wanted to take Mr. Bauer to the hospital, but he wanted to go home to rest and wait for his MRI results. Tr. 34:15–19. They obtained a wheelchair for Mr. Bauer with the assistance of the Coffey County Health Department. Pet'r's Ex. 1 ¶ 16. Petitioner recalled that by the time they obtained the wheelchair, Mr. Bauer "said that his hands were not working[,] and he had no strength in his arms." *Id.* Petitioner then took Mr. Bauer home to rest. *Id.* After Petitioner and Mr. Bauer arrived home, Mr. Bauer told Petitioner that she should return to work. Tr. 34:20–35:1.

When Petitioner was on her way home from work for lunch at around 11:30 AM, she received a call from her and Mr. Bauer's son, who reported that Mr. Bauer was on the floor. Tr. 35:2–4. Petitioner recalled arriving home and finding Mr. Bauer on the living room floor. Tr. 35:5–

6. She stated that Mr. Bauer "could not get up[,]" and he was unable "to use his legs or his arms." Pet'r's Ex. 1 ¶ 17. When Mr. Bauer was unable to get up, Petitioner received help from a neighbor, who supplied a wheelchair and helped Mr. Bauer into it. Tr. 35:6–15. When Petitioner and the neighbor tried to help Mr. Bauer to the bathroom, Mr. Bauer reported difficulty breathing. Tr. 35:15–17. Petitioner testified that another neighbor arrived to help, and they attempted to get Mr. Bauer into the car to go to the hospital. Tr. 35:18–23. However, Petitioner called an ambulance when they were unable to get Mr. Bauer into the car. Tr. 35:24–36:1. She stated that Mr. Bauer "had no strength in his arms or his legs[]" and that he could not assist at all with getting himself into the car. Tr. 35:23–24.

Petitioner recalled speaking with Dr. Jarvis in the ER while awaiting a helicopter to transport Mr. Bauer to another facility. Pet'r's Ex. 1 ¶ 18. Petitioner stated that "Dr. Jarvis explained that [Mr. Bauer] had received a flu shot and had [GBS]." *Id.* She continued that Dr. Jarvis explained that the other facility would "wash his blood" and that Mr. Bauer would "have a lengthy recovery." *Id.* Mr. Bauer passed away while waiting for the helicopter, and Petitioner noted that no autopsy was performed. *Id.* ¶¶ 18–19.

Petitioner discussed her conversation with Dr. Jarvis after Mr. Bauer passed away. Tr. 37:2–12. Petitioner stated that Dr. Jarvis explained "[t]hat the flu shot can cause this [GBS], and it slowly paralyzes and shuts everything off as it comes up your body. His potassium had went sky-high. He had never, ever had high potassium. And then everything just shuts down." Tr. 37:8–12.

Petitioner did not recall Mr. Bauer having an illness, such as a virus, between August and October of 2017. Tr. 37:22–38:1. She testified that Mr. Bauer began taking spironolactone sometime around November or December of 2016. Tr. 38:2–8. Petitioner did not know whether Mr. Bauer had any kidney problems or low potassium levels prior to the vaccination. Tr. 38:22–39:4.

When asked about the rate of progression of Mr. Bauer's symptoms between October 12, 2017 and October 20, 2017, Petitioner stated that the symptoms "started slowly, and then they got real dramatic the last few days." Tr. 40:3–7. She did not recall Mr. Bauer experiencing leg weakness, leg pain, falling, or difficulty walking or going up and down stairs before his vaccination. Tr. 40:11–25. She also did not recall him having arm tingling or weakness, fatigue, or difficulty driving before the vaccination. Tr. 41:1–11.

2. Ms. Jennifer Toth

Ms. Jennifer Toth, Petitioner and Mr. Bauer's daughter, testified during the entitlement hearing. Tr. 47–62. Ms. Toth stated that in 2016 and 2017, she saw her father two to three times per month and spoke with him on the phone "all the time." Tr. 48:22–49:4. She testified that Mr. Bauer was active with his grandchildren before October 12, 2017, and had recently helped build and carry a bed frame for Ms. Toth and helped finish Ms. Toth's basement. Tr. 49:5–13. She described him as "super active," and she did not recall him complaining about difficulties with his arms and legs or fatigue during the activities she described. Tr. 49:13–22.

Ms. Toth recalled the trip to the zoo on October 13, 2017, and she stated that Mr. Bauer indicated his legs "were[not] feeling right[]" when they were looking at an elephant exhibit. Tr. 50:9–14. She recalled that they then got water for Mr. Bauer and sat on a bench. Tr. 50:16–17. Ms. Toth testified that Mr. Bauer said that "he could[not] feel his legs, that they just were[not] feeling right[,]" and that "[t]hey were kind of tingly, not muscle crampy." Tr. 51:18–20. Ms. Toth continued that after sitting for twenty minutes, they decided to take Mr. Bauer home. Tr. 51:23–52:4. Ms. Toth stated that they believed Mr. Bauer was dehydrated and that Mr. Bauer required assistance walking out of the zoo. Tr. 52:5–14. Ms. Toth recalled helping Mr. Bauer up the stairs when they arrived to her house and giving him water and pickle juice for possible dehydration. Tr. 52:8–19. Like Petitioner, Ms. Toth recalled Mr. Bauer quickly leaving the restaurant later that evening. Tr. 54:7–12. She testified that this behavior "was super unlike him." Tr. 54:11–12.

Discussing the events of October 14, 2017, Ms. Toth testified that Petitioner and Mr. Bauer decided to leave due to not feeling well even though they had originally intended to stay at Ms. Toth's house for the entire weekend. Tr. 55:17–20. Ms. Toth recalled telling Mr. Bauer that he should go to the doctor on Monday. Tr. 56:10–12. Ms. Toth stated that she spoke to Mr. Bauer on October 20, 2017, at around 11:30 AM and that Mr. Bauer stated that he was waiting for Petitioner to arrive home for help going to the bathroom. Tr. 57:2–8. Ms. Toth also recalled that Mr. Bauer reported experiencing numbness and tingling in his arms. Tr. 57:8–10. She stated that she spoke to Petitioner on the phone after Mr. Bauer was taken to the hospital via ambulance. Tr. 57:24–58:1. Ms. Toth stated that she "got the phone call [from Petitioner] that he had [GBS] and that they were going to 'life-wash' him to Topeka, they were going to wash his blood, and it was going to be about three to five years before he was ever back to normal, but this was the first step." Tr. 58:5–10, 59:8–16. She stated that she and her brother began driving to Topeka before receiving the news that Mr. Bauer passed away. Tr. 58:10–17.

III. Experts

A. Expert Review

1. Petitioner's Expert, Christopher Jarvis, M.D.

Dr. Jarvis submitted an affidavit and two expert reports, and he testified at the entitlement hearing. Pet'r's Ex. 9, ECF No. 1-11; Pet'r's Ex. 13, ECF No. 18-1; Pet'r's Ex. 14, ECF No. 24-2; Tr. 64–169. He earned his medical degree from the University of Kansas in 1996. Pet'r's Ex. 13 at 1. He then completed an internship and residency at Truman Medical Center in Kansas City, Missouri. *Id.* Dr. Jarvis is board-certified by the American Board of Family Medicine, and he is a member of the American Academy of Physicians. *Id.* He is licensed to practice medicine in Missouri and Kansas. *Id.* Dr. Jarvis testified that he has practiced medicine in Burlington, Kansas since 1999 and that he had been Mr. Bauer's PCP since approximately November of 2003. Tr. 64:4–17. Dr. Jarvis was not proffered as an expert before or during his entitlement hearing testimony. Following the conclusion of all testimony, Petitioner's counsel indicated that Mr. Jarvis testified as a treating physician and as an expert on GBS, acute kidney injury, and chronic kidney injury in his capacity as a family medicine/primary care physician. *See* Tr. 265.

2. Respondent's Expert, Brian Callaghan, M.D., M.S.

Dr. Callaghan submitted two expert reports and testified during the entitlement hearing. Resp't's Ex. A, ECF No. 23-1; Resp't's Ex. E, ECF No. 26-1; Tr. 171-263. He received his medical degree from the University of Pennsylvania in 2004. Resp't's Ex. B at 1. He completed an internship in preliminary medicine and a residency in neurology at the University of Pennsylvania between 2004 and 2008. *Id.* Following his residency, he completed a neuromuscular fellowship at the University of Michigan Health System in 2009. *Id.* He is board-certified by the American Board of Psychiatry and Neurology and by the American Board of Electrodiagnostic Medicine. *Id.* Dr. Callaghan joined the faculty of the Department of Neurology at the University of Michigan Health System in 2009, and he became an associate professor in 2018. *Id.* He has been a staff physician in the VA Ann Arbor Health System's Department of Neurology since 2015, and he has also served as the director of the ALS Clinic at the University of Michigan Health System. *Id.* Dr. Callaghan's research interests include "[t]he effects of the metabolic syndrome on the development of neuropathy[, t]he evaluation of peripheral neuropathy[, and e]fficient diagnostic testing in common neurologic disorders." *Id.* at 2. He has received multiple research grants and has served as an editor and reviewer for numerous journals. *Id.* at 2–4. He is an author of more than eighty peer-reviewed journal articles and publications and has also authored numerous book chapters and abstracts. *Id.* at 10–17. He is the "vice chair of education for the Peripheral Nerve Society, which is the same international society which studies GBS and other inflammatory neuropathies." Tr. 173:15–18. In addition, he serves on the International Diabetes Neuropathy Consortium Board and is the vice chair of the Health Services Research committee at the American Academy of Neurology. Tr. 173:18–22.

Dr. Callaghan is "a neuromuscular specialist with a primary interest in patients with neuropathy such as [GBS]." Resp't's Ex. A at 1. During the hearing, he testified that he treats patients "in both neuromuscular cases as well as general neurology." Tr. 172:20–22. He estimated that twenty percent of his time is spent in clinical practice. Tr. 173:5. Dr. Callaghan stated that he is "a neuromuscular expert" and that his "particular interest . . . is on neuropathy, including things like GBS." Tr. 173:15–18. Describing his experience treating GBS mimics, Dr. Callaghan testified that such mimics are "common things that we have to think about all the time." Tr. 173:24–25. He continued that, as a neurologist, he "see[s] quite a few cases of that[]" and has "to tease them out from the cases that do[not] have it, which is equally as common as GBS." Tr. 174:1–5. He noted that he treats patients who do not have GBS but who present with GBS-like symptoms. Tr. 174:12. During the hearing, Dr. Callaghan was admitted as an expert in neurology and neuromuscular neurology. Tr. 176:3–8.

3. Respondent's Expert, Derek Fine, M.D.

Dr. Fine received his medical degree from Johns Hopkins University School of Medicine in 1994, and he completed his internship and residency at Johns Hopkins Hospital between 1994 and 1997. Respt't's Ex. H at 1, ECF No. 55-2. Between 1997 and 1999, Dr. Fine completed a postdoctoral fellowship in nephrology, also at Johns Hopkins. *Id.* He joined the Johns Hopkins University School of Medicine faculty in 1999, and he has been a full professor since 2021. *Id.* at 1–2. He has been the Clinical Director of the medical school's Division of Nephrology since 2018 and the Medical Director of the Johns Hopkins Hospital/Fresenius Medical Care, Caroline Street

Outpatient Dialysis Clinic since 2016. *Id.* at 1. He is an author of numerous publications and has served as an editor for various journals. *See id.* at 2–11. He is board-certified in nephrology by the American Board of Internal Medicine, and his clinical work involves "frequent[] care for patients . . . who develop hyperkalemia in the context of compromised kidney function." Resp't's Ex. H at 13; Resp't's Ex. G at 1, ECF No. 55-1.

B. Expert Reports and Testimony

1. Petitioner's Expert, Dr. Jarvis

In his affidavit,[48] executed on July 29, 2022, Dr. Jarvis stated that he listed "possible GBS]" on Mr. Bauer's death certificate "because [they] did not do a tap to determine whether [Mr.] Bauer's cerebral spinal fluid protein levels were elevated." Pet'r's Ex. 9 ¶ 4. Dr. Jarvis stated that it is his "opinion that [Mr.] Bauer sustained an illness, i.e. [GBS], in association with the flu vaccine." *Id.* ¶ 5. Dr. Jarvis continued that he believed GBS contributed to Mr. Bauer's death and that his opinion is that Mr. Bauer would not have died in October of 2017 but-for the vaccination. *Id.* ¶¶ 6–7.

In his first expert report, Dr. Jarvis stated that he reviewed the Vaccine Injury Table's requirements to establish a GBS Table injury. Pet'r's Ex. 13 at 1. Dr. Jarvis asserted that Mr. Bauer suffered from GBS within the Table's time frame, three to forty-two days post vaccination. *Id.* Dr. Jarvis opined that "it is more probably true than not true that," between his October 12, 2017 vaccination and October 20, 2017 death, Mr. Bauer experienced "a. sensory abnormalities and/or autonomic dysfunction; b. symmetric, flaccid, bilateral motor limb weakness; and c. decreased or absent deep tendon reflexes in his weak limbs." *Id.* at 2. Dr. Jarvis acknowledged that "[n]o electrophysiologic studies were performed on" Mr. Bauer post vaccination, but Dr. Jarvis opined that Mr. Bauer "sustained a Table injury, to wit, [GBS] and death." *Id.*

In his second expert report, Dr. Jarvis averred that "[s]evere [GBS] can cause acute rental failure[]" and that "[k]idney failure or renal function deterioration is the most common cause of hyperkalemia." Pet'r's Ex. 14 at 1. To support a relationship between GBS and acute renal failure, Dr. Jarvis cited a study by Khajehdehi et al.[49] *Id.* (citing Pet'r's Ex. 15, ECF No. 44-1). In this study, thirty patients with GBS were compared with thirty controls. Pet'r's Ex. 15 at 1. Seven of the thirty GBS patients developed acute renal failure, and six of those seven patients "had dysautonomia[50] and became oliguric[51] while being in a hypotensive[52] state." *Id.* Acute renal

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[48] Although this is an affidavit rather than an expert report, I am discussing it along with Dr. Jarvis's expert reports and testimony because it includes his opinions.

[49] Parviz Khajehdehi et al., *Acute Renal Failure Due to Severe Landry-Guillain-Barré Syndrome*, 13 NEPHROL DIAL TRANSPLANT 2388 (1998).

[50] Dysautonomia is "malfunction of the autonomic nervous system[,]" which is "the portion of the nervous system concerned with regulation of the activity of cardiac muscle, smooth muscle, and glandular epithelium; usually restricted to the two visceral efferent peripheral components, the sympathetic nervous system, and the parasympathetic nervous system." *Dorland's* at 569, 1829.

[51] Oliguria is "diminished urine production and excretion as compared with fluid intake." *Dorland's* at 1300.

[52] Hypotension is "abnormally low blood pressure." *Dorland's* at 894.

failure occurred more often in patients with GBS than in the controls. *Id.* at 3. The authors concluded that "acute renal failure can occur commonly in cases with severe [GBS] particularly in those with dysautonomia, causing high mortality." *Id.* at 1. The study only included patients with normal renal function tests at initial presentation. *Id.* at 2. Khajehdehi et al. proposed that "ischaemic[53] damage to tubular cells during the hypotensive crises could be the underlying mechanism in [their] cases with" GBS and acute renal failure. *Id.* at 3.

Dr. Jarvis opined that Mr. Bauer's "October 20, 2017 hyperkalemia was caused by acute kidney failure injury related to or associated with his" GBS. Pet'r's Ex. 14 at 1. Dr. Jarvis asserted that GBS and hyperkalemia are not mutually exclusive. *Id.* Citing Mr. Bauer's medical records, Dr. Jarvis noted that Mr. Bauer began taking spironolactone approximately ten months before his death. *Id.* Dr. Jarvis asserted that "[i]f [Mr. Bauer's] hyperkalemia was caused by spironolactone, it would not have taken ten [10] months for the symptoms of hyperkalemia to develop." *Id.* at 2. Dr. Jarvis further averred that "[s]pironolactone is well-tolerated in patients with early-stage [chronic kidney disease ("CKD")] (i.e. GFR 30 to 89)." *Id.* Dr. Jarvis cited a study by Edwards et al.[54] *Id.* (citing Pet'r's Ex. 16 ECF No. 44-2). Edwards et al. found that less than one percent of study participants with early-stage CKD who were treated with spironolactone experienced serious hyperkalemia. Pet'r's Ex. 16 at 5. They noted that "[c]hanges in potassium, eGFR and systolic blood pressure were most apparent in the first month of treatment . . . ." *Id.* Edwards et al. concluded that their study "provide[d] support for the safety and tolerability of low dose spironolactone with concurrent ACE inhibitors or [angiotensin receptor blocker[55]] treatment in selected patients with non-diabetic early stage CKD." *Id.* They purposely excluded patients with a history of diabetes and Stage 4 or 5 CKD from their study. *Id.*

Dr. Jarvis stated that Mr. Bauer's "extremity weakness slowly progressed" for eight days following vaccination and that Mr. Bauer's extremity weakness began post vaccination. Pet'r's Ex. 14 at 1. Dr. Jarvis denied that Mr. Bauer had GBS symptoms before his vaccination. *Id.* Dr. Jarvis acknowledged that Mr. Bauer experienced fourth- and fifth-digit numbness during a cardiac follow-up visit with Dr. Jarvis on July 17, 2017, and leg cramps on June 21, 2017, when Mr. Bauer was evaluated for sleep apnea. *Id.* Dr. Jarvis stated that these symptoms were likely related to Mr. Bauer's "preexisting ulnar[56] mononeuropathy, olecranon bursitis[,[57]] and/or perineal tendonitis."[58] *Id.* Dr. Jarvis asserted that "[t]hose preexisting conditions were incidental findings and not the presenting complaints related to any evaluation by any healthcare provider." *Id.* He

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[53] Ischemia is "deficiency of blood in a part, usually due to functional constriction or actual obstruction of a blood vessel." *Dorland's* at 949.

[54] Nicola C. Edwards et al., *The Safety and Tolerability of Spironolactone in Patients with Mild to Moderate Chronic Kidney Disease*, 73(3) BR J. CLIN PHARMACOL 447 (2011).

[55] An angiotensin receptor blocker or antagonist is "any of a class of antihypertensive agents that block the vasoconstrictor and aldosterone-secreting effects of angiotensin II by competitive binding with angiotensin receptors." *Dorland's* at 96.

[56] Ulnar "pertain[s] to the ulna or to the medial aspect of the forearm as compared with the lateral (radial) aspect." *Dorland's* at 1968.

[57] Olecranon bursitis is "inflammation and enlargement of the bursa over the olecranon, caused by resting the weight of the body of the elbow." *Dorland's* at 260.

[58] Tendonitis, or tendinitis, is "inflammation of tendons and of tendon-muscle attachments." *Dorland's* at 1852.

continued that "[a]n isolated report of finger numbness and a separate isolated report of leg cramps does not support a diagnosis of pre-flu vaccine hyperkalemia." *Id.*

During his testimony, Dr. Jarvis estimated that he saw Mr. Bauer two or three times between December of 2016 and October of 2017. Tr. 119:25. Dr. Jarvis recalled evaluating Mr. Bauer on October 17, 2017. Tr. 66:21. Dr. Jarvis stated that Mr. Bauer reported neuromuscular complaints, weakness in his legs, and tingling, and Dr. Jarvis "was trying to fit it together[] . . . so [he] ordered the EMG to evaluate [Mr. Bauer's] muscular function and then the MRI to look for structural issues." Tr. 66:23–67:1. When Dr. Jarvis learned Mr. Bauer had received a flu vaccine, this was "another piece of the puzzle, and that made sense." Tr. 67:3–5. Regarding whether Mr. Bauer had symptoms of hyperkalemia on October 17, 2017, Dr. Jarvis testified that the medical record from that date does not indicate gastrointestinal issues or cardiac symptoms that can occur with hyperkalemia. Tr. 74:22–75:2. However, Dr. Jarvis acknowledged that Mr. Bauer was "experiencing this tingling, and that could be associated with electrolyte issues." Tr. 75:2–4. Dr. Jarvis did not note any clinical signs of worsening kidney failure or acute kidney injury on October 17, 2017. Tr. 75:5–11. Based on the medical record, Dr. Jarvis testified that Mr. Bauer had symptoms of GBS, including extremity weakness and paresthesia, on October 17, 2017. Tr. 76:3–9. Dr. Jarvis stated that he did not diagnose Mr. Bauer with GBS on that date because GBS "was way down in the differential[]" and because Mr. Bauer had no recent respiratory or gastrointestinal infections. Tr. 76:10–16. Dr. Jarvis noted that he did not know about Mr. Bauer's flu vaccination at the time. Tr. 76:16–17.

Dr. Jarvis addressed the note from his October 17, 2017 appointment with Mr. Bauer indicating that Mr. Bauer's symptoms began a couple of weeks before that date. Tr. 79:3–5. Dr. Jarvis noted that he dictated this record, but "that is not as accurate a note as [he] would like it to be." Tr. 79:5–6. He clarified that he "used that 'couple weeks' as a general term because [he] did[not] have the exact date of his symptoms when [he] dictated the note at the end of the day." Tr. 79:7–9. Dr. Jarvis noted that he sees up to thirty patients per day and that "there could be an error." Tr. 79:9–11. Dr. Jarvis testified that he believed that the note indicating that Mr. Bauer's symptoms had been ongoing for a couple of weeks was inaccurate, but Dr. Jarvis could not recall when he became aware of this inaccuracy or whether he signed the medical record before Mr. Bauer presented to the ER. Tr. 133:23–134:14. Dr. Jarvis stated that Mr. Bauer did not mention the flu vaccination during the October 17, 2017 appointment and that he would have asked Mr. Bauer different questions if he knew about the vaccination at the time. Tr. 135:1–3.

Dr. Jarvis testified that when Mr. Bauer presented to the ER on October 20, 2017, "it was obvious [he] was in distress." Tr. 77:15–16. Dr. Jarvis observed that Mr. Bauer would need to be intubated, and Dr. Jarvis worked on transferring Mr. Bauer to a larger hospital with capacity to care for him. Tr. 77:16–19. Dr. Jarvis recalled that upon arrival to the ER, Mr. Bauer told him that he had recently received a flu vaccine. Tr. 77:20–22. Dr. Jarvis explained that it "all kind of fell together at that point with the weakness and the paresthesia in his legs and moving proximally." Tr. 77:22–24. Dr. Jarvis recalled contacting a hospitalist at a larger hospital to see if they had room in their intensive care unit. Tr. 77:25–78:2. Dr. Jarvis could not remember who he spoke with, but he testified that he "talked to them about [Mr. Bauer's] presentation, and they agreed with [Dr. Jarvis] it sounded like – looked like [GBS] clinically." Tr. 78:2–5. Dr. Jarvis recalled that after Mr. Bauer went into cardiac arrest, before they were able to transport him to another hospital, his

labs revealed acute renal failure and a high potassium level of over 10. Tr. 78:6–11. Dr. Jarvis identified Mr. Bauer's "ascending weakness from his lower extremities to his upper extremities, the weakness, the paresthesias, [and] respiratory issues[]" as symptoms of GBS that Mr. Bauer presented with on October 20, 2017. Tr. 78:14–19.

Dr. Jarvis believed that the October 20, 2017 ER note indicating that Mr. Bauer's leg weakness started on the first of the week was more reliable than the record from October 17, 2017, indicating that symptoms had been ongoing for weeks. Tr. 79:13–18. When asked why he believed the October 20, 2017 note to be more reliable, Dr. Jarvis testified that he "would state the sentinel moment was [Mr. Bauer] telling [Dr. Jarvis] when he had the flu shot, and then that kind of locked everything in." Tr. 79:19–24.

Dr. Jarvis recalled speaking with Petitioner after Mr. Bauer's passing. Tr. 80:23–25. He recalled telling Petitioner that he "had talked to the hospitalist, and [he] knew about the flu shot with these ascending symptoms and the respiratory failure." Tr. 81:8–10. Dr. Jarvis told Petitioner that he "knew about the flu shot with these ascending symptoms and the respiratory failure, and [he] told her that likely [Mr. Bauer] had died from [GBS.]" Tr. 81:8–11. Explaining his basis for his conclusion that Mr. Bauer likely died from GBS, Dr. Jarvis testified, "[i]t falls within the window – after getting the vaccine, it falls within the window of when [GBS] can start[,] and with the symptoms and everything it seemed to fit, in [Dr. Jarvis's] opinion." Tr. 81:13–19. When asked on cross-examination about whether Mr. Bauer's alleged GBS was caused by the flu vaccine, Dr. Jarvis stated, "[i]t falls within the time frame[] . . . so, yes." Tr. 113:13–15.

Dr. Jarvis acknowledged that Mr. Bauer had CKD prior to his October 12, 2017 flu vaccination. Tr. 90:10–12. Dr. Jarvis testified that Mr. Bauer "was a diabetic, and he had high blood pressure, cardiovascular disease, so they all go hand in hand." Tr. 90:17–19. Dr. Jarvis continued that Mr. Bauer's CKD "was[not] severe." Tr. 90:19. Dr. Jarvis acknowledged that Mr. Bauer's eGFR of 31 on May 17, 2017, indicated that he had Stage 3b CKD, according to the stages described by the National Kidney Foundation.[59] Tr. 138:10–13; Tr. 168:23–169:12. (citing Pet'r's Ex. 20, ECF No. 39-4). The National Kidney Foundation describes the extent of the kidney damage each stage of CKD indicates as well as the eGFRs associated with each stage. Pet'r's Ex. 20 at 2. Stage 3a CKD, characterized by an eGFR of 45 to 59, indicates "[m]ild to moderate loss of kidney function." *Id.* An eGFR of 30 to 44 indicates Stage 3b CKD, a "[m]oderate to severe loss of kidney function." *Id.* Stage 4, or "[s]evere loss of kidney function[,]" occurs when the eGFR is 15 to 29, and an eGFR under 15 indicates Stage 5 CKD, which is kidney failure. *Id.* The National Kidney Foundation states that "[l]ater stage CKD does cause symptoms[,]" which can include more or less frequent urination and muscle cramps as well as other symptoms. *Id.* at 1.

Dr. Jarvis testified that he had "plenty of patients that are in their thirties with their eGFR and doing just fine." Tr. 139:6–7. Dr. Jarvis noted that Mr. Bauer's "microalbumin creatinine ratio [on May 17, 2017,] was 15, which is normal" and that he had a normal microalbumin level of 10. Tr. 168:7–10. Dr. Jarvis explained that "when people have significant kidney disease, you can see significant protein loss . . . from their kidneys." Tr. 168:10–11. Dr. Jarvis opined that these findings indicated that Mr. Bauer had CKD "that looked pretty stable." Tr. 168:17–18.

[59] National Kidney Foundation, Diagnostic Tests & Procedures: Estimated Glomerular Filtration Rate (2022).

Dr. Jarvis testified that Mr. Bauer's CKD worsened after the flu vaccination at issue "[b]ased on the labs [taken] in the [ER]." Tr. 90:22–23. Dr. Jarvis called this lab work "concerning." Tr. 91:2–3. Dr. Jarvis also asserted that "[t]here[is] no doubt that [Mr. Bauer] passed away from hyperkalemia." Tr. 92:8–9. Dr. Jarvis described 10.8, the potassium level Mr. Bauer had when he presented to the ER on October 20, 2017, as "severe[]" and "not consistent with life." Tr. 141:15–19. He acknowledged that a potassium level of 10.8 could lead to sudden death. Tr. 141:23.

Addressing Mr. Bauer's medications, Dr. Jarvis testified that Mr. Bauer had begun spironolactone and potassium about ten months before his death. Tr. 84:14–15. Dr. Jarvis stated he believed that "if a patient is going to have problems with spironolactone and potassium, . . . that usually occurs within the first week or two to a month after starting it, and [Mr. Bauer] had no problems with those medications." Tr. 84:16–21. Dr. Jarvis continued that Mr. Bauer was following up with endocrinology and cardiology, and "he had had no problems with those medications that [Dr. Jarvis was] aware of." Tr. 84:21–24. Although Dr. Jarvis stated that Mr. Bauer's "labs had been okay[]" prior to his October 2017 symptoms, he acknowledged that he was unaware of how often Mr. Bauer's potassium levels and kidney function were measured by his endocrinologist and cardiologist. Tr. 103:8–11; Tr. 137:20–22. Dr. Jarvis testified that he did not observe clinical signs of hyperkalemia in Mr. Bauer between when he was prescribed potassium supplements in May of 2016 and when he received the flu vaccine at issue. Tr. 86:7–25.

Dr. Jarvis acknowledged that the tingling and neuromuscular issues Mr. Bauer reported on October 17, 2017, could be associated with hyperkalemia. Tr. 89:12–14. He later acknowledged that it was possible that Mr. Bauer's potassium levels were elevated prior to the flu vaccination at issue. Tr. 166:10. When asked whether there would be any reason for Dr. Jarvis to know Mr. Bauer had elevated potassium levels if he was not exhibiting clinical signs, Dr. Jarvis stated, "that[is] possible. [Dr. Jarvis was] not going to argue with that." Tr. 166:15. However, Dr. Jarvis maintained that Mr. Bauer "went downhill after the vaccine and started demonstrating all those symptoms." Tr. 166:19–20. Dr. Jarvis asserted that he only had "the end result" and could only "work backwards from there and put the two together." Tr. 166:20–22. Dr. Jarvis stated that Mr. Bauer "was fine for ten months on spironolactone and potassium. He gets the flu vaccine, and then all hell breaks loose not too long after." Tr. 167:4–6.

Dr. Jarvis did not know whether Mr. Bauer was taking spironolactone in October of 2017. *See* Tr. 90:3–6. Dr. Jarvis noted that spironolactone may have contributed to Mr. Bauer's hyperkalemia and death "if he was still taking it while he had the [GBS] symptoms or developed . . . whatever was going on with autoimmune response with the flu vaccine, but the flu vaccine is the sentinel point in this whole thing." Tr. 103:20–25. While Dr. Jarvis was not aware whether Mr. Bauer was still taking his medications prior to his death, Dr. Jarvis acknowledged that "spironolactone[] . . . absolutely could have made things worse for him and also helped generate that elevated potassium." Tr. 112:3–14. However, Dr. Jarvis maintained that Mr. Bauer "had no problems with hyperkalemia prior to th[e] flu vaccine[]" and that, thus, "something occurred with the flu vaccine that led to his kidney injury, which led to his hyperkalemia." Tr. 111:13–16.

When asked if he had any reason to believe that Mr. Bauer was not taking spironolactone in October of 2017, Dr. Jarvis stated that he "took care of [Mr. Bauer] a long time, and he might not have been." Tr. 143:1–2. However, Dr. Jarvis testified that "where [Mr. Bauer] was at in 2017," Dr. Jarvis thought that "he was doing a really good job of managing his medical conditions[]" and had gotten his diabetes under control. Tr. 143:3–7. Dr. Jarvis stated that Mr. Bauer "was doing everything right at that point[] . . . to make sure he was the healthiest he could be." Tr. 143:9–12.

Discussing his opinion that Mr. Bauer's CKD was significantly aggravated by his October 12, 2017 flu vaccination, Dr. Jarvis testified that Mr. Bauer "had an acute kidney injury," caused by "a possible couple [sic] mechanisms." Tr. 91:6–7. Dr. Jarvis continued that "the flu shot, when given, produces an antibody to an antigen, and there are case studies [which talk] about the antibody produced to the flu vaccine, the antigen, will cross-react with the glomeruli." Tr. 91:7–11. Dr. Jarvis stated that this would "impair filtration and cause kidney injury." Tr. 91:11–12. Describing another potential mechanism, Dr. Jarvis stated that GBS can cause muscle injury, and "elevation of the muscle enzymes and myoglobin."[60] Tr. 91:13–15. He testified that "that can lead to renal failure and acute kidney injury and shut down the kidneys, which then all that would lead to hyperkalemia" and death. Tr. 91:15–17. Dr. Jarvis recalled that he had "seen a case of [GBS] with acute renal failure and injury." Tr. 92:4–6. He recalled that when he was doing a critical care rotation in medical school, he observed a patient with GBS who almost passed away after experiencing rhabdomyolysis[61] that "shut down his kidneys." Tr. 91:18–92:2.

Dr. Jarvis testified that there is medical literature supporting a connection between the flu vaccine and GBS. Tr. 95:21. He reiterated his opinion that Mr. Bauer's flu vaccination led to "his renal failure, acute kidney injury, whether it was myoglobin built in with his muscle injury or if it was due to the antibodies[] . . . attacking his glomeruli and shutting down his kidneys, [Dr. Jarvis did not] know that mechanism." Tr. 96:23–97:4. However, Dr. Jarvis maintained that the mechanism "has to be something in that area." Tr. 97:3–5. Dr. Jarvis noted that Mr. Bauer had muscle pain and "possibly had some elevation of his myoglobin from muscle breakdown, and that could have impacted his kidney function and shut him down, led to acute kidney injury." Tr. 97:16–19. Dr. Jarvis stated that medical literature contains "documented cases" connecting GBS and acute kidney injury. Tr. 97:23–24.

Dr. Jarvis indicated that an acute kidney injury could worsen CKD and that "any medications [Mr. Bauer] was taking could impact that also once he ha[d] the acute kidney injury . . . from the vaccine." Tr. 99:8–10. Dr. Jarvis opined that Mr. Bauer suffered an acute kidney injury which then significantly aggravated his CKD and led to acute renal failure. Tr. 99:18. Describing how an acute kidney injury could worsen CKD, Dr. Jarvis stated that "with the cross-reactivity, with the antibodies, the . . . autoimmune response, attacking his kidneys, shuts them down, and then you have acute kidney injury worsening his chronic kidney disease." Tr. 99:24–100:4.

[60] Myoglobin is "the oxygen-transporting pigment of muscle, a type of hemoprotein resembling a single subunit of hemoglobin, composed of one globin polypeptide chain and one heme group (containing one iron atom)." *Dorland's* at 1206.

[61] Rhabdomyolysis is "disintegration or dissolution of muscle, associated with excretion of myoglobin in the urine." *Dorland's* at 1611.

Dr. Jarvis stated that he identified three potential mechanisms in this case. Tr. 104:8. These mechanisms include (1) "[p]ossibl[e] elevation of myoglobin[,] . . . which can occur with [GBS], which could impact his filtration in his kidney and shut it down," (2) "the antibody from cross-reacting with the myelin sheath,"[62] and (3) "an antibody from the flu vaccine injuring the kidney." Tr. 104:10–15. Dr. Jarvis stated that he could not tell which of these mechanisms occurred but that "something happened at that point in time." Tr. 104:16–17. Dr. Jarvis opined that "it fits with [GBS] with his neuromuscular symptoms that progressed, and he [got] weaker and weaker at that point." Tr. 104:17–20. When asked which of the mechanisms was the most probable in this case, Dr. Jarvis testified that it is more probable that Mr. Bauer had GBS. Tr. 104:24. When asked to explain how a flu vaccination caused Mr. Bauer to suffer from GBS, Dr. Jarvis stated that "an immune response to the vaccine was generated, and then he had, unfortunately, an autoimmune response where the antibody attacked his own body." Tr. 95:14–18. He stated that he should have listed GBS, along with acute kidney injury, hyperkalemia, and cardiopulmonary arrest, instead of "possible" GBS on Mr. Bauer's death certificate. Tr. 105:6–9. He stated that he should have listed "the freaking vaccine" as a contributing factor "if [he] would have filled it out completely correct." Tr. 105:9–14.

On cross-examination, Dr. Jarvis acknowledged that two of the three mechanisms he presented required Mr. Bauer to have developed GBS. Tr. 125:3. However, Dr. Jarvis then stated that it is not "necessarily the case" that the mechanisms would not apply in this case but-for GBS "because it[is] an autoimmune response, and if the flu vaccine generated an antibody that impacted his glomeruli and his filtration system and he went into renal failure that way, . . . the vaccine is still a possibility." Tr. 125:8–13. Dr. Jarvis stated that Mr. Bauer could have developed glomerulonephritis[63] post vaccination, but he could only rely on clinical signs and symptoms because there were no available kidney tissue samples. Tr. 125:16–22. I asked Dr. Jarvis to clarify what biological mechanism he thought most likely occurred in this case, and he opined that Mr. Bauer's symptoms were most likely caused by GBS. Tr. 165:15–17.

When I asked Dr. Jarvis how many patients he had seen who were ultimately diagnosed with GBS by a neurologist, Dr. Jarvis acknowledged that he had only seen one case of GBS in his career, "[a] long time ago." Tr. 153:24–154:1. He stated that Mr. Bauer's case would be the second case of GBS he believes he has seen. Tr. 154:6. Dr. Jarvis acknowledged that Mr. Bauer was not seen by a neurologist, but he stated that this was because Mr. Bauer "did[not] have time." Tr. 154:11. Regarding his experience reviewing neurology literature, Dr. Jarvis stated that has "been looking at literature for years[]" because his daughter has a neuromuscular condition. Tr. 115:10–12.

When asked to describe the symptoms of hyperkalemia, Dr. Jarvis stated that he had "seen patients with nausea, vomiting, diarrhea, chest pain, heart palpitations, [and] irregular heartbeat." Tr. 136:15–17. He also noted that patients with hyperkalemia can have paresthesias and twitches. Tr. 136:17–18. Dr. Jarvis acknowledged that "the weakness and numbness that [Mr. Bauer] was experiencing could be associated with hyperkalemia." Tr. 136:23–24.

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[62] A myelin sheath is "the cylindrical covering on the axons of some neurons." *Dorland's* at 1673.
[63] Glomerulonephritis is "nephritis accompanied by inflammation of the capillary loops in the renal glomeruli." *Dorland's* at 777. Nephritis is "inflammation of the kidney." *Id.* at 1223.

Petitioner filed multiple case reports of hyperkalemia and hyperkalemic paralysis. She filed a case report by Muensterer[64] in which a patient with renal impairment, a potassium level of 9.2, and a creatinine level of 3.9 "presented to the [ER] with acute onset of flaccid motor paralysis from the neck downward[]"after taking potassium supplements for ten days. Pet'r's Ex. 18 at 1, ECF No. 39-2. The patient fully recovered once her potassium levels were corrected. *Id.* Muensterer noted that a "potassium imbalance associated with paralysis" may be caused by primary renal failure and medications including spironolactone. *Id.* He stated that "[h]yperkalemic paralysis should be considered in a patient with impaired renal function and motor weakness." *Id.* at 2. In another case report, Kimmons and Usery[65] discussed "a case of ascending muscle paralysis in a patient with [CKD] prescribed multiple potassium-elevating agents." Pet'r's Ex. 22 at 1, ECF No. 39-6. The patient presented to the ER after experiencing weakness and paresthesias in her upper and lower extremities, progressive weakness, and difficulty walking. *Id.* Her medical history included hypertension and diabetes, and her medications included spironolactone (25 mg twice per day), potassium supplements (20 mEq twice per day), carvedilol (25 mg twice per day), and lisinopril, an ACE inhibitor. *Id.* She presented with a high potassium level of over 8, a creatinine level of 3.5, and a BUN level of 114, and she was assessed with acute renal failure and hyperkalemia. *Id.* at 1–2. Her symptoms returned to baseline after her potassium levels were corrected. *Id.* at 2. A case report by Udezue and Harrold[66] involved a patient who presented with "progressive muscular paralysis, starting from the lower and reaching the upper limbs within a period of [five] days[]" and a potassium level of 9.3. Pet'r's Ex. 23 at 1, ECF No. 39-7. He had been taking spironolactone for two years at the time of presentation. *Id.*

Regarding whether hyperkalemia is a mimic of GBS, Dr. Jarvis testified that "there is literature that documents that." Tr. 142:11. I asked Dr. Jarvis whether he would have attributed Mr. Bauer's symptoms to hyperkalemia if Mr. Bauer had not had a recent flu vaccine, and Dr. Jarvis responded, "without the flu vaccine, hyperkalemia does mimic [GBS]." Tr. 158:9–10. However, Dr. Jarvis asserted that "we can[not] take the flu shot out of the question." Tr. 158:11. He did not discuss the Walling and Dickson[67] article filed by Petitioner, which states that to diagnose GBS, "other potential causes [of symptoms] must be excluded." Pet'r's Ex. 21 at 1, ECF No. 39-5.

Dr. Jarvis testified that he had seen "[p]robably a handful[]" of patients who experienced hyperkalemia as a direct cause of death. Tr. 146:17. He noted that "we see neuromuscular symptoms in" such cases. Tr. 146:22–23. He acknowledged that the course of hyperkalemia, whether it is progressive or acute, depends "on the patient and the medical problem." Tr. 147:2–3. Dr. Jarvis indicated that the onset of hyperkalemia can manifest in many different ways and depends on "the patient's ability . . . to compensate." Tr. 147:7–9. Dr. Jarvis recalled that he had "had patients . . . that were diabetics with kidney disease and[] . . . ready for dialysis that . . . had

[64] Oliver J. Muensterer, *Hyperkalemia Paralysis*, 32 AGE & AGEING 114 (2003).
[65] Lauren A. Kimmons & Justin B. Usery, *Acute Ascending Muscle Weakness Secondary to Medication-Induced Hyperkalemia*, CASE REPS. MEDICINE (2014).
[66] E.O. Udezue & B.P. Harrold, *Hyperkalemic Paralysis Due to Spironolactone*, 56 POSTGRADUATE MED. J. 254 (1980).
[67] Anne D. Walling & Gretchen Dickson, *Guillain-Barré Syndrome*, 87(3) AM. FAMILY PHYSICIAN 191 (2013).

potassium[levels] over eight, and . . . they[are] walking around [with [no] symptoms." Tr. 147:9–13.

When asked whether there is always a cause when a patient presents with acute hyperkalemia, Dr. Jarvis answered "yes[]" and stated that "[w]e can find a reason." Tr. 158:18–20. I asked Dr. Jarvis whether medical problems such as diabetes, CKD, and the other medical problems Mr. Bauer had could cause acute hyperkalemia, and Dr. Jarvis acknowledged that these medical issues "make him more susceptible[]" to hyperkalemia. Tr. 158:25. However, Dr. Jarvis maintained that, even in an individual susceptible to hyperkalemia, "there needs to be a trigger, in [Dr. Jarvis's] experience." Tr. 159:8–9. I asked Dr. Jarvis if he asks all of his patients with hyperkalemia about their vaccination history, and Dr. Jarvis noted that "this was an acute hyperkalemia, so it was a different presentation in the [ER], so [Dr. Jarvis] had to go with what [he] had and go backwards." Tr. 150:16–18. He explained that when he evaluates patients with hyperkalemia in his clinic, he evaluates their medications and medical problems to determine the problem. Tr. 150:19–22. He stated that "when [he] ha[s] somebody that has an acute severe hyperkalemia, we have got to look at everything at that point[]" to determine "what caused the injury." Tr. 150:24–151:2. He noted that he or a nurse will ask patients about their vaccination history if they present to the ER with acute hyperkalemia. Tr. 151:24–152:2. He stated that he does not ask every patient with hyperkalemia about their vaccination history, but he does not "see severe hyperkalemia that frequently." Tr. 152:14–17. Dr. Jarvis clarified that he does not ask about vaccination history if he knows what caused the hyperkalemia in a particular case. Tr. 153:3–4.

Although Dr. Jarvis accepted that Mr. Bauer presented with severe hyperkalemia, that he had risk factors for developing hyperkalemia, and that hyperkalemia can mimic GBS, he explained that "[h]is point is that [GBS] can progress and there can be cross-reactivity with the antibodies that are produced to the myelin sheath with [GBS] that causes the neuromuscular issues, and they can also cross-react with the glomeruli and the kidneys." Tr. 92:10–15. Dr. Jarvis stated that this is "the pathway that [he] see[s] that this occurred." Tr. 92:15–16. Although Dr. Jarvis acknowledged that Mr. Bauer had CKD, Dr. Jarvis maintained that Mr. Bauer "did not have acute renal failure or chronic severe renal failure where he needed dialysis or anything like that." Tr. 92:17–21. Dr. Jarvis continued that "it was[not] even time for a nephrologist yet." Tr. 92:21–22. Dr. Jarvis thus opined that "the sentinel point in this is the – or the inflection point . . . is the flu shot, and all of this cascaded to [Mr. Bauer] in the [ER] and passing away." Tr. 92:22–93:1.

Dr. Jarvis opined that "based on the timing, the flu vaccine did cause an injury to" Mr. Bauer. Tr. 93:19–20. Dr. Jarvis "believe[d] [Mr. Bauer] was experiencing symptoms of [GBS], which were progressing." Tr. 93:23–24. Dr. Jarvis was "not sure of which mechanism [] impacted his renal function, but something . . . impacted him and led to acute kidney injury and his hyperkalemia." Tr. 93:24–94:3. Dr. Jarvis opined that "[t]he cascading effect of the GBS definitely could[]" lead to hyperkalemia. Tr. 94:6–7. Discussing the possibility that "the antibody that[is] produced [by the flu vaccine] will cross-react with either the nerves or even in the glomeruli[,]" Dr. Jarvis stated that "we see autoimmune diseases all the time in medicine where the body produces an antibody to something and attacks an organ." Tr. 94:10–16. He opined that "it[is] very plausible, probable that[is] what happened[]" in this case. Tr. 94:18–19.

Addressing whether there is a logical sequence of cause and effect to support that GBS caused Mr. Bauer's acute kidney injury, Dr. Jarvis testified that he believes there is a logical sequence of cause and effect "with the timing of the vaccine and then his symptoms that led to progression of acute kidney injury, we can put together clinically in [Dr. Jarvis's] opinion." Tr. 98:10–13.

Although Dr. Jarvis opined that the flu vaccine could cause kidney injury without first causing GBS, Dr. Jarvis stated that he did not think kidney injury occurred without GBS in this case because of Mr. Bauer's neuromuscular complaints. Tr. 101:17–21. Dr. Jarvis did not "believe you would see [neuromuscular complaints] necessarily[]" in that case. Tr. 101:21–22. Dr. Jarvis clarified that the flu vaccine could have caused kidney injury in this case without intervening GBS, but he explained that he was opining based on "the clinical picture of what took place and lab work[]" because Mr. Bauer passed away in the ER. Tr. 102:9–14. Dr. Jarvis stated that "it seems a very probable event that [Mr. Bauer] had GBS, whether it was cross-reactivity, an autoimmune response with antibody that[is] produced to the myelin sheath, it also hit the kidneys, or if he had an antibody that was produced just impacting the glomeruli." Tr. 102:21–103:1.While he acknowledged that he was "not going to be able to give [] that information," he maintained that "clinically, [Mr. Bauer] had signs of [GBS] progressing to the point" Mr. Bauer presented to the ER with renal failure and hyperkalemia. Tr. 103:1–6.

Dr. Jarvis noted multiple times that Mr. Bauer was "perfectly fine prior to the flu vaccine[]" and experienced "a rapid decline[]" post vaccination. Tr. 103:14–16; Tr. 103:25–104:1. During my questioning, Dr. Jarvis acknowledged that he believed the flu vaccination was responsible for Mr. Bauer's decline because of the timing between the flu vaccination and weakness and the symptoms he experienced in relationship to the timing of the flu vaccination. Tr. 149:18–24. When asked to clarify what outside of the timing made him believe the flu vaccination was responsible for Mr. Bauer's symptoms, Dr. Jarvis answered, "[w]ell, and everything [Dr. Jarvis] saw that came with it." Tr. 150:3. When asked what his opinion would be if Mr. Bauer's symptoms manifested at a different time, Dr. Jarvis stated that if the symptoms started two weeks post flu vaccination, "[i]t still falls within the window." Tr. 150:7. He continued that "[i]f it[is] six months out," Dr. Jarvis would not think the symptoms were related to the vaccination, "but if it[is] still within four to six weeks of the vaccination, . . . that still can be in the time frame." Tr. 150:7–10.

Further describing the bases of his opinions in this case, Dr. Jarvis stated that Mr. Bauer "had [CKD], and[] . . . when he came into the [ER], his kidney function had worsened." Tr. 121:16–18. Dr. Jarvis continued:

> So what occurred between the time he had normal kidney function to the point where he[is] in the ER and he expire[d] from hyperkalemia and renal failure[?] [S]o then you have to try to put the pieces of the puzzle together, and to [Dr. Jarvis] it looked like GBS, worsening, and then [Dr. Jarvis] reviewed the literature many times looking for mechanisms that would make sense. [Dr. Jarvis] can[not] tell [] which mechanism occurred, but the sentinel event is the flu vaccine, and then something took place, and was it an autoimmune response from – you know, that[is] what it looks like, so the [GBS] and the antibody generated against – you know, during that pathway or is it the flu vaccine producing an antibody that attacks the

> glomeruli? . . . [T]here[are] a couple different theories, or did he have myoglobinuria[68] and, you know, knock out his kidneys with that[?] [Dr. Jarvis] . . . [he] just [knew] that [Mr. Bauer] . . . had no problems except for [CKD], flu vaccine, eight days later, and that[is] what [Dr. Jarvis was] working with.

Tr. 121:19–122:14.

2. Respondent's Expert, Dr. Callaghan

Dr. Callaghan asserted that "there is very little supporting evidence for a diagnosis of GBS in this case." Resp't's Ex. E at 1. He noted that "EMG/[nerve conduction study ("NCS")] studies and/or lumbar puncture[69] studies are the best tests available to establish a diagnosis of GBS, but neither were performed in this case." *Id.* He explained that these tests can help evaluate lots of different mimics[]" and that laboratory testing can identify "multiple electrolyte disorders that can cause GBS-like symptoms." Tr. 176:23–177:3. He opined that "hyperkalemia, rather than GBS, [is] by far the most likely diagnosis[]" and that "[t]his is especially true in the context of worsening kidney failure, potassium supplementation, and ongoing spironolactone treatment . . . since they can all contribute to hyperkalemia." Resp't's Ex. E at 1.

During the hearing, Dr. Callaghan described how he evaluates patients who present with GBS-like symptoms. He explained that when evaluating a patient presenting with "numbness and weakness in all four extremities, there[are] lots of things" to consider. Tr. 176:16–19. His evaluation includes an examination, which can help to distinguish between "problems in the brain or spinal cord versus problems in the peripheral nerves or muscles," and testing. Tr. 176:19–22.

Dr. Callaghan explained that "GBS is a polyneuropathy, mostly at the nerve root level but also more diffusely, that hurts the covering of the nerves[]" due to "an autoimmune attack on those nerves." Tr. 181:4–7. GBS "leads to full extremity weakness and numbness and sometimes respiratory failure as well." Tr. 181:7–9. He continued that signs of GBS on examination include "weakness, areflexia, or reduced reflexes[]" as well as sensory abnormalities. Tr. 181:12–14. Dr. Callaghan noted that the Brighton criteria, which include three levels, are the most widely used criteria to diagnose GBS. Tr. 181:18–20. These criteria include that "it has to be a monophasic prodrome that . . . takes less than a few weeks to get to the worst part of their disease course, and it has to have numbness and weakness in four limbs." Tr. 181:25–182:4. He continued, "[a]nd at the level three, the main thing . . . that applies to this case is the absence of an alternative cause." Tr. 182:6–9. Dr. Callaghan asserted that an alternative cause in this case is the high potassium level. Tr. 182:9–10. Although Dr. Callaghan noted that Mr. Bauer did not have a lumbar puncture or EMG, findings of high protein and demyelinating[70] neuropathy on these tests "would be what would be required for levels one or two on the Brighton criteria." Tr. 182:10–16. Regarding the third level of the Brighton criteria, Dr. Callaghan explained that it "is the lowest level[]" and that "not having a reason to have those symptoms[] . . . such as hyperkalemia would not allow [a

[68] Myoglobinuria is "the presence of myoglobin in the urine." *Dorland's* at 1206.
[69] Lumbar puncture is "the withdrawal of fluid from the subarachnoid space in the lumbar region." *Dorland's* at 1532.
[70] Demyelination is "destruction, removal, or loss of the myelin sheath of a nerve or nerves." *Dorland's* at 480.

patient] to qualify for a level [three]." Tr. 182:21–24. Dr. Callaghan stated that it is "important to have an exclusionary factor because not everybody that has numbness and weakness in all four limbs has GBS." Tr. 183:3–5. He stated that there is "a long list of other potential conditions." Tr. 183:5–6. These other conditions include infections, cancers, autoinflammatory disorders of the nerve roots, spinal cords problems, neuromuscular junction problems, and "several different electrolyte problems, such as high magnesium, low phosphate, high or low potassium, all of which can lead to a very similar presentation." Tr. 183:7–14. When asked how to tell whether a patient is presenting with GBS or with a GBS mimic, Dr. Callaghan explained that this would be evaluated through diagnostic tests, including lab tests looking for electrolyte disturbances, lumbar puncture, imaging of the brain and spinal cord, and EMG. Tr. 183:18–184:3. Dr. Callaghan testified that he has diagnosed at least fifty patients with GBS and was involved in treating patients with the condition. Tr. 211:3–6. He noted that about ninety percent of the patients he had diagnosed with GBS had lumbar punctures confirming diagnosis, but he acknowledged that some of these patients did not have lumbar punctures. Tr. 211:13–17.

Dr. Callaghan stated that "hyperkalemia is a well-known mimic of GBS" and that "[h]yperkalemia can lead to ascending weakness and cardiopulmonary arrest." Resp't's Ex. A at 1. He continued that symptoms of hyperkalemia include sensory symptoms and sometimes respiratory failure [] ." *Id.* He explained that "[h]yperkalemia can lead to dramatic fluctuations in weakness such as in this case (trouble with legs to paralysis in hours), whereas GBS leads to slow progression of weakness over days to weeks." Dr. Callaghan testified that he has experience treating patients with elevated potassium levels, and he noted that their treatment depends on their potassium level. Tr. 177:6–9. When asked to define hyperkalemia, Dr. Callaghan noted that potassium levels above 5.5 are abnormal, and levels of above 7 are "pretty severe hyperkalemia." Tr. 180:3–6. He agreed with Dr. Jarvis that patients with hyperkalemia can be asymptomatic, but Dr. Callaghan explained that the main symptoms occur as a patient's potassium levels become critically high. Tr. 180:7–11. Dr. Callaghan noted that as hyperkalemia progresses, "you reach a certain threshold, and then all of a sudden, your body can[not] tolerate it," leading to "a dramatic change[]" in symptoms. Tr. 257:20–23. Dr. Callaghan explained that these symptoms are "neuromuscular symptoms of weakness that can happen very suddenly, where there can be a sudden change in weakness which cannot be seen in GBS[]" as well as "changes in the heart," including "changes in [] EKG that leads to instability and, unfortunately, death." Tr. 180:12–17.

He explained that a sudden potassium increase is "usually not a big concern," but it can be life-threatening if it reaches a "very high" level such as 10.8, the potassium level Mr. Bauer had on October 20, 2017. Tr. 177:9–13. At such levels, hyperkalemia becomes "a life-threatening emergency, and so everything is about trying to correct that potassium level before it leads to sudden cardiac death." Tr. 177:13–16.

Dr. Callaghan explained that "high potassium levels can cause symptoms that are indistinguishable from GBS." Tr. 179:9–10. These symptoms can include "very severe weakness and numbness and respiratory failure," and hyperkalemia is "much more likely to lead to death because it can cause asystoli and other arrhythmias of the heart." Tr. 179:10–14. Dr. Callaghan testified that "the three most common causes of hyperkalemia" are supplementation, which causes iatrogenic[71] hyperkalemia; kidney failure; and medications." Tr. 180:20–24. Dr. Callaghan noted

[71] Iatrogenic is "resulting from the activity of physicians." *Dorland's* at 899.

that all three of these causes "apply in this case." Tr. 180:24. Dr. Callaghan noted that hyperkalemia is not associated with autoimmune diseases besides diabetes. Tr. 256:14.

Dr. Callaghan cited two case reports in which patients who took spironolactone or a similar medication were initially diagnosed with GBS before ultimately being assessed with hyperkalemia instead. A case report by Freeman and Fale[72] involved a patient with a history of diabetes and a one-week history of treatment with amiloride-hydrochlorothiazide[73] who presented "with a short history of progressive ascending muscular weakness[]" and experienced respiratory failure. Resp't's Ex. C at 1, ECF No. 23-3. He experienced "rapid recovery of muscle power after emergency treatment of the hyperkalemia." *Id.* at 1–2. This, in addition to normal cerebrospinal fluid, "confirmed that hyperkalemia was the cause of his paralysis." *Id.* at 2. In a case report by Evers et al.,[74] a patient presented with a one-week history of progressive limb paralysis and a potassium level of 8.8. Resp't's Ex. D at 1–2, ECF No. 23-4. He had a one-year history of mild CKD and a two-month history of taking 200 mg per day of spironolactone. *Id.* at 2. The patient's symptoms resolved after his potassium levels were lowered, and Evers et al. stated that his hyperkalemia "was most probably due to an additive effect of his mild chronic renal failure in conjunction with spironolactone medication." *Id.* Evers et al. noted that "[i]n most patients, chronic renal failure or diuretic intake, especially spironolactone, is the cause of secondary hyperkalemic paralysis." *Id.* at 4.

Discussing Mr. Bauer's medical history and medications, Dr. Callaghan noted that "Mr. Bauer had trouble with hypokalemia during [a hospital] admission [in December of 2016,] and his potassium supplementation was increased." Resp't's Ex. A at 2. Dr. Callaghan also noted that Mr. Bauer "was started on spironolactone, a diuretic that reduces potassium excretion by the kidney." *Id.* Dr. Callaghan also noted Mr. Bauer's pre-vaccination history of hand numbness, reported to Dr. Jarvis on July 17, 2017, and "leg cramps that would awaken him dating back to" June 21, 2017. *Id.* Dr. Callaghan noted that the medical records indicate that, as of March of 2017, Mr. Bauer was prescribed 25 mg of spironolactone twice per day. Tr. 186:14–15. Dr. Callaghan "notice[d] that there really [was] a lack of monitoring of [Mr. Bauer's] potassium in the entire ten months from when he was started on spironolactone to the time he came to the ER." Tr. 186:17–21. Dr. Callaghan testified that when testing on May 17, 2017, revealed that Mr. Bauer had Stage 3b CKD with an eGFR of 31, "the dose should [have been] changed to be much lower than 25 [mg] twice a day at this level." Tr. 187:17–18. In response to Dr. Jarvis's contention that spironolactone is well-tolerated in patients with early stage CKD, Dr. Callaghan noted Mr. Bauer no longer had early stage CKD in May of 2017 when his eGFR was 31 and that his CKD "was likely worse by October [of] 2017." Resp't's Ex. E at 2. Dr. Callaghan asserted that "[s]pironolactone is not well tolerated in patients with more advanced kidney failure, such as Mr. Bauer." *Id.* While Dr. Callaghan noted that Mr. Bauer's creatinine was elevated, he opined that "the eGFR is really the important number because it really allows" doctors to evaluate the state of the kidneys. Tr. 187:21–188:1. Responding to Dr. Jarvis, Dr. Callaghan asserted that Mr. Bauer's normal albumin level

[72] S.J. Freeman & A.D. Fale, *Muscular Paralysis and Ventilatory Failure Caused by Hyperkalemia*, 70 BRIT. J. ANAESTHESIA 226 (1993).
[73] Amiloride hydrochloride is a potassium-sparing diuretic, and hydrochlorothiazide is a thiazide diuretic. *Dorland's* at 60, 867.
[74] Stefan Evers et al., *Secondary Hyperkalemia Paralysis*, 64 J. NEUROL NEUROSURG PSYCHIATRY 249 (1998).

was "not really of relevance." Tr. 188:9–10. Dr. Callaghan explained that "micro or macroalbinuria [sic] predicts future kidney failure[]" but that Mr. Bauer "already ha[d] very significant kidney dysfunction." Tr. 188:10–13. Dr. Callaghan stated that he had done research on diabetic complications that revealed a disconnect between albumin levels of eGFR, and he maintained that GFR is "the important parameter." Tr. 188:18–21.

Dr. Callaghan cited Mr. Bauer's weakness in May of 2017 as something that "definitely stands out" because weakness "can be associated with both GBS and its mimics." Tr. 189:18–22. Discussing the leg cramps Mr. Bauer reported when he underwent sleep apnea testing in June of 2017, Dr. Callaghan noted that "because leg cramps are such a prominent symptom that he had right before his earlier [sic] hospitalization in October, it[is] worthy to note that[] . . . some of these symptoms date back to June of 2017." Tr. 190:4–15. Dr. Callaghan stated that he reviewed Petitioner's affidavit prior to the hearing, and he noted "just how abruptly [Mr. Bauer's] symptoms changed." Tr. 191:25–192:2. Dr. Callaghan noted that Mr. Bauer went to work on October 20, 2017, and that while "he had problems the previous days, . . . there was kind of a fluctuating course." Tr. 192:2–5. He noted that Mr. Bauer was still able to walk, sometimes with assistance, during these days and was able to go to work and be there by himself. Tr. 192:6–8. However, Dr. Callaghan noted that "within hours [of Mr. Bauer going to work on October 20, 2017,] he[was] dead, and that just is not really compatible with GBS." Tr. 192:9–10. Dr. Callaghan explained that this type of quick deterioration and death "is very consistent with hyperkalemia, which can – in a manner of minutes [] change." Tr. 192:13–15. He explained, for example, that a change in potassium level from normal to 10 can result in immediate paralysis. Tr. 192:15–16. Dr. Jarvis acknowledged that GBS can cause death in rare cases but that "it takes[] . . . weeks and months to progress to that level." Tr. 192:11–13. Dr. Callaghan opined that the change in Mr. Bauer's symptoms prior to his death "is really important as far as trying to distinguish between GBS and hyperkalemia." Tr. 192:17–18.

Dr. Callaghan opined that Mr. Bauer's hyperkalemia began before the vaccination at issue. *See* Tr. 203:12–14. Dr. Callaghan asserted that Mr. Bauer's "symptoms of hyperkalemia started out relatively mild with cramps, as indicated in a few of his notes in the months preceding [his death], and that it really escalated in the last . . . couple or three weeks," as evidenced by the October 17, 2017 medical record. Tr. 203:12–17. Dr. Callaghan continued that Mr. Bauer's hyperkalemia then "reached a dramatic and critical stage on the day of his death, going from[] . . . someone that could walk . . . and go to work to someone that died only a handful of hours later." Tr. 203:17–21. Dr. Callaghan later reiterated that Mr. Bauer's "symptoms of hyperkalemia probably were there for months . . . but increased in the last few weeks before his hospitalization." Tr. 217:25–218:2. Dr. Callaghan acknowledged that no medical record from before the flu vaccination at issue documented that Mr. Bauer suffered from weakness. Tr. 218:13. While Dr. Callaghan did not know whether Mr. Bauer experienced weakness pre vaccination, Dr. Callaghan nevertheless believed that "the medical records are clear that he had symptoms prior to [the vaccination], because that[is] what he describe[d] to Dr. Jarvis and to other providers." Tr. 218:24–219:2. These symptoms included "leg cramps [Mr. Bauer reported to] multiple providers and then to Dr. Jarvis." Tr. 219:3–4. Further, the October 17, 2017 medical record indicated that Mr. Bauer's symptoms had been ongoing for a couple of weeks, and that his symptoms predated vaccination. Tr. 219:4–7. Dr. Callaghan explained that while Mr. Bauer suffered from dramatic symptoms due to hyperkalemic paralysis, this "does[not] mean [he could not] have [had] subtler

hyperkalemia predating that for quite a while." Tr. 221:7–14. Dr. Callaghan stated that the "sudden change" from Mr. Bauer's ability to walk to being "completely paralyzed[]" a few hours later "makes it very, very clear that there[is] something other than GBS going on, and then the potassium [level] of 10.8 makes it very easy to tell what[is] going on." Tr. 221:14–19.

Dr. Callaghan opined that "the cause of [Mr. Bauer's] symptoms is quite clear." Tr. 193:14–15. Dr. Callaghan explained, "it[is] not like his potassium is subtly high." Tr. 193:15–16. He agreed with Dr. Jarvis that a potassium level of 10.8 "is more compatible with death." Tr. 193:17–18. Dr. Callaghan expanded on this, explaining that a potassium level of "10.8 is incredibly high, and so there[is] really not a need for a second explanation when there[is] such an obvious one staring you in the face, which is that his potassium [was] incredibly high." Tr. 193:20–23. Dr. Callaghan remarked that there was "nothing about his history" that was incompatible with this explanation. Tr. 193:23–24. Rather, "it[is] all very compatible with his high potassium causing all of these symptoms, including, unfortunately, his sudden cardiac death." Tr. 193:24–194:2. Discussing his opinion that Mr. Bauer did not have GBS, Dr. Callaghan stated that he "would never say anything is 100 percent, but this case is probably as close as you can get to that even from afar." Tr. 262:1–3. He further stated that "it[is] so clear in this case that [Mr. Bauer] had a GBS mimic, and[] . . . there[is] very little to support a GBS diagnosis and an incredible amount of information to support secondary hyperkalemia leading to paralysis." Tr. 248:16–20.

Dr. Callaghan asserted that "GBS cannot be diagnosed in the setting of extreme hyperkalemia such as in this case." Resp't's Ex. A at 1. While theoretically possible that someone could have such severe hyperkalemia and GBS simultaneously, Dr. Callaghan compared this scenario of "two incredibly rare things happen[ing]" to "win[ning] the lottery and get[ting] struck by lightning." Tr. 194:10–14. Dr. Callaghan maintained that even if someone had both conditions at once, Dr. Callaghan would first correct the patient's potassium levels and see if the symptoms persisted before "evaluating both GBS and its [other] mimics." Tr. 194:14–17.

Dr. Callaghan described this case as "very obviously a case of hyperkalemia causing neuromuscular respiratory weakness." Tr. 194:22–23. Dr. Callaghan also asserted that it is obvious "that the hyperkalemia is due to the three most common factors[:] kidney failure, supplementation, and medications, in this case spironolactone." Tr. 194:22–195:1. He explained that spironolactone's "job is to keep potassium in the body." Tr. 195:2–4. Dr. Callaghan stated that spironolactone is "tolerated especially in people with normal kidneys[]" but is not tolerated well by those with kidney dysfunction. Tr. 195:25–196:2. He continued that a dose of 25 mg twice per day is acceptable for people with eGFRs above 60, "but once [the GFR] drops below that, [doctors are] supposed to at least half the dose, if not more like a fourth or an eighth of a dose." Tr. 196:3–7. When eGFR drops below 30, as Mr. Bauer's was when he presented to the ER, "you are not allowed to be on spironolactone. That[is] a no-no." Tr. 196:7–9. Dr. Callaghan opined that spironolactone is not well-tolerated once a patient "start[s] to get worsening kidney failure like [Mr. Bauer] did in May of 2017." Tr. 196:9–11. Dr. Callaghan explained that the timing between beginning spironolactone and developing hyperkalemia is "highly variable, and the biggest variable [in the present case] is his kidney function, and his kidney function dramatically change[d] . . . from a GFR of near 60 to a GFR of 31." Tr. 228:13–16. Dr. Callaghan noted that maintaining Mr. Bauer on the same dosage of spironolactone as his kidney function declined "basically increase[ed] his dose of spironolactone." Tr. 228:25–229:1. Furthermore, Dr. Callaghan noted that

even a small decline in GFR, especially when a patient already has a low GFR, "can have a dramatic change on [] potassium levels, especially when" the patient is also taking a medication like spironolactone, "because it[is] a . . . double whammy." Tr. 258:19–25. Regarding potassium supplementation, Dr. Callaghan stated that hyperkalemia caused by treatment of hypokalemia is common, and he explained that "[a]ny time you try to correct something, you can overcorrect." Tr. 261:1–3.

Dr. Callaghan noted that Mr. Bauer had Stage 4 CKD when he presented to the ER on October 20, 2017, with an eGFR of 18. Tr. 196:18–19. Because Mr. Bauer was close to Stage 4 when he had an eGFR of 31 in May of 2017, Dr. Callaghan stated that "it did[not] take much to go from there to [S]tage 4." Tr. 196:22–24. Dr. Callaghan opined that this change did not constitute a dramatic worsening. Tr. 196:24–197:1.

Discussing why he believed that Mr. Bauer's flu vaccination was unrelated to his hyperkalemia and death, Dr. Callaghan stated that "the main reason is that his symptoms[] . . . predated the flu vaccine." Tr. 204:5–7. In addition, "he did not have GBS or a condition that [Dr. Callaghan knew] of that was associated with the [flu] vaccine." Tr. 204:7–9.

Regarding the potential biological mechanisms discussed by Dr. Jarvis, Dr. Callaghan noted that while Dr. Jarvis "kind of talked about antibodies that would attack the myelin, that would also attack the kidney," he did not present "any more specifics on that or any kind of evidence" in support of a proposed biological mechanism. Tr. 205:9–14. Dr. Callaghan explained that rhabdomyolysis is muscle breakdown and that it can cause kidney damage when "bad enough." Tr. 205:16–17. However, because Mr. Bauer's liver enzymes, AST and ALT, were normal, Dr. Callaghan opined that "there[is] pretty concrete evidence that that was not a mechanism by which [Mr. Bauer] would have kidney failure." Tr. 205:20–206:2. Dr. Callaghan noted that these enzymes are also muscle enzymes which become elevated "when [] muscles get very injured." Tr. 205:23–24.

Dr. Callaghan acknowledged that "[t]here are case reports[]" linking GBS and acute kidney injury. Tr. 238:24. He also noted that "[t]here are cases[]" linking the flu vaccine and kidney injury. Tr. 239:15. Dr. Callaghan stated that "GBS and kidney problems are[not] really tightly linked[]" and that he would expect to see a multi-organ attack in cases where GBS causes secondary kidney problems. Tr. 254:20–25. He explained that such organ damage is due to the body's inability to get a sufficient amount of blood and oxygen to the organs rather than due to an autoimmune attack on the organs themselves. Tr. 256:6–11.

Discussing his clinical experience, Dr. Callaghan stated that patients with GBS typically "do not have kidney disease, but if they are sick enough in the hospital," multiple organ failure can occur. Tr. 242:8–12. However, he opined that this "does not necessarily mean that GBS causes kidney failure." Tr. 242:13–14. Dr. Callaghan continued that when people with GBS develop kidney problems, this is typically because they are in the hospital for a long time and are very sick and have autonomic instability and blood pressure issues, which then "leads to secondary problems with their kidneys." Tr. 242:24–243:4. Dr. Callaghan explained that all organs can worsen when a patient has autonomic instability and blood pressure fluctuations. Tr. 243:12–14. He continued that respiratory failure can "can also impair organs." Tr. 243:15–16.

Dr. Callaghan acknowledged that acute kidney injury can worsen CKD. Tr. 239:18. When asked whether he disagreed with Dr. Jarvis's October 20, 2017 diagnosis of acute kidney injury, Dr. Callaghan stated that this is difficult to tell because Mr. Bauer's kidney function had not been tested since May of 2017. Tr. 241:2–4. Dr. Callaghan noted that the decline in eGFR, from 31 in May of 2017 to 18 on October 20, 2017, "can be due to [Mr. Bauer's] diabetes and hypertension, which are the two most common causes of kidney dysfunction." Tr. 241:4–7. Dr. Callaghan noted that doctors typically diagnose acute kidney injury when a patient presents with a decline in kidney function, but Dr. Callaghan did not "know the exact acuity here." Tr. 241:11–14. Dr. Callaghan acknowledged that an acute kidney injury could cause hyperkalemia. Tr. 242:2.

Noting Mr. Bauer's history of diabetes, hypertension, and coronary artery disease, Dr. Callaghan stated that "[i]t[is] not a mystery why Mr. Bauer had kidney dysfunction." Tr. 252:10–17. Dr. Callaghan noted that diabetes and hypertension are "the top two causes for kidney failure." Tr. 252:10–11. He explained that diabetes "complications continue to develop even with excellent control of diabetes." Tr. 253:8–9.

3. Respondent's Expert, Dr. Fine

Dr. Fine submitted an expert report following the entitlement hearing. Resp't's Ex. G, ECF No. 55-1. Summarizing his opinions, Dr. Fine opined that Mr. Bauer's death was "due to severe hyperkalemia in the context of worsening kidney function and multiple factors that increase blood creatinine, including: an angiotensin converting enzyme (ACE) inhibitor (benazepril), an aldosterone inhibitor (spironolactone), and potassium supplementation with large doses of potassium." *Id.* at 1–2. Citing medical literature, Dr. Fine explained that "ACE-inhibitors and potassium sparing diuretics[]" are used to treat hypertension and congestive heart failure but "are also known to cause hyperkalemia." *Id.* at 2 (citing Resp't's Ex. I at 1–2, ECF No. 56-1;[75] Resp't's Ex. J, ECF No. 56-2;[76] Resp't's Ex. K, ECF No. 56-3;[77] Resp't's Ex. L, ECF No. 56-4;[78] Resp't's Ex. M at 11, ECF No. 56-5).[79] Dr. Fine also noted that Mr. Bauer was "taking a beta-blocker which further increases risk of hyperkalemia." *Id.* (citing Resp't's Exs. I at 2, J at 2, K at 2, M at 8). Dr Fine averred that "Mr. Bauer's cardiac arrest was caused by hyperkalemia[]" and that "[t]he muscle spasms [he] suffered in the weeks prior to his death are most consistent with those seen in patients with hyperkalemia." *Id.* Dr. Fine further asserted that "[t]here is no evidence to suggest" that Mr. Bauer's flu vaccination "contributed to his worsening kidney function or hyperkalemia." *Id.*

Dr. Fine noted that ACE-inhibitors and spironolactone can cause hyperkalemia individually and "especially if used together (synergistic effects) and in those with impaired kidney function." *Id.* at 3. He continued that "potassium supplementation in context of impaired kidney function further increase[s] the risk of hyperkalemia" and that beta-blockers "have also been

[75] Tasnim Momoniat et al., *ACE Inhibitors and ARBs: Managing Potassium and Renal Function*, 86(9) CLEVELAND CLINIC J. MEDICINE 601 (2019).
[76] Lisa Dolovich et al., *Hyperkalemia Associated with Spironolactone Therapy*, 51 CANADIAN FAMILY PHYSICIAN 357 (2005).
[77] Prescriber Update: Medicines and Hyperkalemia (2015).
[78] David B. Mount, *Clinical Manifestations of Hyperkalemia in Adults*, UPTODATE (Aug. 2022).
[79] David B. Mount, *Causes and Evaluation of Hyperkalemia in Adults*, UPTODATE (Aug. 2022).

described as increasing risk of hyperkalemia." *Id.*; *see also* Resp't's Ex. K at 2. He noted that spironolactone is a potassium sparing diuretic that results in potassium retention, as opposed to other types of diuretics that "can result in potassium wasting." *Id.* Dr. Fine noted that Mr. Bauer's eGFR and creatinine levels worsened between December 9, 2016, when his eGFR was 56 and his creatinine was 1.4, and May 17, 2017, when his eGFR was 31 and his creatinine was 2.15. *Id.* at 4. Dr. Fine identified these changes as "a significant decline in [Mr. Bauer's] kidney function[,]" but he noted that "[t]his decline in kidney function was not commented on [by Mr. Bauer's endocrinology provider,] and no changes were made to his antihypertensive, lipid-lowering, or diabetes medications." *Id.* Dr. Fine explained that an eGFR of 31 "would be classified as Stage 3b" CKD, which indicates "moderately to severely decreased kidney function." *Id.* This was a substantial decline from Mr. Bauer's baseline kidney function, which Dr. Fine stated was between a 59 and 76 eGFR. *Id.* at 6. Dr. Fine noted that after Mr. Bauer's eGFR declined to 31 in May of 2017, it declined again to 18 by the time he presented to the hospital in October of 2017. *Id.* Dr. Fine explained that an eGFR of 18 indicates Stage 4 CKD and "severely decreased kidney function[]." *Id.* He opined that the eGFR of 18 in October of 2017 "is consistent with a decline in kidney function that had begun earlier that year." *Id.* He noted that this decline was of unclear etiology, but he stated that "this decline contributed significantly to his hyperkalemia (in addition to his being on potassium supplement[ation] and two medications that increase potassium levels[]." *Id.*

Dr. Fine noted that "[h]yperkalemia is most often due to impaired urinary potassium excretion in the context of acute or chronic kidney disease and/or due to disorders or drugs that inhibit the renin-angiotensin-aldosterone axis."[80] *Id.* Such drugs include "ACE-inhibitors, angiotensin II receptor blockers, and mineralocorticoid antagonist." *Id.* Dr. Fine noted that "Mr. Bauer had both chronic kidney disease with significantly reduced kidney function . . . and was on both an ACE-inhibitor, benazepril, and a mineralocorticoid antagonist [], spironolactone." *Id.* Dr. Fine noted that Mr. Bauer's potassium supplementation "added to his potassium load[]" and that ibuprofen, a non-steroidal anti-inflammatory drug that can exacerbate hyperkalemia, was listed on his medications. *Id.* at 6–7. However, Dr. Fine acknowledged that ibuprofen appeared to be a medication Mr. Bauer took as needed. *Id.* at 7.

Dr. Fine explained how worsening kidney function can cause hyperkalemia. *Id.* He noted that "[w]hen patients lose kidney function, they lose nephrons (the basic units of the kidney), and this reduction of nephron mass compromises the ability of the kidney to excrete potassium. *Id.* Dr. Fine noted that hyperkalemia is common in patients with CKD due to this loss of kidney function. *Id.* In Mr. Bauer's case, "[t]hough he previously had low potassium levels, with ongoing kidney function decline, lower potassium excretion was likely to occur, with development of higher potassium levels." *Id.* Dr. Fine asserted that this effect was exacerbated by Mr. Bauer's use of benazepril, an ACE-inhibitor, and spironolactone. *Id.* Dr. Fine explained that "ACE-inhibitors reduce aldosterone levels[,] and spironolactone directly inhibits aldosterone effects on the kidney." *Id.* He continued that both of these medications can cause hyperkalemia, and cause hyperkalemia more often in the setting of CKD, because "aldosterone is the primary hormone involved in potassium excretion in the kidneys." *Id.* Dr. Fine opined that because "Mr. Bauer was taking high doses of potassium supplementation with doses noted as high as 40mEq three times a day (120

[80] The renin-angiotensin-aldosterone system is "the regulation of sodium balance, fluid volume, and blood pressure by renal secretions." *Dorland's* at 1832.

mEq/day)[,] . . . it is not surprising that the potassium at the time of admission was elevated to a life-threatening level." *Id.*

Dr. Fine wrote that "the fact that [Mr. Bauer] had symptoms for weeks proceeding his presentation is very consistent with his level [of] hyperkalemia[]" because "[i]n order for potassium to get to [a level of 10.8], it would have to occur over some time." *Id.* Dr. Fine noted that a potassium level over 5.2 is abnormal and that levels of greater than 6.5-7.0 are "life-threatening, ultimately leading to cardiac arrhythmias and death." *Id.* Dr. Fine explained that in cases like Mr. Bauer's when the rise in potassium levels occurs slowly, patients can tolerate "extreme elevation" of potassium levels." *Id.* However, "[e]ventually, the potassium elevation will result in cardiac arrhythmia and death if it continues to increase, as it appears to have done in this case." *Id.* Dr. Fine noted that symptoms including muscle weakness and paralysis are associated with potassium levels above seven. *Id.* at 8. He also noted that "hyperkalemia 'can cause ascending muscle weakness that begins with the legs and progresses to the trunk and arms.'" *Id.* (quoting Resp't's Ex. L at 2). He further noted that "this can progress to flaccid paralysis mimicking" GBS. *Id.* Dr. Fine stated that these were Mr. Bauer's symptoms "in the weeks preceding his hyperkalemic cardiac arrythmia." *Id.* Citing Mr. Bauer's medical record from his October 17, 2016 visit with Dr. Jarvis, Dr. Fine asserted that Mr. Bauer's "muscle symptoms appeared to precede his vaccine exposure." *Id.*

In response to Dr. Jarvis's contention that GBS can cause acute kidney injury, Dr. Fine averred that Dr. Jarvis did not mention the "significant decrease" in Mr. Bauer's kidney function that occurred between December of 2016 and May of 2017. *Id.* Furthermore, Dr. Fine noted that Mr. Bauer's eGFR of 59 in December of 2016 indicated a "loss of function prior to th[e] more precipitous drop in function[]" that occurred by May of 2017. *Id.* Dr. Fine stated that "[n]o intervention was instituted" for Mr. Bauer's declining kidney function and that "one would expect ongoing decline in kidney function in the absence of an intervention." *Id.*

Dr. Fine opined that Mr. Bauer's kidney disease was caused by his "long-standing diabetes, microvascular disease[,] and hypertension." *Id.* Dr. Fine continued that Mr. Bauer's medications, including an ACE-inhibitor and possibly ibuprofen, likely contributed to his worsening kidney function. *Id.* Dr. Fine noted, however, that "the drop in GFR by May [of] 2017 suggested that he was suffering from progressive kidney disease, and there appeared to be no intervention that would reverse the course." *Id.*

Dr. Fine wrote that "acute kidney injury is rare in those presenting with GBS[,]" and he cited medical literature to support that "[r]eview articles regarding [GBS] do not mention kidneys, except in context of workup to assess for other causes of flaccid paralysis such as electrolyte disorders (such as hyperkalemia)." *Id.* (citing Resp't's Ex. O, ECF No. 56-7;[81] Resp't's Ex. P, ECF

[81] Nobuhiro Yuki & Hans-Peter Hartung, *Guillain-Barré Syndrome*, 366 NEW ENGLAND J. MEDICINE 2294 (2012).

No. 56-8;[82] Resp't's Ex. Q, ECF No. 56-9;[83] Resp't's Ex. R, ECF No. 56-10).[84] Discussing the Khajehdehi et al. paper submitted by Petitioner, Dr. Fine noted that all of the patients who developed acute kidney injury "did so after presenting with normal creatinine levels at the time of their hospitalization[,]" unlike Mr. Bauer. *Id.* at 8–9. Dr. Fine indicated that "[t]here was no evidence of an [acute kidney injury-]inciting event, such as hypotension, in Mr. Bauer's case." *Id.* at 9. Thus, "Mr. Bauer far more likely was suffering from progression of his" CKD. *Id.*

Dr. Fine further stated that he could not find any articles attributing hyperkalemia to flu vaccinations, and he asserted that "[t]here is no scientific basis to this hypothesis." *Id.* He also noted that flu vaccines have not been linked to progressive kidney disease. *Id.* Dr. Fine opined that Mr. Bauer's flu vaccination "neither caused nor significantly aggravated [his] hyperkalemia leading to his cardiac arrest and death." *Id.*

IV. Applicable 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) that the petitioner suffered an "off-Table injury," one not listed on the Table, as a result of his receiving a covered vaccine. *See* § 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). In this case, Petitioner must prove by preponderant evidence that Mr. Bauer suffered a Table injury or that his injury was caused-in-fact or significantly aggravated by a Table vaccine.

A. Table GBS

To establish a GBS Table injury following a flu vaccination, a petitioner must demonstrate by preponderant evidence that the onset of his GBS occurred at least three days but no more than forty-two days post vaccination. 42 C.F.R. § 100.3(a). The Table's Qualifications and Aids to Interpretation ("QAIs") define GBS as:

> [A]n acute monophasic peripheral neuropathy that encompasses a spectrum of four clinicopathological subtypes . . . . For each subtype of GBS, the interval between the first appearance of symptoms and the nadir of weakness is between 12 hours and 28 days. This is followed in all subtypes by a clinical plateau with stabilization at the nadir of symptoms, or subsequent improvement without significant relapse. Death my occur without a clinical plateau.

42 C.F.R. § 100.3(c)(15)(i).

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[82] Hugh J. Wilson et al., *Guillain-Barré Syndrome*, 388 LANCET 717 (2016).
[83] Sonja E. Leonard et al., *Diagnosis and Management of Guillain-Barré Syndrome in Ten Steps*, 15 NATURE REVS. NEUROLOGY 671 (2019).
[84] Swathy Chandrashekhar & Mazen M. Dimachkie, *Guillain-Barré Syndrome in Adults: Pathogenesis, Clinical Features, and Diagnosis*, UPTODATE (2022).

The Table identifies the four subtypes of GBS as acute inflammatory demyelinating polyneuropathy ("AIDP"), acute motor axonal neuropathy ("AMAN"), acute motor and sensory neuropathy ("AMSAN"), and Fisher Syndrome ("FS"). 42 C.F.R. § 100.3(c)(15)(ii)–(iii). It provides requirements for the diagnosis of the different subtypes of GBS. *See id.* Evidence of "electrophysiologic findings consistent with GBS or an elevation of cerebral spinal fluid (CSF) protein with a total CSF white blood cell count below 50 cells per microliter[]" is not required to establish a diagnosis of GBS consistent with the Table, but it is "supportive" evidence. 42 C.F.R. § 100.3(c)(15)(iv). The QAIs also specify that "[t]o qualify as any subtype of GBS, there must not be a more likely alternative diagnosis for the weakness." 42 C.F.R. § 100.3(c)(15)(v). The QAIs state that "[e]xclusionary criteria for the diagnosis of all subtypes of GBS include the ultimate diagnosis of any of" a list of conditions, which include hyperkalemia and hypokalemia. 42 C.F.R. § 100.3(c)(15)(vi).

B. Causation-in-Fact

To establish causation-in-fact, a petitioner must demonstrate by a preponderance of the evidence that the vaccine was the cause of the injury. § 300aa-13(a)(1)(A). A petitioner is required to prove that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321–22 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)).

In the seminal case of *Althen v. Sec'y of Health & Hum. Servs.*, the Federal Circuit set forth a three-pronged test used to determine whether a petitioner has established a causal link between a vaccine and the claimed injury. *See* 418 F.3d 1274, 1278–79 (Fed. Cir. 2005). The *Althen* test requires petitioners to set forth: "(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." *Id.* at 1278. To establish entitlement to compensation under the Program, a petitioner is required to establish each of the three prongs of *Althen* by a preponderance of the evidence. *Id.* "[C]lose calls regarding causation are resolved in favor of injured claimants." *Id.* at 1280. Further, evidence used to satisfy one prong of the test may overlap to satisfy another prong. *Capizzano*, 440 F.3d at 1326.

Under the first prong of *Althen*, a petitioner must offer a scientific or medical theory that answers in the affirmative the question: "can the vaccine[] at issue cause the type of injury alleged?" *See Pafford v. Sec'y of Health & Hum. Servs.*, No. 01-0165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004), *mot. for rev. den'd*, 64 Fed. Cl. 19 (2005), *aff'd*, 451 F.3d 1352 (Fed. Cir. 2006). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such theory must only be "legally probable, not medically or scientifically certain." *Id.* at 548–49. Petitioners are not required to identify "specific biological mechanisms" to establish causation, nor are they required to present "epidemiologic studies, rechallenge[] the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities." *Capizzano*, 440 F.3d at 1325 (quoting *Althen*, 418 F.3d at 1280). Scientific and "objective confirmation" of the medical theory with additional medical documentation is unnecessary. *Althen*, 418 F.3d at 1278–81; *see also Moberly*, 592 F.3d at 1322.

However, as the Federal Circuit has made clear, "simply identifying a 'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof." *LaLonde v. Sec'y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) (citing *Moberly*, 592 F.3d at 1322). Indeed, the Federal Circuit has "consistently rejected theories that the vaccine only 'likely caused' the injury and reiterated that a 'plausible' or 'possible' causal theory does not satisfy the standard." *Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, (Fed. Cir. 2019) (citing *Moberly*, 592 F.3d at 1322 and *LaLonde*, 746 F.3d at 1339). Rather, "[a] petitioner must provide a reputable medical or scientific explanation that pertains specifically to the petitioner's case." *Moberly*, 592 F.3d at 1322. In general, "the statutory standard of preponderance of the evidence requires a petitioner to demonstrate that the vaccine more likely than not caused the condition alleged." *LaLonde*, 746 F.3d at 1339.

Furthermore, establishing a sound and reliable medical theory connecting the vaccine to the injury often requires a petitioner to present expert testimony in support of her claim. *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357,1361 (Fed. Cir. 2000). The Supreme Court's opinion in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993), requires that courts determine the reliability of an expert opinion before it may be considered as evidence. However, in the Vaccine Program, the *Daubert* factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66–67 (2010) ("[U]niquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted."); *see also Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)). Under *Daubert*, the

> factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

Terran, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

The *Daubert* factors are "meant to be helpful, not definitive." *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 151 (1999). The factors do not "constitute 'a definitive checklist or test'" and may be applied differently depending on the facts of a particular case. *Id.* at 150 (quoting *Daubert*, 509 U.S. at 593).

"In short, the requirement that an expert's testimony pertain to 'scientific knowledge' establishes a standard of evidentiary reliability." *Daubert*, 509 U.S. at 590 (citation omitted). Thus, for Vaccine Act claims, a "special master is entitled to require some indicia of reliability to support the assertion of the expert witness." *Moberly*, 592 F.3d at 1324. Nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder v. Sec'y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 743 (2009) (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)); *see also D'Tiole v. Sec'y of Health & Hum. Servs.*,

No. 15-085V, 2016 WL 7664475, at *24 (Fed. Cl. Spec. Mstr. Nov. 28, 2016) (stating that the Vaccine Act "require[s] a chain of reliable propositions supporting [a] petitioner's theory[]").

Under the second prong of *Althen*, a petitioner must prove that the vaccine actually did cause the alleged injury in a particular case. *See Pafford*, 2004 WL 1717359, at *4; *Althen*, 418 F.3d at 1279. The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). A petitioner does not meet this obligation by showing only a temporal association between the vaccination and the injury; instead, the petitioner "must explain *how* and *why* the injury occurred." *Pafford*, 2004 WL 1717359, at *4 (emphasis in original). The special master in *Pafford* noted petitioners "must prove [] both that her vaccinations were a substantial factor in causing the illness . . . and that the harm would not have occurred in the absence of the vaccination." 2004 WL 1717359, at *4 (citing *Shyface*, 165 F.3d at 1352). A reputable medical or scientific explanation must support this logical sequence of cause and effect. *Hodges v. Sec'y of Health & Hum. Servs.*, 9 F.3d 958, 961 (Fed Cir. 1993) (citation omitted). Nevertheless, "[r]equiring epidemiologic studies . . . or general acceptance in the scientific or medical communities . . . impermissibly raises a claimant's burden under the Vaccine Act and hinders the system created by Congress . . . ." *Capizzano*, 440 F.3d at 1325–26. "[C]lose calls regarding causation are resolved in favor of injured claimants." *Althen*, 418 F.3d at 1280.

In Program cases, contemporaneous medical records and the opinions of treating physicians are favored. *Capizzano*, 440 F.3d at 1326 (citing *Althen*, 418 F.3d at 1280). Indeed, when reviewing the record, a special master must consider the opinions of treating physicians. *Capizzano*, 440 F.3d at 1326. 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. 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." *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). 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." § 300aa-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).

To satisfy the third *Althen* prong, a petitioner must establish a "proximate temporal relationship" between the vaccination and the alleged injury. *Althen*, 418 F.3d at 1281. This "requires preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). Typically, "a petitioner's failure to satisfy the proximate temporal relationship prong is due to the fact that onset was too late after the administration of a vaccine for the vaccine to be the cause." *Id.* However, "cases in which onset is too soon" also fail this prong; "in either case, the temporal relationship is not such that it is medically acceptable to conclude that the vaccination and the injury are causally linked." *Id.*; *see also Locane v. Sec'y of Health & Hum. Servs.*, 685 F.3d 1375, 1381 (Fed. Cir. 2012) ("[If] the illness was present before the vaccine was administered, logically, the vaccine could not have caused the illness.").

Although a temporal association alone is insufficient to establish causation, under the third prong of *Althen*, a petitioner must show that the timing of the injury fits with the causal theory. *See Althen*, 418 F.3d at 1278. The special master cannot infer causation from temporal proximity alone. *See Thibaudeau v. Sec'y of Health & Hum. Servs.*, 24 Cl. Ct. 400, 403–04 (1991); *see also Grant*, 956 F.2d at 1148 ("[T]he inoculation is not the cause of every event that occurs within the ten[-]day period . . . [w]ithout more, this proximate temporal relationship will not support a finding of causation." (quoting *Hasler v. United States*, 718 F.2d 202, 205 (6th Cir. 1983))).

A petitioner who satisfies all three prongs of the *Althen* test has established a prima facie showing of causation. *Hammitt v. Sec'y of Health & Hum. Servs*., 98 Fed. Cl. 719, 726 (2011). A petitioner who demonstrates by a preponderance of the evidence that he suffered an injury caused by vaccination is entitled to compensation unless the respondent can demonstrate by a preponderance of the evidence that the injury was caused by factors unrelated to the vaccination. *See Althen*, 418 F.3d at 1278; *Knudsen*, 35 F.3d at 547. In such a case, the government must not merely prove the existence of an alternative cause, but that such an alternative actually caused the injury. *Knudsen*, 35 F.3d at 549. Consequently, when and if the petitioner establishes a prima facie case, the burden then shifts to the government to prove that an alternative cause, unrelated to the administration of the vaccine, was the "sole substantial factor" in causing the alleged injury. *See de Bazan*, 539 F.3d at 1354; *see also Hammitt*, 98 Fed. Cl. at 726 (explaining that the respondent's burden is to show that the "factor unrelated" was the "sole substantial factor" in causing the injury). Additionally, a factor unrelated "may not include 'any idiopathic, unexplained, unknown, hypothetical, or undocumentable cause, factor, injury, illness or condition.'" § 300aa-13(a)(2); *see also Doe v. Sec'y of Health & Hum. Servs.*, 601 F.3d 1349 (Fed. Cir. 2010) (stating that an idiopathic diagnosis cannot be a "factor unrelated," as it is idiopathic).

C. Significant Aggravation

Petitioners must establish causation in all off-Table cases; however, petitioners may establish they are entitled to compensation based on a claim that vaccination significantly

aggravated a preexisting condition. The Vaccine Act defines significant aggravation as "any change for the worse in a preexisting condition which results in markedly greater disability, pain, or illness accompanied by substantial deterioration of health." § 300aa-33(4). When a petitioner makes this argument, the evidentiary burden is expanded. *See Loving v. Sec'y of Health and Hum. Servs.*, 86 Fed. Cl. 135, 144 (2009). In *Loving*, the Court set forth a six-factor test, which requires establishing the following:

> (1) the person's condition prior to administration of the vaccine, (2) the person's current condition (or the condition following the vaccination if that is also pertinent), (3) whether the person's current condition constitutes a "significant aggravation" of the person's condition prior to vaccination, (4) a medical theory causally connecting such a significantly worsened condition to the vaccination, (5) a logical sequence of cause and effect showing that the vaccination was the reason for the significant aggravation, and (6) a showing of a proximate temporal relationship between the vaccination and the significant aggravation.

Loving, 86 Fed. Cl. at 144.

The *Loving* analysis requires the special master to "evaluat[e] whether the vaccine made the person worse than the person would have been but for the vaccination. In doing so, the natural course of the disease must be considered." *Locane v. Sec'y of Health & Hum. Servs.*, No. 99-589V, 2011 WL 3855486 at *10 (Fed. Cl. Spec. Mstr. Feb. 17, 2011), *mot. for review den'd*, 99 Fed. Cl. 715 (2011), *aff'd*, 685 F.3d 1375 (Fed. Cir. 2012); *see also Hennessey v. Sec'y of Health & Hum. Serv.*, No. 01-190V, 2009 WL 1709053, at *41–42 (Fed. Cl. Spec. Mstr. May 29, 2009), *mot. for review den'd*, 91 Fed. Cl. 126 (2010). However, a petitioner is not required "to demonstrate an expected outcome and that her current-post vaccination condition was worse than such expected outcome." *Sharpe v. Sec'y of Health & Hum. Servs.*, 964 F.3d 1072, 1081 (Fed. Cir. 2020).

V. Discussion

A. GBS

I find that Petitioner has not presented preponderant evidence that Mr. Bauer suffered from GBS pursuant to the Table or for the purpose of a causation-in-fact or significant aggravation claim. To establish that he suffered a Table GBS injury, Petitioner must prove by preponderant evidence that Mr. Bauer's symptoms began between three- and forty-two-days post vaccination. However, in this case, although the parties dispute whether Petitioner's symptoms predated his October 12, 2017 vaccination, Petitioner's affidavit and testimony provide preponderant evidence that Mr. Bauer's symptoms started by no later than October 13, 2017, one day post vaccination. This is outside the Table's three-to-forty-two-day window. Furthermore, the Table specifies that an ultimate diagnosis of hyperkalemia is an exclusionary criterion for a GBS diagnosis. It is undisputed that Mr. Bauer suffered from severe hyperkalemia when he presented to the ER on October 20, 2017. Thus, Petitioner cannot prevail on a Table GBS claim.

Mr. Bauer's undisputed hyperkalemia also prevents Petitioner from presenting preponderant evidence that Mr. Bauer suffered from GBS that was caused-in-fact or significantly aggravated by his flu vaccination. Dr. Callaghan, a neurologist with expertise in GBS and GBS

mimics, explained that the Brighton criteria commonly used to diagnose GBS require "the absence of an alternative cause" of symptoms to diagnose GBS. Tr. 182:6–9. Dr. Callaghan explained that "hyperkalemia is a well-known mimic of GBS" and that it "can cause symptoms that are indistinguishable from GBS." Resp't's Ex. A at 1; Tr. 179:9–10. He further stated that "GBS cannot be diagnosed in the setting of extreme hyperkalemia such as in this case[,]" and he unequivocally stated that this case is "very obviously a case of hyperkalemia causing neuromuscular respiratory weakness." Resp't's Ex. A at 1; Tr. 194:22–23. He further explained that the progression of Mr. Bauer's condition, from being able to go to work on the morning of October 20, 2017, to being unable to walk and passing away hours later, "is very consistent with hyperkalemia," which can change quickly, but "not really compatible with GBS." Tr. 192:6–15. I find Dr. Callaghan's statements and opinions persuasive.

Dr. Jarvis, Mr. Bauer's PCP, diagnosed Mr. Bauer with possible GBS along with hyperkalemia. However, while the opinions of treating physicians are entitled to some weight, special masters are not required to accept a treating physician's conclusions. Indeed, the Federal Circuit has clearly stated that 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. When determining the reliability of medical or expert opinions or determining the relative weight to give to competing opinions, special masters may consider whether the issues opined on are within a witness's area of expertise. *See Wyatt v. Sec'y of Health & Hum. Servs.*, 825 Fed. Appx. 880, 886 (Fed. Cir. 2020) (holding that "the factual findings of the Special Master regarding GBS [were] not arbitrary and capricious[]" when, among other issues, "the Special Master determined that Dr. DeMio's expert testimony should be given little weight because Dr. DeMio has no specialized training in autoimmune or neurological disorders and had conducted no research in either field[]"). "This is most obviously necessary when an expert offers an opinion that plainly exceeds his training or individual competence." *Hughes v. Sec'y of Health & Hum. Servs.*, No. 16-930V, 2021 WL 839092, at *24 (Fed. Cl. Spec. Mstr. Jan. 4, 2021). Indeed, special masters have previously deemed opinion evidence unreliable due to an expert's lack of pertinent qualifications. *See R.K. v. Sec'y of Health & Hum. Servs.*, No. 03-0632V, 2015 WL 10936124, at *118 (Fed. Cl. Spec. Mstr. Sept. 28, 2015) ("Doctor Deth's medical opinions regarding A.K.'s gastrointestinal inflammation and its purported relationship to two vaccinations were outside his expertise and unsupported by other evidence. I find them inherently unreliable based on Dr. Deth's lack of qualifications to diagnose this child and to opine on the cause of a neurodevelopmental condition.").

In this case, I grant greater weight to Dr. Callaghan's statements and opinions regarding GBS and its diagnostic criteria as well as GBS mimics than to those of Dr. Jarvis. Dr. Callaghan is a neurologist with significant experience diagnosing, treating, and researching GBS and GBS mimics. Petitioner, however, did not proffer Dr. Jarvis as an expert in neurology or GBS, and Dr. Jarvis acknowledged that he was only involved in treating one case of GBS years ago. Although Dr. Jarvis testified that he is familiar with medical literature regarding neuromuscular conditions and that he researched medical literature for this case, the extent of his familiarity with literature specifically pertaining to diagnostic criteria for GBS and distinguishing it from hyperkalemia is unclear. Furthermore, he did not indicate that he has any specialized training in neurology or evaluation of GBS.

Additionally, Petitioner has presented evidence that supports Dr. Callaghan's contention that GBS cannot be diagnosed in the setting of a mimic, such as hyperkalemia, without first correcting the hyperkalemia. Petitioner filed a paper by Walling and Dickson, which states that to diagnose GBS, "other potential causes [of symptoms] must be excluded." Pet'r's Ex. 21 at 1. Dr. Jarvis admitted that "without the flu vaccine, hyperkalemia does mimic [GBS]" and that medical literature documents that hyperkalemia is a GBS mimic. Tr. 158:9–10, 142:11. Dr. Jarvis repeatedly emphasized his opinion that Mr. Bauer likely suffered from GBS due to his neuromuscular symptoms and the temporal relationship between Mr. Bauer's symptoms and flu vaccination. However, Petitioner has presented no evidence that a temporal relationship between symptoms and vaccinations is considered a diagnostic criterion for GBS. Despite his reliance on Mr. Bauer's symptoms, Dr. Jarvis acknowledged that "the weakness and numbness that [Mr. Bauer] was experiencing could be associated with hyperkalemia." Tr. 136:23–24. Petitioner filed multiple case reports of patients who experienced symptoms including paralysis, weakness, and paresthesias and who were assessed with hyperkalemia rather than GBS. *See generally* Pet'r's Ex. 18; Pet'r's Exs. 22–23. These are in addition to the case reports filed by Respondent of patients who experienced similar symptoms, including respiratory failure, and were initially thought to have GBS before ultimately being diagnosed with hyperkalemia instead. *See generally* Resp't's Exs. C–D. This evidence demonstrates that hyperkalemia can produce GBS-like symptoms and supports Dr. Callaghan's contention that GBS cannot be diagnosed in the setting of severe hyperkalemia.

Furthermore, although an EMG and lumbar puncture were not performed in this case due to Mr. Bauer's passing, there is no diagnostic testing in this case to support a GBS diagnosis. Petitioner has also not presented an opinion from a neurologist, whether a treating physician or an expert witness, supporting that a GBS diagnosis is appropriate in this case. Thus, I find that Petitioner has failed to present preponderant evidence that Mr. Bauer suffered from GBS.

B. *Loving* and *Althen*

In her amended petition, Petitioner alleged that Mr. Bauer suffered from injuries that were caused or significantly aggravated by his October 12, 2017 flu vaccination. Dr. Jarvis clarified during his testimony that these alleged injuries include hyperkalemia and acute kidney injury leading to worsening of preexisting CKD and, as a result, death. I find that Petitioner has not established by preponderant evidence that Mr. Bauer's flu vaccination caused him to suffer from these injuries.

1. Onset and *Loving* Prongs One, Two, and Three

a. Onset and *Loving* Prong One – Condition Prior to Vaccination

The parties agree that Mr. Bauer suffered from hyperkalemia and worsening of his CKD by October 20, 2017, but they dispute whether these changes likely occurred pre or post vaccination. Regarding Mr. Bauer's CKD, Dr. Jarvis noted that Mr. Bauer had CKD since about 2014 and that he had an eGFR of 31 and Stage 3b CKD by May 17, 2017, approximately five months before his October 12, 2017 flu vaccination. Tr. 138:10–13; Tr. 168:23–169:12. However, the medical records do not indicate that Mr. Bauer's eGFR was tested again before his October 20,

2017 hospitalization. Dr. Fine, Respondent's expert nephrologist, noted that Mr. Bauer had an eGFR of 56 on December 9, 2016, approximately five months before May 17, 2017. Resp't's Ex. G at 3. Dr. Fine asserted that Mr. Bauer's eGFR of 31 in May of 2017, indicating Stage 3b kidney disease or "moderately to severely decreased kidney function[,]" was a substantial decline from Mr. Bauer's baseline kidney function. *Id.* Dr. Fine explained that when Mr. Bauer presented to the hospital on October 20, 2017 with an eGFR of 18, he had Stage 4 CKD, or "severely decreased kidney function[]." *Id.* Dr. Fine noted that this further decline from May of 2017 "is consistent with a decline in kidney function that had begun earlier" in 2017. *Id.*

The National Kidney Foundation defines Stage 3a CKD, or "mild to moderate loss of kidney function[,]" as an eGFR of 45 to 59. Pet'r's Ex. 20 at 2. It identifies Stage 3b CKD, or a "moderate to severe loss of kidney function[,]" as an eGFR of 30 to 44. *Id.* The National Kidney Foundation further defines Stage 4 CKD, or "severe" CKD, as an eGFR between 15 and 29. *Id.* Mr. Bauer's medical records indicate that he had Stage 3a CKD on December 9, 2016. His eGFR of 56 on that date suggests that he was relatively close to the less severe Stage 2 CKD, according to the ranges identified by the National Kidney Foundation. Within approximately five months, by May 17, 2017, his eGFR dropped by twenty-five points, to only two points above Stage 4. Although Dr. Jarvis opined that Mr. Bauer's CKD appeared "pretty stable[,]" in May of 2017 due to his microalbumin creatinine ratio and normal microalbumin level, this is inconsistent with the marked decline in Mr. Bauer's eGFR between December of 2016 and May of 2017 and with Dr. Fine's opinion that Mr. Bauer was experiencing a decline in kidney function during this time. Tr. 168:17–18. Because Dr. Fine is a board-certified nephrologist with expertise in CKD and because it is unclear whether Dr. Jarvis has specialized training in nephrology, I grant Dr. Fine's opinions on CKD and nephrology greater weight than Dr. Jarvis's. Dr. Fine's opinion is also supported by Dr. Callaghan, who asserted that eGFR is the important number when considering the level of CKD, and the National Kidney Foundation, which uses eGFR rather than other factors to define CKD stages. On October 20, 2017, an additional approximate five months later, Mr. Bauer's eGFR dropped by another thirteen points, to 18. Due to the rate at which Mr. Bauer's eGFR dropped between December of 2016 and May of 2017, and his close proximity to Stage 4 CKD in May of 2017, it is unlikely that Mr. Bauer still had Stage 3b CKD on October 12, 2017. Indeed, Dr. Callaghan noted that it would not "take much" to decline from an eGFR of 31 to Stage 4 CKD. Tr. 196:22–24. Additionally, the National Kidney Foundation states that symptoms of later stage CKD can include muscle cramps and frequent urination. Pet'r's Ex. 20 at 1. It is thus notable that Mr. Bauer reported nocturia, although this was attributed to an unrelated condition, on May 18, 2017, and June 21, 2017, and leg cramps on June 21, 2017. Due to the progression of Mr. Bauer's eGFR and the medical literature and expert opinions indicating that Mr. Bauer was experiencing a decline in kidney function in 2017, I find by a preponderant standard, that Mr. Bauer had Stage 4 CKD or a severe loss in kidney function prior to his October 12, 2017 flu vaccination.

Although Respondent's experts contended that Mr. Bauer's hyperkalemia began weeks, or possibly months, before his October 12, 2017 flu vaccination, Dr. Jarvis denied that Mr. Bauer had symptoms of hyperkalemia prior to vaccination. Furthermore, Petitioner recalled in her affidavit and testimony that Mr. Bauer was in his normal state of health and was not experiencing symptoms such as weakness, pain, tingling, or difficulty walking before his vaccination. Drs. Callaghan and Fine cited Mr. Bauer's October 17, 2017 medical record from Dr. Jarvis indicating that his symptoms had started a "couple weeks" prior as evidence that Mr. Bauer's hyperkalemia predated

his vaccination. *See* Pet'r's Ex. 4 at 27. However, Dr. Jarvis disputed the accuracy of that medical record. Based on the overall record, I find by a preponderant standard that Mr. Bauer's hyperkalemia predated his October 12, 2017 flu vaccination.

Dr. Callaghan explained that hyperkalemia is a condition that can progress until a person reaches a threshold where he cannot tolerate it and experiences "a dramatic change[]" in symptoms. Tr. 257:20–23. Dr. Callaghan noted that the dramatic symptoms Mr. Bauer experienced on October 20, 2017, do not mean that he could not have had "subtler hyperkalemia predating that for quite a while." Tr. 221:7–14. Dr. Callaghan noted that the leg cramps Mr. Bauer reported in June of 2017 possibly indicate that he had hyperkalemia at that time. Tr. 203:12–17. Dr. Fine also explained that "[i]n order for potassium to get to [the level of 10.8 that Mr. Bauer had on October 20, 2017], it would have to occur over some time." Resp't's Ex. G at 7. Dr. Fine noted that a potassium level of over 5.2 is high and that levels as low as 6.5 may be life-threatening. *Id.* He noted that Mr. Bauer's potassium level of 10.8 on October 20, 2018, is "very consistent" with hyperkalemia existing for weeks prior. *Id.* Dr. Fine also explained that when patients experience a slow rise in potassium levels, they can tolerate "extreme elevation" of their potassium until "the potassium elevation [] result[s] in cardiac arrhythmia and death if it continues to increase." *Id.* I find Drs. Callaghan and Fine's opinions persuasive in light of their expertise and because Petitioner has not presented evidence to support that a person's potassium level could rise from a normal level of below 5.2 to a severely elevated level of 10.8 within eight days. Furthermore, Dr. Fine's assertions that potassium levels of 6.5 to 7 may be life-threatening but that individuals who experience a slow rise in levels can tolerate "extreme elevation" until it continues to increase could account for why Mr. Bauer did not experience cardiac arrest and respiratory failure until his potassium level was well-above 6.5 to 7. In addition, Dr. Jarvis acknowledged that it is possible that Mr. Bauer's hyperkalemia predated his vaccination and occurred before any clinical signs. Tr. 166:10–15. He also noted that onset of hyperkalemia can manifest differently depending on the patient and underlying medical issue and that he had observed patients with potassium levels over 8 who were asymptomatic. Tr. 147:2–13.

The October 17, 2017 medical record stating that Mr. Bauer had experienced symptoms for a "couple weeks" further supports that Mr. Bauer's hyperkalemia predated his October 12, 2017 vaccination. Pet'r's Ex. 4 at 27. However, Dr. Jarvis asserted that this record was "not as accurate a note as [he] would like it to be." Tr. 79:5–6. He explained that he "used that 'couple weeks' as a general term because [he] did[not] have the exact date of his symptoms when [he] dictated the note at the end of the day." Tr. 79:7–9. Dr. Jarvis noted that the October 20, 2017 record from the ER stated that Mr. Bauer's symptoms began on the first of the week, and Dr. Jarvis believed that this record was more accurate than the October 17, 2017 record. When asked why he believed the October 20, 2017 note to be more reliable, Dr. Jarvis testified that he "would state the sentinel moment was [Mr. Bauer] telling [Dr. Jarvis] when he had the flu shot, and then that kind of locked everything in." Tr. 79:19–24.

I find the contemporaneous October 17, 2017 record regarding onset overall more persuasive than the October 20, 2017 record and other testimony regarding onset. Where there are inconsistencies, special masters are within their discretion to award contemporaneous medical records greater weight than later conflicting testimony. *See Cucuras*, 993 F.2d at 1528 (holding that the special master's reliance on contemporaneous medical records over conflicting oral

testimony given after the fact was not arbitrary or capricious); *see also Burns v. Sec'y of Health & Hum. Servs.*, 3 F.3d 415, 417 (Fed. Cir. 1993) (holding that the decision of whether to accord greater weight to contemporaneous medical records or later given testimony is "uniquely within the purview of the special master"). Although Dr. Jarvis asserted that the October 17, 2017 record was inaccurate regarding onset, he did not recall when he discovered this inaccuracy. He also dictated twice on October 17, 2017, that Mr. Bauer's symptoms "really started hitting him in the last couple of weeks[]" and "just occurred in the last few weeks." Pet'r's Ex. 4 at 27. However, Dr. Jarvis did not persuasively explain how he remembered years later that a "couple" or a "few" weeks was an estimate rather than an accurate description of what Mr. Bauer reported. When asked why he believed the October 20, 2017 record to be more reliable, Dr. Jarvis noted that this is when Dr. Jarvis learned about the flu vaccination. However, Mr. Bauer's mention of the flu vaccination does not indicate whether Mr. Bauer reported experiencing symptoms for weeks or days on October 17, 2017. Furthermore, the October 20, 2017 record states that Mr. Bauer's symptoms began on the first day of the week encompassing October 20, 2017, which would be Sunday, October 15, 2017 or Monday, October 16, 2017. *See* Pet'r's Ex. 5 at 305. This is inconsistent with the testimony of Petitioner and Ms. Toth, who both recalled observing Mr. Bauer's symptoms on October 13, 2017, during their trip to the zoo. The October 17, 2017 medical record was also created closer-in-time to the onset of symptoms.

Because the record contains preponderant evidence that Mr. Bauer's hyperkalemia predated his vaccination, Petitioner cannot establish that it was caused-in-fact by the vaccination. *See W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1358 (Fed. Cir. 2013) ("If a petitioner has a disorder before being vaccinated, the vaccine logically cannot have caused the disorder.").

b. *Loving* Prong Two – Post-Vaccination Condition

The record contains preponderant evidence that Mr. Bauer continued to experience hyperkalemia and to have Stage 4 CKD between his vaccination and death on October 20, 2017. It is undisputed that Mr. Bauer's symptoms worsened between his vaccination and death. Based on Dr. Callaghan and Dr. Fine's explanations for how individuals can tolerate hyperkalemia until it passes a certain threshold, I find that the record contains preponderant evidence that Mr. Bauer's hyperkalemia worsened following his vaccination and before he experienced cardiac arrest and respiratory failure. It is, however, unclear whether his CKD worsened between October 12 and October 20, 2017. Dr. Jarvis asserted that Mr. Bauer experienced an acute kidney injury that worsened his CKD, but the record does not contain preponderant evidence that an acute kidney injury occurred post vaccination. Dr. Callaghan explained that it is difficult to tell whether Mr. Bauer experienced an acute kidney injury because his kidney function had not been tested since May of 2017. Tr. 241:2–4. It is especially difficult to assess whether or when Mr. Bauer experienced an acute kidney injury leading to a decline in kidney function given that the medical records support that he was experiencing worsening CKD since at least May of 2017. I find that the record does not contain preponderant evidence that Mr. Bauer's CKD worsened between October 12 and October 20, 2017.

c. *Loving* Prong Three – Significant Aggravation

Because the record does not contain preponderant evidence that Mr. Bauer's CKD worsened following his vaccination, Petitioner cannot establish that Mr. Bauer experienced a significant aggravation of his CKD. Although Mr. Bauer's hyperkalemia worsened after October 12, 2017, Petitioner has not presented preponderant evidence that this worsening constitutes a significant aggravation of his CKD. Dr. Fine persuasively explained that an elevated potassium level can continue to increase until it causes cardiac arrhythmia and death, and he opined that this is what occurred in this case. Resp't's Ex. G at 7. Petitioner has not rebutted Dr. Fine's explanation of how hyperkalemia progresses.

Petitioner has not demonstrated by preponderant evidence that Mr. Bauer suffered from GBS. I have also determined that the record does not contain preponderant evidence that Mr. Bauer suffered from an acute kidney injury or worsening CKD following vaccination. Although I have found that Mr. Bauer's hyperkalemia worsened post vaccination, Petitioner has failed to establish by preponderant evidence that this worsening constitutes a significant aggravation. Thus, Petitioner has not proven by preponderant evidence that any of the injuries Mr. Bauer allegedly suffered that caused his cardiac arrest, respiratory failure, and death were caused or significantly aggravated by Mr. Bauer's October 12, 2017 flu vaccination. Accordingly, Petitioner's claim must fail. *See Broekelschen*, 618 F.3d at 1346 ("[A] careful reading of *Althen*[] shows that each prong of the *Althen* test is decided relative to the injury.").

2. *Loving* Prongs Four–Six

Although Petitioner's claim fails based on the analysis above, I will address the remaining *Loving*/*Althen* prongs. I find that Petitioner has not established a medical theory, a logical sequence of cause and effect, or a medically-acceptable temporal relationship pursuant to the remaining prongs by preponderant evidence.

a. *Loving* Prong Four/*Althen* Prong One – Medical Theory

Dr. Jarvis presented three possible mechanisms: (1) "[p]ossibl[e] elevation of myoglobin[,] . . . which can occur with [GBS], which could impact his filtration in his kidney and shut it down," (2) "the antibody [] cross-reacting with the myelin sheath," and (3) "an antibody from the flu vaccine injuring the kidney." Tr. 104:10–15. Regarding how an acute kidney injury could worsen preexisting CKD, Dr. Jarvis stated that "with the cross-reactivity, with the antibodies, the . . . autoimmune response, attacking his kidneys, shuts them down, and then you have acute kidney injury worsening his chronic kidney disease." Tr. 99:24–100:4. When asked to clarify what biological mechanism he thought most likely occurred in this case, Dr. Jarvis stated that Mr. Bauer's symptoms were most likely caused by GBS. Tr. 165:15–17. When asked to explain how a flu vaccination caused Mr. Bauer to suffer from GBS, Dr. Jarvis stated that "an immune response to the vaccine was generated, and then he had, unfortunately, an autoimmune response where the antibody attacked his own body." Tr. 95:14–18. Dr. Jarvis stated that after an autoimmune response due to GBS, "there[is] no way of knowing [whether] the antibody also cross-react[ed] with the . . . nephrin [sic] in the kidney[or] the glomeruli[]" or whether it resulted in kidney shutdown and failure. Tr. 160:4–10. He explained that "[h]is point is that [GBS] can progress and

there can be cross-reactivity with the antibodies that are produced to the myelin sheath with [GBS] that causes the neuromuscular issues, and they can also cross-react with the glomeruli and the kidneys." Tr. 92:10–15.

Petitioners are not required to present specific biological mechanisms to prevail, but they must present enough explanation for a special master to determine whether there is a sound and reliable mechanism linking the vaccine received with the injuries suffered. Petitioner has not provided sufficient information for me to evaluate whether the mechanisms are sound and reliable. For instance, Dr. Jarvis opined that, in this case, the flu vaccine caused GBS because an immune response to the vaccine resulted in an autoimmune attack, and he indicated that that this autoimmune attack may affect the myelin sheath. He did not provide an explanation for how a general immune response generated by the flu vaccine can lead to an autoimmune attack specifically against the myelin sheath rather than against other parts of the body. Although GBS is a Table injury for the flu vaccine, petitioners have the burden to present medical theories pursuant to *Loving* prong four/*Althen* prong one to prevail on off-Table claims. Similarly, Dr. Jarvis did not provide a general explanation for how GBS could cause myoglobin elevation resulting in kidney injury or how GBS could result in an autoimmune attack on the kidneys. He mentioned rhabdomyolysis in the context of a case of GBS he observed in medical school, but it is unclear how that applies to this case. He also did not provide a general explanation for how an antibody from the flu vaccine could injure the kidneys or cross-react with a component of the kidneys to cause injury. While Petitioner filed some medical literature linking GBS and kidney injury, Dr. Callaghan explained that these conditions are not "tightly linked." Tr. 254:20–25. He explained that kidney injury in the context of GBS usually occurs when a patient experiences autonomic instability and blood pressure problems during a long hospitalization and illness. Tr. 242:24–243:4. Dr. Callaghan's position is consistent with the Khajehdehi et al. paper, in which the authors linked the development of acute kidney failure in the context of GBS to damage caused by "hypotensive crises" rather than GBS itself. *See* Pet'r's Ex. 14 at 1, 3. Furthermore, Petitioner has not presented a medical theory for how the flu vaccine could cause or worsen hyperkalemia in the absence of GBS or a post-vaccination kidney injury. Dr. Fine asserted that "[t]here is no scientific basis to th[e] hypothesis[]" linking a flu vaccination and hyperkalemia. Resp't's Ex. G at 7. I find that Petitioner has not presented preponderant evidence of a medical theory pursuant to *Loving* prong four/*Althen* prong one.

b. *Loving* Prong Five/*Althen* Prong Two – Actual Causation

Petitioner has not provided preponderant evidence of a logical sequence of cause and effect between the flu vaccine that Mr. Bauer received and the alleged kidney injury and hyperkalemia that he suffered. Dr. Jarvis emphasized throughout his testimony that his opinion that the flu vaccination caused Mr. Bauer's injuries is based on the temporal relationship between Mr. Bauer's symptoms and the flu vaccine as the "sentinel event." *See, e.g.*, Tr. 92:22–93:1, 103:20–25, 121:19–122:14, 149:18–150:3. When asked to clarify whether his opinion on causation was based solely on timing, Dr. Jarvis again noted the timing between the vaccination and symptoms, indicating that his opinion was based on timing "and everything [he] saw that came with it." Tr. 150:3. However, it is well established in the Program that temporal proximity between a vaccination and injury is insufficient to support causation. *Moberly*, 592 F.3d at 1323 (quoting *Althen*, 418 F.3d at 1278) ("[N]either a mere showing of a proximate temporal relationship

between vaccination and injury, nor a simplistic elimination of other potential causes of the injury suffices, without more, to meet the burden of showing actual causation."); *Sumner v. Sec'y of Health & Hum. Servs.*, No. 99-946V, 2015 WL 5173644, at *9 (Fed. Cl. Spec. Mstr. Aug. 13, 2015) ("[W]here a petitioner's expert views the temporal relationship as the 'key' indicator of causation, the claim must fail."). Dr. Jarvis did not clearly describe what he meant by "everything" that came with the timing of Mr. Bauer's vaccine. As such, Petitioner is unable to establish actual causation by a preponderance of the evidence.

Furthermore, the record contains preponderant evidence that factors unrelated to Mr. Bauer's vaccination caused Mr. Bauer's worsening CKD and his hyperkalemia and, therefore, his death. Dr. Fine persuasively explained that Mr. Bauer's CKD was caused by his "long-standing diabetes, microvascular disease[,] and hypertension." Resp't's Ex. G at 7. Dr. Fine opined that Mr. Bauer's medications, including an ACE-inhibitor, likely contributed to his declining kidney function. *Id.* Noting "the drop in [Mr. Bauer's] GFR by May of 2017[,]" Dr. Fine explained that Mr. Bauer "was suffering from progressive kidney disease, and there appeared to be no intervention that would reverse the course." *Id.* Dr. Fine stated that Mr. Bauer's death was "due to severe hyperkalemia in the context of worsening kidney function and multiple factors that increase blood creatinine." *Id.* at 1. These factors included Mr. Bauer's ACE-inhibitor, Lotrel; aldosterone inhibitor, spironolactone; and potassium supplementation. *Id.* Dr. Fine also noted that the medical records indicate that Mr. Bauer was taking a beta-blocker, which can also contribute to hyperkalemia risk. *Id.* at 2. Dr. Fine noted that a decline in kidney function "compromises the ability of the kidney to excrete potassium. *Id.* at 7. Drs. Fine and Callaghan noted that spironolactone can increase potassium levels because it is a potassium-sparing diuretic, and Dr. Callaghan explained that treatment of hypokalemia with potassium supplementation can lead to hyperkalemia. *Id.* at 3; Tr. 195:2–4, 261:1–3. Although Dr. Fine noted that Mr. Bauer previously had low potassium levels, his potassium levels were likely to rise due to "ongoing kidney function decline." Resp't's Ex. G at 7. Dr. Fine explained that this rise was exacerbated by the ACE-inhibitor, which reduces aldosterone, "the primary hormone involved in potassium excretion in the kidneys," and spironolactone, which inhibits aldosterone. *Id.* I find Drs. Fine and Callaghan's explanations persuasive in light of their expertise regarding CKD and hyperkalemia.

Although Dr. Jarvis contended that hyperkalemia due to spironolactone would likely occur within the first weeks of spironolactone treatment, this does not take into account the potential for progressive elevation of potassium levels, Mr. Bauer's other medications, or his decline in kidney function revealed in May of 2017. In addition, Petitioner filed one case report, by Udezue and Harrold, in which a patient experienced hyperkalemic paralysis after two years of spironolactone treatment. *See* Pet'r's Ex. 23 at 1. Petitioner has not presented preponderant evidence that Mr. Bauer's flu vaccination caused, significantly aggravated, or contributed to his conditions or death.

c. *Loving* Prong Six/*Althen* Prong Three – Temporal Relationship

Petitioner has not presented evidence of what a medically acceptable temporal relationship to infer causation between a flu vaccination and worsening hyperkalemia, acute kidney injury, or worsening CKD would be. Further, Petitioner has not presented preponderant evidence that Mr. Bauer experienced an acute kidney injury or worsening CKD following his vaccination. Thus, Petitioner has presented insufficient evidence to fulfill this prong.

VI. Conclusion

After a careful review of the record, Petitioner has failed to prove by preponderant evidence that Mr. Bauer experienced Table GBS or injuries that were caused or significantly aggravated by his October 12, 2017 flu vaccination. Accordingly, I **DENY** Petitioner's claim and **DISMISS** her petition.[85]

IT IS SO ORDERED.

s/Herbrina D. Sanders
Herbrina D. Sanders
Special Master

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