

In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 19-929V

Filed: September 3, 2025

ABBY ADAMS, *Administrator of the
Estate of DONALD C. ADAMS,*

Petitioner,

v.

SECRETARY OF HEALTH AND
HUMAN SERVICES,

Respondent.

Special Master Horner

*Phyllis Widman, Widman Law Firm, LLC, Linwood, NJ, for petitioner.
Alexa Roggenkamp, U.S. Department of Justice, Washington, DC, for respondent.*

DECISION¹

On June 26, 2019, petitioner, Abby Adams, filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10-34 (2012),² on behalf of her late father, Donald Adams, alleging that he suffered cardiac arrest and anoxic brain injury post-cardiac arrest as a result of the pneumococcal and influenza (“flu”) vaccines that he received on October 11, 2018.³ (ECF No. 1.) Alternatively, petitioner argues that

¹ Because this document 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 document 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 Within this decision, all citations to § 300aa will be the relevant sections of the Vaccine Act at 42 U.S.C. § 300aa-10-34.

³ The decedent's vaccine administration record confirmed that his pneumococcal vaccination was a “Pneumovax 23” vaccine rather than a “Prevnar” vaccine. However, only pneumococcal conjugate vaccines are covered by the Vaccine Injury Table, 42 C.F.R. § 100.3(a)(XII), and the Pneumovax 23 vaccine is a polysaccharide rather than conjugate vaccine, *e.g.*, *Morrison v. Sec'y of Health & Human Servs.*, No. 04-1683V, 2005 WL 2008245 (Fed. Cl. Spec. Mstr. July 26, 2005). Accordingly, this case turns on whether petitioner can implicate the flu vaccine in particular as a cause of her father's death.

the pneumococcal and flu vaccines significantly aggravated a pre-existing condition, leading to Mr. Adams's untimely death. (*Id.*) Petitioner clearly suffered a profound loss, and I offer my sincerest condolences. However, for the reasons set forth below, I conclude that petitioner is *not* entitled to an award of compensation.

I. Applicable Statutory Scheme

Under the National Vaccine Injury Compensation Program, compensation awards are made to individuals who have suffered injuries after receiving vaccines. In general, to gain an award, a petitioner must make a number of factual demonstrations, including showing that an individual received a vaccination covered by the statute; received it in the United States; suffered a serious, long-standing injury or death; and has received no previous award or settlement on account of the injury. Finally – and the key question in most cases under the Program – the petitioner must also establish a *causal link* between the vaccination and the injury. In some cases, the petitioner may simply demonstrate the occurrence of what has been called a “Table Injury.” That is, it may be shown that the vaccine recipient suffered an injury of the type enumerated in the “Vaccine Injury Table,” corresponding to the vaccination in question, within an applicable time period following the vaccination also specified in the Table. If so, the Table Injury is presumed to have been caused by the vaccination, and the petitioner is automatically entitled to compensation, unless it is affirmatively shown that the injury was caused by some factor other than the vaccination. § 300aa-13(a)(1)(A); § 300aa-11(c)(1)(C)(i); § 300aa-14(a); § 300aa-13(a)(1)(B).

Alternatively, if no injury falling within the Table can be shown, a petitioner could still demonstrate entitlement to an award by instead showing that the vaccine recipient's injury or death was caused-in-fact by the vaccination in question. § 300aa-13(a)(1)(A); § 300aa-11(c)(1)(C)(ii). In particular, a petitioner must demonstrate that the vaccine was “not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury.” *Moberly v. Sec’y of Health & Human Servs.*, 592 F.3d 1315, 1321-22 (Fed. Cir. 2010) (quoting *Shyface v. Sec’y of Health & Human Servs.*, 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); *Pafford v. Sec’y of Health & Human Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006). To successfully demonstrate causation-in-fact, petitioner bears a burden to show: (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 proximate temporal relationship between vaccination and injury.⁴ *Althen v. Sec’y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005).

⁴ Additionally, where a petitioner in an off-Table case is seeking to prove that a vaccination significantly aggravated a pre-existing injury, the petitioner must establish the three *Althen* prongs along with three additional factors described in the prior *Loving* case. See *Loving ex rel. Loving v. Sec’y of Health & Human Servs.*, 86 Fed. Cl. 135, 144 (2009) (combining the first three *Whitcotton* factors for claims regarding aggravation of a Table injury with the three *Althen* factors for off table injury claims to create a six-part test for off-Table aggravation claims); see also *W.C. v. Sec’y of Health & Human Servs.*, 704 F.3d 1352, 1357 (Fed. Cir. 2013) (applying the six-part *Loving* test). The additional *Loving* factors require petitioners to demonstrate aggravation by showing: (1) the vaccinee's condition prior to the administration

In this case, petitioner has not alleged any condition listed on the Vaccine Injury Table. Accordingly, petitioner must satisfy the above-described *Althen* test for establishing causation-in-fact.

Vaccine Program petitioners bear a “preponderance of the evidence” burden of proof. § 300aa-13(1)(a). That is, a petitioner must offer evidence that leads the “trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact’s existence.” *Moberly*, 592 F.3d at 1322 n.2 (alternation in original); see also *Snowbank Enters., Inc. v. United States*, 6 Cl. Ct. 476, 486 (1984) (explaining that mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec’y of Health & Human Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In finding causation, a program fact-finder may rely upon “circumstantial evidence,” which the court found to be consistent with the “system created by Congress, in which close calls regarding causation are resolved in favor of injured claimants.” *Althen*, 418 F.3d at 1279-80. However, a petitioner may not receive a Vaccine Program award based solely on her assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. § 300aa-13(a)(1). The supporting medical opinion must be based on “sound and reliable” medical or scientific explanation. *Boatmon v. Sec’y of Health & Human Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019).

Cases in the Vaccine Program are assigned to special masters who are responsible for “conducting all proceedings, including taking such evidence as may be appropriate, making the requisite findings of fact and conclusions of law, preparing a decision, and determining the amount of compensation, if any, to be awarded.” Vaccine Rule 3(b)(1). Special masters must ensure each party has had a “full and fair opportunity” to develop the record. Vaccine Rule 3(b)(2). However, special masters are empowered to determine the format for taking evidence based on the circumstances of each case. Vaccine Rule 8(a); Vaccine Rule 8(d). Special masters are not bound by common law or statutory rules of evidence but must consider all relevant and reliable evidence in keeping with fundamental fairness to both parties. Vaccine Rule 8(b)(1). The special master is required to consider “all [] relevant medical and scientific evidence contained in the record,” including “any diagnosis, conclusion, medical judgment, or autopsy or coroner’s report which is contained in the record regarding the nature, causation, and aggravation of the petitioner’s illness, disability, injury, condition, or death,” as well as the “results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions.” § 300aa-13(b)(1)(A). The special master is required to consider the entire record, draw plausible inferences, and articulate a rational basis for the decision. *Winkler v. Sec’y of Health & Human Servs.*, 88 F.4th 958, 963 (Fed. Cir. 2023) (citing *Hines ex rel. Sevier v. Sec’y of Health & Human Servs.*, 940 F.2d 1518, 1528 (Fed. Cir. 1991)).

of the vaccine, (2) the vaccinee’s current condition, and (3) whether the vaccinee’s current condition constitutes a “significant aggravation” of the condition prior to the vaccination. *Loving*, 86 Fed. Cl. at 144.

II. Procedural History

Petitioner filed medical records marked Exhibits 1-6 between July and October of 2019. (ECF Nos. 7, 14.) Respondent filed his Rule 4 report on June 11, 2020, recommending against compensation. (ECF No. 22.) Respondent asserted that none of Mr. Adams's treating physicians definitively associated his condition with his vaccination. (*Id.* at 10-12.) Respondent further asserted that petitioner had not presented any evidence regarding the natural course of any pre-existing condition or any evidence to support significant aggravation of a pre-existing condition.⁵ (*Id.* at 12-13.)

In August of 2020, petitioner filed additional medical records, marked Exhibit 7. (ECF No. 25.) Thereafter, in May of 2022, petitioner filed an expert report by cardiac electrophysiologist Dr. Mayer Rashtian, along with accompanying medical literature. (ECF Nos. 39, 41; Exs. 8-18.) On December 14, 2022, petitioner filed additional medical records, marked Exhibit 19. (ECF No. 48.) Respondent subsequently filed records from Carlisle Fire Company, marked Exhibit C.⁶ (ECF No. 67.) Respondent also filed an expert report by cardiologist Dr. Shane J. LaRue, along with accompanying medical literature. (ECF No. 62; Ex. A-B.) Thereafter, petitioner filed a supplemental expert report by Dr. Rashtian. (ECF No. 64, Ex. 21.) Petitioner also filed updated medical records, marked Exhibit 22, on December 12, 2023. (ECF No. 68.) Respondent responded to Dr. Rashtian's supplemental report with a supplemental expert report by Dr. LaRue, along with accompanying medical literature. (ECF No. 72; Ex. D.) Finally, petitioner filed medical records marked as Exhibit 23 in May of 2024. (ECF No. 75.)

An entitlement hearing was held on October 23, 2024. (See Transcript of Proceedings ("Tr."), at ECF No. 93.) During the hearing, petitioner testified as well as Drs. Rashtian and LaRue. (See *id.*) The parties opted not to file post-hearing briefs. (ECF No. 91.) Accordingly, the parties have had a full and fair opportunity to develop the record, and this case is now ripe for resolution.

⁵ Additionally, respondent initially questioned whether administration of the subject vaccines was preponderantly established (ECF No. 22, pp. 9-10); however, he later stipulated that the decedent received the vaccines as alleged (ECF No. 81).

⁶ On December 19, 2022, petitioner filed a Statement of Unavailability of Records, detailing counsel's efforts to locate outstanding medical records. (ECF No. 50.) Petitioner indicated that she had been unable to locate the EMS transport records. (*Id.*) Thereafter, respondent was provided an opportunity to pursue additional avenues of discovery pursuant to Vaccine Rule 7. (Non-PDF Scheduling Order, filed Dec. 21, 2022.) Respondent indicated his intent to file a motion for leave to issue a subpoena for the outstanding records and requested that petitioner file all billing records in connection with Mr. Adams' treatment on October 12, 2018, to assist in his search for the EMS provider to which a subpoena may be issued. (ECF Nos. 51-52.) Petitioner filed the requested billing records on March 23, 2023. (ECF No. 53.) On April 25, 2023, respondent filed two motions, requesting authorization to issue subpoenas to Carlisle Fire Co., Inc. and Hart to Heart Transportation, Inc., respectively, which were subsequently granted. (ECF Nos. 56-59.)

III. Factual History

a. As reflected in the medical records

i. Pre-vaccination

Mr. Adams was born on July 29, 1949. (Ex. 2; Ex. 7, p. 2.) At the time of vaccination, he was sixty-nine years old. (Ex. 1, p. 1; Ex. 2; Ex. 7, p. 2.) His past medical history was significant for diabetes mellitus, hypertension, hyperlipidemia, atypical chest pain, and cigarette smoking. (See *generally* Ex. 4.)

Mr. Adams underwent an echocardiogram in 2002 that revealed concentric left ventricular hypertrophy with normal cavity size and preserved systolic function, mild mitral regurgitation, dilated left atrium, and mild tricuspid regurgitation. (Ex. 4, p. 199.) At sixty-one years of age, Mr. Adams underwent an electrocardiogram (“EKG”) that showed sinus rhythm with occasional premature ventricular contractions (“PVCs”) but was otherwise normal. (*Id.* at 198.)

On May 3, 2012, Mr. Adams presented to his cardiologist with complaints of a two-month history of intermittent, left-sided chest pain when coughing. (Ex. 4, p. 175.) A physical examination and review of his recent laboratory results were unremarkable. (*Id.* at 175-78.) His cardiologist’s assessed Mr. Adams with chest pain, hypertension, hyperlipidemia, diabetes mellitus, tobacco use, and morbid obesity. (*Id.* at 177-78.) It was noted that he underwent a stress test in 2010, and he reported that the results were “fine;” however, there were “[n]o results to review.” (*Id.* at 178.) He was advised to undergo a repeat stress test, which he completed on May 31, 2012. (*Id.* at 171, 178.) The results of the stress test showed no evidence of dipyridamole induced myocardial ischemia or scar. (*Id.* at 171.) Left ventricular ejection fraction was normal at 61%. (*Id.*)

Mr. Adams presented to the emergency department on February 13, 2017, with complaints of a three-day history of numbness and tingling in his right upper extremity. (Ex. 23, p. 7.) He also reported two days of tingling in his face that had since resolved. (*Id.*) He denied headache, speech/vision changes, weakness, chest pain, shortness of breath, nausea, vomiting, diarrhea, fever, and chills. (*Id.*) A physical examination was normal with the exception of elevated blood pressure. (*Id.* at 8-9.) An EKG revealed abnormal conduction with an incomplete right bundle branch block and frequent PVCs. (*Id.* at 9.) Imaging studies of Mr. Adams’s head, brain, and neck were unremarkable with the exception of observed atrophy with chronic small vessel ischemic change, which was seen on CT scan of petitioner’s head without contrast. (*Id.* at 12-13.) Although it was recommended that Mr. Adams be admitted for a full stroke work up, he ultimately opted to be discharged home. (*Id.* at 13-14.)

Throughout 2017, Mr. Adams presented to his primary care physician for a nodule in the right lung, tobacco use, hypertension, strain of the right supraspinatus

tendon, adjustment disorder with mixed anxiety and depressed mood, and type 2 diabetes mellitus with hyperlipidemia. (Ex. 4, pp. 105-35.)

On January 12, 2018, Mr. Adams presented to his primary care provider and reported increased and worsening episodes of chest palpitations. (Ex. 4, p. 96.) He described a “buzzing feeling” in his heart and associated fatigue, dizziness, and vision changes. (*Id.*) A physical exam was unremarkable. (*Id.* at 99.) The differential diagnosis mentioned Mr. Adams’s chronic hypertension and diabetes mellitus. (*Id.*) It was also noted that Mr. Adams had recently quit smoking and was experiencing some exertional dyspnea and atypical chest pain with intermittent cardiac arrhythmia for one month. (*Id.*) An EKG, echocardiogram, and Holter monitor were ordered, and Mr. Adams was referred to a cardiologist. (*Id.*) Despite the study being technically difficult with many images being suboptimal in quality, a January 16, 2018 echocardiogram showed diastolic dysfunction, trace mitral regurgitation, and trace tricuspid regurgitation. (*Id.* at 93-95.) Mr. Adams had “low normal” left ventricular systolic function with a left ventricular ejection fraction of 50-55%. (*Id.* at 93.)

On January 25, 2018, Mr. Adams saw cardiologist Laeeq Ahmer, M.D., for an evaluation and management of palpitations, shortness of breath, and coronary artery disease. (Ex. 4, pp. 54-59.) A history of hypertension, diabetes, and hyperlipidemia, as well as coronary artery calcification on a prior CT scan of the chest, was noted. (*Id.* at 54.) Mr. Adams reported intermittent palpitations, which had persisted for several months, and brief heart flutters, which typically lasted only seconds. (*Id.*) Although he had been wearing a Holter monitor, he had not experienced any palpitations in the last few days. (*Id.*) Additionally, he was not complaining of chest pain but noted exertional dyspnea for the past several months. (*Id.*) His blood pressure was mildly elevated during this encounter but otherwise well controlled with Coreg, amlodipine, and lisinopril. (*Id.*) His lipids were also well controlled on Lipitor. (*Id.*) A physical examination was normal. (*Id.* at 57.) Dr. Ahmer diagnosed Mr. Adams with coronary artery disease involving native coronary artery of native heart without angina pectoris and ordered a stress test with myocardial perfusion SPECT. (*Id.* at 58.) He further diagnosed palpitations, shortness of breath, hypertension, and hyperlipidemia. (*Id.* at 58-59.) Mr. Adams underwent a stress test on January 31, 2018, which showed a medium-sized, partially reversible defect in the inferolateral wall consistent with mild ischemia as well as mildly depressed left ventricle function. (*Id.* at 44-45.)

Mr. Adams followed up with Dr. Ahmer on February 9, 2018, to discuss the results of his stress test and Holter monitor. (Ex. 4, pp. 38-43.) A physical exam was normal. (*Id.* at 41.) His mild exertional dyspnea and blood pressure were stable during this encounter. (*Id.* at 38.) Mr. Adams noted that his palpitations had gradually resolved. (*Id.*) While his Holter monitor uncovered very frequent premature ventricular contractions, Mr. Adams did not notice any palpitations during monitoring. (*Id.* at 38, 42.) He also had frequent ventricular triplets and runs of nonsustained ventricular tachycardia. (*Id.* at 42.) Dr. Ahmer increased his prescription for Coreg and recommended an ischemic workup and a repeat Holter monitor. (*Id.*) Regarding Mr. Adams’ abnormal stress test, Dr. Ahmer was concerned that he might have silent

ischemia and suggested that he undergo a cardiac catheterization for further evaluation. (*Id.* at 42-43.) Mr. Adams underwent a cardiac catheterization on February 15, 2018, which showed mild, non-obstructive coronary artery disease by angiography, coronary artery calcification, and left ventricular ejection fraction of 50%. (*Id.* at 36-37.) He followed up with Dr. Ahmer on March 8, 2018, and was noted to be doing well on an increased dose of Coreg with no further palpitations noted. (*Id.* at 28-33.) An EKG performed on that day showed normal sinus rhythm without premature ventricular contractions and did not show acute ischemia. (*Id.* at 32.) Mr. Adams denied chest pain and his lipids and blood pressure were well controlled. (*Id.* at 31-33.)

On June 8, 2018, Mr. Adams returned to Dr. Ahmer, and it was noted that he was doing well with only infrequent premature ventricular contractions and no palpitations. (Ex. 5, pp. 224-5, 229.) He presented to endocrinologist James Hays, M.D., on July 30, 2018, for management of his diabetes. (Ex. 6, pp. 2-5.) Although it was noted that Mr. Adams had quit smoking approximately one year prior, he was receiving three insulin injections per day and his glucose monitoring was still labile. (*Id.* at 5.)

On October 11, 2018, Mr. Adams presented to his primary care provider for a prescription to control his blood sugar and a referral for a new endocrinologist. (Ex. 4, p. 1.) He described his diabetes as worsening due to diet noncompliance and missed medication and being associated with nocturia, peripheral neuropathy, and weight gain. (*Id.* at 5.) His hypertension was unchanged and associated with fatigue and vision changes. (*Id.* at 5-6.) A physical exam was normal. (*Id.* at 6-7.) During this encounter, Mr. Adams received the subject flu and pneumococcal 23-polyvalent vaccines. (*Id.* at 7; Ex. 1; Ex. 7.) On the flu vaccine consent form, Mr. Adams denied that he “ever had a serious allergic reaction to a previous dose of flu vaccine.” (Ex. 1, p. 2.) Mr. Adams’s primary care physician referred him to an endocrinologist and deferred insulin management to his new specialist. (Ex. 4, p. 7.) Mr. Adams was also prescribed empagliflozin⁷ for management of his diabetes in the interim. (*Id.*)

ii. Post-vaccination

At around 8:00 p.m. on the day of the vaccination at issue, Mr. Adams complained that he was tired and decided to watch television in bed. (Ex. 23, p. 161; see also Ex. 5, p. 262.) At around 2:30 a.m. on October 12, 2018, we awoke not feeling well. (Ex. 23, p. 161.) He was feeling hot and diaphoretic.⁸ (*Id.*) He called his daughter before dialing 911. (*Id.*)

At approximately 3:39 a.m., Emergency Medical Services (“EMS”) was dispatched to Mr. Adams’s home due to reports that he was experiencing difficulty breathing and a fever. (Ex. C, pp. 2-3.) It was noted that Mr. Adams had received “2

⁷ Empagliflozin, which is the generic name for Jardiance, is commonly used to help lower blood sugar in patients with type 2 diabetes. *Jardiance (Empagliflozin) – Uses, Side Effects, and More*, WEBMD (June 18, 2024), <https://www.webmd.com/drugs/2/drug-166762/jardiance-oral/details>.

⁸ Diaphoretic pertains to or is characterized by sweating. *Diaphoretic*, DORLAND’S MEDICAL DICTIONARY ONLINE, <https://www.dorlandsonline.com/dorland/definition?id=13777> (last visited Aug. 26, 2025).

shots this morning and believed that he was having a reaction to the shots.” (*Id.* at 3.) Upon arrival, Mr. Adams was lying on the floor and was helped to a sitting position in a chair. (*Id.*) It was noted that he “stood right up without any problems.” (*Id.*) Although Mr. Adams was alert and oriented, his breathing was shallow. (*Id.*) While attempting to take Mr. Adams’s vitals, he collapsed in his chair. (*Id.*; Ex. 23, 135.) Mr. Adams went into agonal breathing and pulseless electrical activity arrest. (Ex. 23, p. 152.) Mr. Adams was lowered to the ground, and his pulse was noted to be “no longer strong and beating adequately.” (Ex. C, p. 3.) He was administered several rounds of epinephrine and resuscitated by CPR. (Ex. 5, p. 262; Ex. 23, pp. 133, 135, 152.) It was noted that EMS did not use an automated external defibrillator. (Ex. C, p. 3.) Mr. Adams “regained pulses and then lost them a short while later.” (*Id.*; Ex. 23, p. 135.) He was then transported to the hospital by ambulance. (Ex. C, p. 3; Ex. 23, p. 133.)

During transport, Mr. Adams’s pulse was restored, and he began “breathing on his own slightly.” (Ex. C, p. 3; Ex. 23, pp. 133, 135.) However, it was noted that Mr. Adams never regained consciousness. (Ex. 23, p. 161.) Upon arrival at the hospital at around 5:08 a.m., Mr. Adams was unresponsive with an oropharyngeal airway through mouth. (*Id.* at 135-37.) Mr. Adams’s wife informed the emergency department physician that Mr. Adams had received the flu and pneumococcal vaccines earlier that day and questioned whether the vaccines could have caused Mr. Adams’s condition. (*Id.* at 135.) Mr. Adams was diagnosed with cardiac arrest, hyperglycemia, and metabolic acidosis with respiratory acidosis. (*Id.* at 133.) Mr. Adams was subsequently transferred to the intensive care unit and placed on a ventilator. (*Id.* at 147, 159.) In the admission note, Mr. Adams’s diagnoses were pulseless electrical activity (“PEA”) cardiac arrest of unknown etiology and lactic acidosis secondary to cardiac arrest. (*Id.* at 159.)

Throughout the morning of October 12, 2018, Mr. Adams underwent a CT of his head without contrast, a CT angiogram of his chest with contrast, and an x-ray of his chest. (Ex. 23, pp. 142-45.) In pertinent part, the CT angiogram showed “[s]mall areas of ill-defined reticulonodular infiltrate . . . involving the right upper lobe, likely inflammatory of infectious,” and the x-ray of the chest showed no acute cardiopulmonary process. (*Id.* at 143-44.) Mr. Adams’s lab results showed an elevated white blood count of 19.3 K/ μ L (reference range of 4.5-11.0 K/ μ L) with 61.0% neutrophils (reference range of 50.0-60.0 %). (*Id.* at 140-41.)

Later that same day, Mr. Adams had a cardiology consult. (Ex. 23, p. 175; see *also* Ex. 5, p. 276.) In the history of present illness, it is noted that Mr. Adams went to bed on the evening of October 11, 2018, “without any complaints” but woke his wife up early the next morning due to difficulty breathing and a cough. (Ex. 23, p. 176.) Although he was warm and diaphoretic, his wife stated that he denied chest pain or palpitations. (*Id.*) It is noted that the paramedics were called and Mr. Adams’s “dyspnea got worse rapidly and he collapsed in front of the paramedics.” (*Id.*) Pulseless electrical activity was noted, and Mr. Adams was intubated. (*Id.*) CPR was performed for several minutes before spontaneous circulation returned. (*Id.*) However, no ventricular tachycardia or ventricular fibrillation was noted, and Mr. Adams did not

required cardioversion during CPR. (*Id.*) Additionally, while the initial EKG showed ischemia, a subsequent EKG, which was performed after Mr. Adams's blood pressure and oxygen had stabilized, showed no ischemia. (*Id.* at 176,188.) During this encounter, Mr. Adams was intubated and unresponsive. (*Id.* at 176.) It is noted,

Initial cardiac enzymes showed troponin 0.3 which is gradually trending up and is now 19. Echocardiogram done today shows low normal LV function and there is no change noted from his prior echocardiogram from January 2018. Patient had been active until recently and according to the family there was no report of exertional chest pain or shortness of breath in the last few days.

(*Id.*) Mr. Adams was diagnosed with cardiopulmonary arrest and there was concern about significant anoxic brain injury, but the cause of his condition was unclear. (*Id.* at 188.) It was further noted that the abnormal cardiac enzymes indicated myocardial infarction, but this was likely secondary to hypoxemia from cardiac arrest. (*Id.*)

On October 13, 2018, Mr. Adams had an infectious disease consult, during which his family reported that he had a reaction to the flu vaccine "some 10 or 15 yrs ago and this was his first flu vaccine in several years." (Ex. 23, p. 161.) It was further noted that Mr. Adams was recently put on a new medication, Jardiance, for his diabetes mellitus, and that he had taken one pill only prior to the incident. (*Id.*) The assessment included cardiac arrest and "[n]on-ST elevation (NSTEMI) myocardial infarction." (*Id.* at 167.) Pulmonary and cardiology causes were ruled out. (*Id.*) It was noted that "it is difficult to say that this was anaphylactic reaction (no wheezing, no [shortness of breath], Vocal cords normal at intubation)" but whether Mr. Adams's condition was related to the vaccinations or the new medication that he started could not be "completely exclude[d]." (*Id.*) Mr. Adams was reported as being in a "normal state of health until the day he got the vaccines." (*Id.*) A VAERS report was contemplated. (*Id.*)

On October 14, 2018, it was noted that Mr. Adams suffered a pulseless electrical activity cardiac arrest of unknown etiology and had spiked a fever, prompting treatment with Zosyn and a cooling blanket. (Ex. 23, p. 189.) Following one episode of fever, which included multiple premature ventricular contractions and nonsustained ventricular tachycardia, Mr. Adams was treated with Amiodarone. (*Id.*) It was noted that Mr. Adams's elevated white blood count could be secondary to stress as a result of the cardiac arrest. (*Id.* at 195.) Mr. Adams's viral panels were negative and there were no signs of infection. (*Id.*) It is noted that Mr. Adams's family was "informed that it is unlikely the flu shot can cause [pulseless electrical activity] arrest but cannot totally rule out." (*Id.*) Mr. Adams underwent an MRI of the brain, which showed hypoxic ischemic injury with mild to moderate diffuse brain edema; moderate new intraventricular hemorrhage with new hydrocephalus; and acute to early subacute left medial frontal, bilateral punctate frontal, and right pontine infarcts. (*Id.* at 196, 243-44.) The radiologist indicated that the bleeding could be secondary to the diffuse hypoxic ischemic injury. (*Id.* at 196.) Mr. Adams's family decided not to pursue aggressive intervention or hospice care and to instead switch his code status to "do not resuscitate with comfort

care.” (*Id.*) He was disconnected from the ventilator and started on a morphine drip for respiratory distress. (*Id.* at 151, 196.) Mr. Adams was pronounced deceased at 8:40 p.m. on October 14, 2018. (*Id.* at 151; Ex. 2.) His death certificate lists his cause of death as anoxic brain injury, intracerebral bleeding, coronary artery disease, and diabetes, and classifies his manner of death as natural. (Ex. 2.) No autopsy was performed. (*Id.*)

b. Testimony

Petitioner authored one affidavit in this case. (Ex. 24.) She also provided testimony at the hearing. (Tr. 8-25.) Petitioner explained that, at around 4:00 a.m. on October 12, 2018, she received a phone call from Mr. Adams, stating that he was “having some kind of attack” and requesting that she come over. (Ex. 24, ¶ 4; Tr. 16.) When petitioner arrived at her father’s house, Mr. Adams was laying on the ground and indicating that he could not breathe. (Ex. 24, ¶ 6; Tr. 16-17.) His wife informed petitioner that Mr. Adams had been complaining that he was hot all night and required multiple electric fans. (Ex. 24, ¶ 7.) Petitioner decided to call 911, and the paramedics arrived thereafter. (*Id.* ¶¶ 7-8; *but see* Tr. 17 (unable to recall whether EMTs arrived first).) Petitioner avers that, at some point, Mr. Adams’s wife informed the paramedics that he had just been to the doctor and received the flu and pneumonia vaccines. (Ex. 24, ¶ 9.) She further describes the paramedics helping Mr. Adams sit up in a chair after he indicated that he could not do it himself. (*Id.* ¶¶ 10-11; Tr. 18.) Mr. Adams was sitting with one hand on his knee for support and his head in his other hand when talking to the paramedics. (Ex. 24, ¶ 11.) “At that point, [Mr. Adams] fell forward onto the floor and his heart stopped.” (*Id.* ¶ 12; Tr. 18-19.) The paramedics that were already there began performing CPR and additional paramedics arrived. (Ex. 24, ¶ 13; Tr. 19-20.) Mr. Adams was then transported by ambulance to the hospital. (Ex. 24, ¶ 16; Tr. 19.) After the paramedics left, petitioner recalls counting five vials of epinephrine “or whatever drug it is they use to try to restart a heart.” (Ex. 24, ¶ 15.) She states her understanding is that Mr. Adams regained a pulse while still at the house but lost the pulse during transport to the hospital. (*Id.* ¶¶ 15-16.)

Petitioner avers that Mr. Adams never regained consciousness. (Ex. 24, ¶ 17; Tr. 22.) She describes how she felt that they made eye contact at one point during his hospitalization, but notes that he could not speak, and that she has “no basis to believe he was conscious.” (Ex. 24, ¶ 17.) She states that Mr. Adams’s physicians eventually stopped running tests and seemed to be waiting for him to regain consciousness to know whether he suffered any brain damage due to lack of oxygen as a result of his cardiac arrest. (*Id.* ¶ 18; Tr. 22-23.) The following day, petitioner states that “a doctor talked to us about our concerns about the vaccines my dad had received, but said nothing conclusive.” (Ex. 24, ¶ 20.) She further avers that “[n]othing seemed to be happening with respect to his care.” (*Id.* ¶ 21.) That night, Mr. Adams “had a fitful night” and spiked a fever. (*Id.* ¶ 23.) Petitioner recalls being very uncomfortable while watching Mr. Adams’s feet move in apparent reflex without the ability to speak. (*Id.*; Tr. 23.) She was concerned that he was in pain with no way to communicate. (Ex. 24, ¶ 23.) She requested that the attending physicians run additional tests or transfer Mr.

Adams to a different hospital because “[i]t seemed they were just waiting for him to die.” (*Id.* ¶ 25.) She was informed that, although another hospital would likely refuse to admit him in his condition, one hospital had agreed to the transfer. (*Id.* ¶ 26.) Before deciding whether to transfer him, they ran additional tests, and the results were grave. (*Id.* ¶¶ 27-28.) Petitioner avers that “[t]here was nothing that could be done that would enable him to regain any form of life that he would want to live, if he would ever regain consciousness.” (*Id.* ¶ 28.) Mr. Adams passed away very soon after his family made the decision to withdraw life support. (*Id.* ¶ 29.)

Petitioner describes,

[m]y father was himself leading up to this. He had quit smoking. He took care of himself more than many, in that he did go to the doctor, and he followed up when he was told to. He almost never missed a day of work. He always got up early – 4 a.m. and started working, took notes of his days, drove to his various meetings throughout his territory, and came home for dinner. He called his sister, Sherry, every day. He called me almost every day, just to check if I “needed anything.” He was the kind of person who loved to cut grass, and his yard was very well-maintained. He always had a clean car and always polished his shoes, which I attribute to his service in the Navy. I last saw him no more than a week before. He would often stop by my house for a chat or I would go to his house for Sunday dinner. I saw no changes in him, and he said nothing to me about feeling bad prior to that terrifying phone call.

(Ex. 24, ¶ 31.)

IV. Expert Opinions

a. Petitioner’s Expert, Cardiac Electrophysiologist Mayer Rashtian, M.D., F.A.C.C., F.H.R.S.⁹

⁹ Dr. Rashtian received his medical degree from Drexel University in Philadelphia, Pennsylvania in 1988, before going on to complete an internship and residency in internal medicine at Loma Lina University Medical Center in Loma Linda, California in 1989 and 1991, respectively; a fellowship in cardiology at LA County-USC Medical Center in Los Angeles, California in 1994; and a fellowship in cardiac electrophysiology at Kaiser Permanente Hospital in Los Angeles, California in 1995. (Ex. 9, p. 1.) He is board certified in cardiovascular disease and clinical cardiac electrophysiology and maintains an active medical license in California, where he practices cardiac electrophysiology at Foothill Cardiology Medical Group. (*Id.* at 2-3; Ex. 8, p. 2.) Additionally, Dr. Rashtian is an Assistant Clinical Professor of Medicine at Keck School of Medicine at the University of Southern California, an Assistant Clinical Professor of Nursing at University of California at Los Angeles, and the Medical Director of the Cardiac Electrophysiology Department at Huntington Memorial Hospital in Pasadena, California. (Ex. 9, p. 3; Ex. 8, p. 2.) Dr. Rashtian states that an integral part of his practice includes caring for patients who have suffered from cardiac arrest; identifying the risk factors, causes, and triggers of cardiac arrest; and prescribing appropriate therapies. (Ex. 8, p. 2.) Another significant part of his practice includes screening patients who are at increased risk of sudden cardiac death and ordering various screening tests to define the substrates that can increase the risk of sudden cardiac death. (*Id.*) He states that he commonly works with patients, such as Mr. Adams, that have been diagnosed with frequent premature ventricular contractions and nonsustained ventricular tachycardia and who continue to have symptoms despite

Dr. Rashtian authored two expert reports on behalf of petitioner in this case. (Exs. 8, 21.) He also provided expert testimony at the hearing. (Tr. 27-77, 127-36.) Dr. Rashtian opines that “the stress of the flu vaccine” caused Mr. Adams to suffer a sudden cardiac death. (Ex. 8, p. 5.) He explains that, prior to the flu vaccine, Mr. Adams had “some form of idiopathic [ventricular arrhythmia],” which was “stable” and included premature ventricular contractions (“PVCs”) and nonsustained ventricular tachycardia. (*Id.* at 4-5; Tr. 35.) Dr. Rashtian notes that Mr. Adams was doing very well on a small dose of beta blockers. (Ex. 8, p. 5.) However, following vaccination, Mr. Adams’s underlying PVCs and nonsustained ventricular tachycardia were “exacerbated,” inducing sudden cardiac death. (*Id.*; Tr. 37-39.)

Although he noted that he agreed with respondent’s summary of the medical records in this case (Ex. 8, p. 4), Dr. Rashtian provided a summary of the relevant chronology of events that led up to Mr. Adams’s death. (Tr. 35-36; *see also* Ex. 10.) He explains that Mr. Adams’s had an underlying medical history of “stable nonobstructive coronary artery disease with established known ventricular arrhythmia, PVC, and nonsustained ventricular tachycardia.” (Tr. 35.) When Mr. Adams visited his primary care doctor in October of 2018, he received the pneumovax and flu vaccines and was also started on a new medication for his diabetes. (*Id.*) Within 14 to 18 hours after this encounter, Mr. Adams started to feel unwell and experienced shortness of breath. (*Id.* at 35-36.) By the time the paramedics arrived, Mr. Adams collapsed and was in pulseless electrical activity (“PEA”). (*Id.* at 36.) As a result of his PEA, Mr. Adams received CPR and epinephrine before subsequently being transported to the hospital. (*Id.*) Upon arrival at the hospital, Mr. Adams was intubated and put on life support. (*Id.*) Dr. Rashtian specifically notes that Mr. Adams had “more evidence of ventricular arrhythmia, nonsustained ventricular tachycardia” at the hospital, and was put on intravenous Amiodarone to suppress that arrhythmia. (*Id.*) After an MRI showed findings indicating that Mr. Adams was essentially brain dead, the family withdrew care and extubated him, and he died shortly thereafter. (*Id.*)

Dr. Rashtian provides an overview of ventricular arrhythmias. (Ex. 8, pp. 2-4.) He explains that ventricular arrhythmia is an umbrella term that includes PVCs, which are extra heartbeats that begin in one of the heart’s two lower pumping chambers and disrupt the regular heart rhythm, causing a sensation of “fluttering” or “skipped beat” in the chest. (*Id.* at 2.) The clinical presentation of ventricular arrhythmia can range from asymptomatic to cardiac arrest with the most life-threatening ventricular arrhythmia being associated with ischemic heart disease, particularly in older patients. (*Id.*) Risk factors for ventricular arrhythmia and sudden cardiac death vary based on underlying cardiac conditions, family history, and genetic variants. (*Id.* at 3.) Long-term monitoring demonstrates that PVCs are found in about 50% of all people with or without heart disease. (*Id.*) However, frequent PVCs – defined as the presence of at least one PVC

medical therapy. (*Id.*) Dr. Rashtian has published 25 articles and 5 abstracts, and he has participated in 30 research experiences. (Ex. 9, pp. 4-9.) Dr. Rashtian was proffered without objection as an expert in cardiology and cardiac electrophysiology. (Tr. 33-34.)

on 12-lead ECG or over 30 PVCs per hour – are associated with increased cardiovascular risk and mortality. (*Id.*) Moreover, in some populations, nonsustained ventricular tachycardia has been independently associated with increased risk of death and other cardiovascular adverse outcomes. (*Id.*) As PVCs and nonsustained ventricular tachycardia are common in cardiovascular disease and have been associated with adverse cardiovascular outcomes, detection of PVCs is generally considered a risk factor for such adverse outcomes and useful in prompting additional testing to determine the presence of underlying conditions. (*Id.*) One such outcome, which is a common consequence of ventricular arrhythmia, is sudden cardiac death. (*Id.*) This outcome accounts for approximately 50% of all cardiovascular deaths. (*Id.*)

Dr. Rashtian describes the mechanisms of ventricular arrhythmia as including enhanced normal automaticity, abnormal automaticity, triggered activity induced by early or late afterdepolarizations, and reentry. (Ex. 8, p. 4.) With regard to reentry, Dr. Rashtian explains that this mechanism requires a trigger to initiate the arrhythmia and a substrate to sustain it. (*Id.*) A potential trigger could be a PVC, which may be due to automaticity. (*Id.*) A potential substrate could be structural remodeling secondary to an underlying disease process, a scar secondary to a prior myocardial infarction or surgical repair, or patchy fibrosis in the setting of cardiomyopathy or hypertrophy. (*Id.*) The electrophysiological substrate is dynamically influenced by a variety of factors including cardiac metabolism, electrolytes, signaling pathways, and autonomic effects. (*Id.*) With regard to enhanced automaticity and abnormal automaticity, ventricular arrhythmia may arise from subordinate pacemaker cells in the His-Purkinje system or ventricular myocardium. (*Id.*) Ventricular arrhythmia that is not associated with underlying structural heart disease or genetic arrhythmia syndrome is commonly referred to as idiopathic. (*Id.*) Idiopathic ventricular arrhythmia tends to be monomorphic and based on a focal mechanism of triggered activity or abnormal automaticity with a few being the result of reentry. (*Id.*) Dr. Rashtian opines that Mr. Adams suffered from an idiopathic ventricular arrhythmia as his cardiac structure was unremarkable, and he did not have previous myocardial infarction. (*Id.*)

Substrate is the underlying pathology of an individual with abnormal cardiac status. (Tr. 39-40.) In this case, Dr. Rashtian notes that Mr. Adams's substrate refers to his stable underlying coronary artery disease with frequent PVCs and nonsustained tachycardia and slightly reduced ejection fraction. (*Id.* at 41; Ex. 8, p. 5.) While Mr. Adams's underlying cardiac condition was stable at the time of vaccination, Dr. Rashtian notes that his substrate left him more susceptible to injury. (Tr. 41, 58.) Specifically, he states that "if you look at 1,000 patients like him, by virtue of having the lower ejection fraction and the PVCs, he would be higher risk of a future cardiac arrest." (*Id.* at 70-71.) Moreover, Dr. Rashtian recognizes that many different factors can impact the type of substrate that Mr. Adams had. (Ex. 8, p. 4; Tr. 71.) Accordingly, for patients with a substrate similar to Mr. Adams who experience an acute cardiac event such as PEA, it is not uncommon for the triggering event to be indeterminable. (Tr. 72-74.)

Dr. Rashtian agrees that Mr. Adams died from a complication of his PEA cardiac arrest. (Tr. 43, 75-76.) He explains that PEA occurs when the heart has a rhythm but

there is no pulse or circulation. (*Id.* at 36.) Essentially, PEA is the end result of a very bad case of hypotension. (*Id.* at 38, 128.) While Dr. Rashtian notes that Mr. Adams was in a PEA cardiac arrest when the paramedics arrived (*Id.* at 36, 38; Ex. 21, p. 3), he acknowledges that it is impossible to determine exactly what was going on before his PEA and the arrival of the paramedics (Tr. 38, 46-47, 59). Therefore, his proposed causal theory focuses on providing “a probable cause and explanation for what preceded the PEA cardiac arrest.” (*Id.* at 43.)

Although Dr. Rashtian agrees that Mr. Adams experienced a PEA cardiac arrest that ultimately led to his death (Tr. 43, 75-76), he opines that Mr. Adams likely first experienced ventricular arrhythmia, such as ventricular fibrillate or ventricular tachycardia, that evolved into a PEA cardiac arrest by the time the paramedics arrived. (Ex. 21, p. 3; Tr. 43-46, 75-76.) While he acknowledges that there is no evidence that Mr. Adams was experiencing ventricular tachycardia when EMS arrived (Tr. 45), Dr. Rashtian notes that it is established that Mr. Adams experienced ventricular tachycardia prior to his vaccination and while hospitalized after his cardiac arrest (*Id.* at 46). Therefore, he contends it is “very likely” that Mr. Adams had ventricular tachycardia before his PEA arrest. (*Id.* at 45, 59.) Specifically, Dr. Rashtian opines that the insult and stress of the flu vaccine combined with his underlying cardiac conditions could have caused Mr. Adams to experience “prolonged nonsustained ventricular tachycardia” that resolved by the time the paramedics arrived and degenerated into PEA. (*Id.* at 44-45, 58.) He contends that any ventricular arrhythmia cardiac arrest that is shockable will degenerate to PEA arrest if not attended to and treated promptly. (Ex. 21, p. 3.) However, Dr. Rashtian concedes it is not common for ventricular tachycardia to degenerate into PEA (Tr. 60-61), noting that “[u]sually a ventricular tachycardia does not terminate in the PEA . . . It degenerates to – usually to a systolic cardiac arrest.” (*Id.* at 47.)

Dr. Rashtian’s opinion implicates the flu vaccine, stating that an acute immunological reaction to the flu vaccine in a patient with an underlying substrate for increased risk of sudden cardiac death is what triggered Mr. Adams’s PEA cardiac arrest. (Ex. 8, p. 5; Tr. 37-39, 58, 75-76.) As a general matter, Dr. Rashtian recognizes the benefits of the flu vaccine, especially for vulnerable cardiac patients, and attests that he strongly advocates the flu vaccine for all his patients, especially those with underlying congestive heart failure, coronary disease, heart attack, and individuals with pacemakers and defibrillators. (Tr. 50-51; Ex. 8, p. 6.) He indicates that based on Mr. Adams’s status at the time of vaccination, “[t]here was no reason not to give him the flu vaccine.” (Tr. 52.) However, he acknowledges that some patients unfortunately suffer an adverse reaction to the flu vaccine. (*Id.* at 51-53.)

Describing the flu vaccine as “both a trigger and aggravation,” Dr. Rashtian contends that the stress of the flu vaccine, acute leukocytosis, and acute inflammatory reaction turned Mr. Adams’s stable substrate into an acutely unstable arrhythmia, which led to his PEA cardiac arrest. (Ex. 8, p. 5; Tr. 37-39.) He explains that Mr. Adams’s reaction to the immune response produced by the vaccine “was too much stress for his heart.” (Tr. 75.) Dr. Rashtian opines that Mr. Adams’s immune response to the flu

vaccine could have exacerbated his underlying PVCs that grew into “long runs of nonsustained ventricular tachycardia that was probably terminated on its own before the paramedic arrived.” (*Id.*; Ex. 8, p. 5.) This sequence of events disturbed the equilibrium of Mr. Adams’s heart and resulted in PEA arrest. (Tr. 75-76; Ex. 8, p. 5.) Moreover, he characterizes his proposed causal hypothesis as “common sense and basic medical theory,” citing *Halverson v. Secretary of Health & Human Services*, No. 15-227V, 2020 WL 992588 (Fed. Cl. Spec. Mstr. Feb. 4, 2020) as support. (Ex. 8, p. 5.) However, Dr. Rashtian also concedes that none of the medical literature filed by petitioner mentions or discusses the flu vaccine as a cause of ventricular arrhythmia, nor does it implicate ventricular arrhythmia as a cause of PEA. (Tr. 60-64 (discussing Sana M. Al-Khatib et al., *2017 AHA/ACC/HRS Guideline for Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death*, 138 CIRCULATION e272 (2018) (Ex. 11); Steven J. Compton, *Ventricular Tachycardia*, MEDSCAPE, <https://emedicine.medscape.com/article/159075> (last updated Dec. 5, 2017) (Ex. 12)).)

Dr. Rashtian contends that the flu vaccine induces a release of cytokines, and vulnerable individuals, such as Mr. Adams, can experience a reaction to this immune response that aggravates and makes an underlying condition more unstable. (Tr. 37-39, 75.) However, he also indicates that nobody can say for certain that Mr. Adams did not suffer a delayed anaphylactic reaction to the flu vaccine. (*Id.* at 38.) Dr. Rashtian states that we know Mr. Adams was hypotensive and short of breath prior to his PEA and stresses that these symptoms are consistent with a delayed anaphylactic reaction. (*Id.* at 38-39.) In the case of a delayed anaphylactic reaction to the vaccine, Dr. Rashtian notes that Mr. Adams’s underlying coronary artery disease and ventricular arrhythmias would have prevented him from tolerating and appropriately responding to the anaphylaxis. (*Id.*) Regardless of whether Mr. Adams’s reaction to the vaccine is characterized as anaphylaxis, Dr. Rashtian opines that it is more probable than not that the vaccination triggered Mr. Adam’s PEA arrest and death. (*Id.* at 74-76.)

However, Dr. Rashtian also notes that he is not providing one direct cause of Mr. Adams’s PEA and death. (Tr. 134.) Rather, he offers a series of possible contributory factors that led to the PEA arrest, noting that Mr. Adams could have experienced “a combination of a nonsustained ventricular tachycardia, delayed anaphylactic reaction, a delayed severe vasovagal reaction, some immune reaction, acute pulmonary edema, any of those or in combination.” (*Id.* at 65.) Dr. Rashtian notes that the symptoms Mr. Adams experienced before his PEA, specifically the elevated temperature, shortness of breath, and hypotension, are indicative of delayed anaphylaxis but also consistent with tachycardia. (*Id.* at 45-46.) He states that Mr. Adams could have been experiencing a cytokine reaction, delayed anaphylaxis, and/or runs of nonsustained ventricular tachycardia, and that all of these clinical scenarios “would give the same symptoms.” (*Id.* at 46.) Therefore, Dr. Rashtian opines that “the combinations of vaccine reaction, cytokine release, runs of a ventricular arrhythmia, possible anaphylactic reaction to [the vaccines], all in combination” more likely than not triggered Mr. Adams’s PEA. (*Id.* at 65; see also *id.* at 37-39, 43-49, 62, 65, 129.)

b. Respondent's Expert, Cardiologist Shane J. LaRue, M.D., M.P.H.S.¹⁰

Dr. LaRue authored two expert reports on behalf of petitioner in this case. (Exs. A, D.) He also provided expert testimony at the hearing. (Tr. 78-126.) Dr. LaRue opines that the subject flu vaccine did not have any bearing on Mr. Adams's clinical course and death. (Ex. A, p. 10; Ex. D, p. 2; Tr. 82-83.)

Dr. LaRue begins by providing a summary of the pertinent medical facts in this case. (Ex. A, pp. 2-5; Tr. 82-83, 91-93.) He notes that Mr. Adams had a medical history significant for non-obstructive coronary artery disease, hypertension, diabetes, mild left ventricular dysfunction, premature ventricular contractions ("PVCs"), and nonsustained ventricular tachycardia. (Ex. A, p. 2.) However, Dr. LaRue agrees with Dr. Rashtian that Mr. Adams's clinical presentation from a cardiac standpoint was stable at the time of vaccination. (Tr. 94.) He describes how Mr. Adams was initially feeling well after receiving the subject vaccination, but notes that he awoke pale, diaphoretic, and warm in the early morning of the day after vaccination. (Ex. A, pp. 4-5 (citing Ex. 5, pp. 237, 267); Tr. 82-83, 95.) Although Mr. Adams did not report chest pain or palpitations, he complained of progressively worsening shortness of breath until he lost consciousness and suffered a PEA cardiac arrest in the presence of EMS personnel. (Ex. A, p. 5 (citing Ex. 5, p. 276); Tr. 83, 95.) Mr. Adams required prolonged resuscitation which led to a brain injury and his ultimate death. (Ex. A, p. 5; Tr. 83.)

Dr. LaRue provides an overview of PEA. (Ex. A, pp. 6-7; Tr. 88-89.) He defines PEA as "any one of a heterogeneous group of organized ECG rhythms without sufficient mechanical contraction of the heart to produce a palpable pulse or measurable blood pressure." (Ex. A, p. 6 (citing Jonathon Elmer, *Advanced Cardiac Life Support (ACLS) in Adults*, UPTODATE (July 13, 2023) (Ex. A, Tab 1; see also Ex. D, Tab 1)).) Further, he notes that, by definition, PEA is a non-perfusing rhythm that requires immediate initiation of CPR. (Ex. A, p. 6 (citing Elmer, *supra*, at Ex. A, Tab 1).) Dr. LaRue explains that with PEA the heart has an electrical pattern but is not actually pumping blood due to an electromechanical dissociation. (Tr. 88.) PEA does not respond to defibrillation because the heart rhythm is not the issue causing the arrest as PEA is a mechanical problem. (Ex. A, p. 6.) Dr. LaRue notes that PEA manifests as a form of cardiac arrest because the heart is not actually circulating blood. (Tr. 88-89.) There are "myriad causes of PEA," and the associated symptoms depend on the underlying cause. (*Id.* at 89.) Dr. LaRue lists a series of common causes of PEA arrests, including

¹⁰ Dr. LaRue received his medical degree in 2005 from the Medical College of Wisconsin in Milwaukee, before going on to complete an internship and residency in internal medicine at the University of Wisconsin Hospitals and Clinics in Madison in 2006 and 2008, respectively, as well as a cardiology fellowship at Washington University School of Medicine in St. Louis, Missouri in 2012. (Ex. B, pp. 1-2.) He is board certified in cardiovascular disease and advanced heart failure and transplant cardiology, and he maintains an active medical license in Missouri and Illinois. (*Id.* at 3.) He currently works as the director of Heart Failure Cardiology at St. Luke's Hospital in Chesterfield, Missouri. (*Id.* at 1; Ex. A, p. 1.) He has authored 40 peer-reviewed articles, covering aspects of advanced heart failure, as well as 4 book chapters, specifically addressing acute systolic heart failure and cardiomyopathy. (Ex. A, p. 1; Ex. B, pp. 7-10.) Dr. LaRue was proffered as an expert in cardiology without objection. (Tr. 81.)

hypovolemia, hypoxia, hydrogen ion acidosis, hyperkalemia, hypokalemia, hypothermia, hypoglycemia, toxins, tamponade, tension PTX, coronary thrombosis, pulmonary thrombosis, and trauma. (Ex. A, p. 7 (citing Laszlo Littmann et al., *A Simplified and Structured Teaching Tool for the Evaluation and Management of Pulseless Electrical Activity*, 23 MED. PRINCIPLES & PRAC. 1 (2014) (Ex. A, Tab 2)).)

In contrast, Dr. LaRue notes that with ventricular arrhythmia such as ventricular tachycardia, the heart rhythm is the issue and is thus an electrical problem. (Ex. A, p. 6; Tr. 85.) He explains that ventricular tachycardia is a specific kind of fast heart rhythm where the ventricles are beating on their own without anything telling them to. (Tr. 85.) Ventricular tachycardia exists on a spectrum and can manifest in different ways with different heart rates and for different durations. (*Id.*) An individual can experience short runs of ventricular tachycardia known as nonsustained ventricular tachycardia, or it can be sustained where it does not stop on its own. (*Id.* at 85-86.) Dr. LaRue notes that individuals experiencing ventricular tachycardia may report buzzing or fluttering sensations in their chest, as Mr. Adams reported to his cardiologist during an encounter prior to vaccination. (*Id.* at 86.) Ventricular arrhythmias can cause cardiac arrest and sudden cardiac death where a person is conscious with a perfusing heart rhythm in one moment and then they collapse because their ventricular arrhythmia is unable to pump blood to the brain. (Ex. A, p. 6.) Because the heart rhythm is the problem with ventricular arrhythmias, they are treated with an external heart shock known as direct current cardioversion. (*Id.*) Dr. LaRue emphasizes that ventricular arrhythmias are not classified as PEA and are not recognized as a potential cause of PEA. (*Id.*) Rather, ventricular arrhythmias and PEA are recognized as the two different types of cardiac arrest. (Tr. 89-90.)

Dr. LaRue agrees that Mr. Adams suffered a PEA arrest that ultimately led to his death. (Ex. A, pp. 5, 7, 10; Ex. D, p. 2; Tr. 82-83, 96.) Specifically, he indicates that Mr. Adams's PEA cardiac arrest occurred at the point when Mr. Adams "was sitting, talking to EMS and lost consciousness." (Tr. 95.) Dr. LaRue opines that Mr. Adams's PEA arrest is of unknown etiology (*Id.* at 106), and he does not offer a theory as to what caused the PEA (*Id.* at 113). However, he stresses that no specific cause of Mr. Adams's PEA arrest was ever suggested by any of his treating providers. (Ex. A, p. 7.) Additionally, he indicates that it is not uncommon for the cause of a PEA that occurs outside of the hospital to remain idiopathic. (Tr. 121-22.) Dr. LaRue opines that the available evidence does not strongly implicate a specific cause of Mr. Adams's PEA, and that sometimes you are left not knowing definitively what caused the cardiac event. (*Id.* at 118-22.)

Dr. LaRue acknowledges that the symptoms Mr. Adams experienced prior to his PEA, including shortness of breath, elevated temperature, and sweating, are not specific symptoms indicative of a specific cause of his PEA. (Tr. 95-96.) However, he contends that the objective data and clinical evidence do not support Dr. Rashtian's opinion that Mr. Adams suffered from ventricular arrhythmia prior to his PEA. (Ex. A, p. 8; Tr. 97.) He emphasizes that, although Mr. Adams required prolonged CPR and multiple rounds of epinephrine, his arrest was characterized as PEA, no ventricular

tachycardia or ventricular fibrillation was noted, and no defibrillator shocks were administered during his resuscitation. (Ex. A, p. 5 (citing Ex. 5, p. 276); Tr. 97-99.) Dr. LaRue explains that ventricular tachycardia and ventricular fibrillation can be treated with defibrillator shocks or direct current cardioversion (i.e., external heart shock). (Ex. A, pp. 5-6 (citing Elmer, *supra*, at Ex. A, Tab 1); Tr. 90, 97-99, 123-24.) Therefore, he suggests that the fact that Mr. Adams did not receive any defibrillator shocks implies that he did not have ventricular tachycardia or ventricular fibrillation. (Ex. A, pp. 5-6; Tr. 97-99.) Additionally, Dr. LaRue stresses that when Mr. Adams experienced ventricular tachycardia and PVCs prior to vaccination, he described feeling buzzing sensations and palpitations in his chest. (Tr. 98; Ex. A, p. 3 (citing Ex. 4, p. 96).) However, Mr. Adams did not complain of any fluttering or buzzing sensations on the morning he suffered his PEA arrest, and he specifically denied experiencing chest pain or palpitations. (Tr. 95-96, 98; Ex. A, p. 5.) While Dr. LaRue acknowledges that an arrest due to ventricular arrhythmia could “in theory” represent an exacerbation of Mr. Adams’s PVCs and nonsustained ventricular tachycardia, Mr. Adams suffered a PEA arrest that is, by definition, not related to a ventricular arrhythmia. (Ex. A, p. 8.) In sum, Dr. LaRue opines that there is no specific evidence demonstrating that Mr. Adams’s PEA arrest was due to an exacerbation of his underlying ventricular arrhythmias. (*Id.* at 9; Tr. 97-99.)

Furthermore, Dr. LaRue opines that the fact that Mr. Adams experienced ventricular tachycardia after his PEA when he was in the hospital is not of any significance because it happened after the arrest and resuscitation. (Tr. 96.) Further, he notes that the EMS personnel were monitoring Mr. Adams and did not detect any arrhythmias when they resuscitated him or in transport to the hospital. (*Id.* at 96-97.) There were other stressors, such as the fevers Mr. Adams experienced while hospitalized, that certainly could have brought on the ventricular arrhythmias he experienced after his cardiac arrest. (*Id.* at 97.) However, the fact that Mr. Adams experienced ventricular tachycardia while hospitalized does not indicate that he arrested from a ventricular arrhythmia. (*Id.*)

Moreover, Dr. LaRue specifically contests Dr. Rashtian’s assertion that “any ventricular arrhythmia cardiac arrest that is shockable will degenerate to PEA arrest if not attend[ed] to and treated promptly.” (Ex. D, pp. 1-2.) He states that, in theory, it is possible that a ventricular arrhythmia could be followed by PEA arrest where an individual who experienced a sustained ventricular tachycardia and arrested is shocked back to a normal cardiac rhythm, but their heart nonetheless cannot pump. (Tr. 90.) However, while conceding that it is not impossible, Dr. LaRue notes that this phenomenon, generally speaking, is “not likely or doesn’t happen.” (*Id.*) In the context of this case, he explains that, although Mr. Adams was feeling unwell, he was still alert when the paramedics arrived. (Ex. D, p. 1 (citing Ex. 5, p. 237); Tr. 97.) Therefore, any ventricular tachycardia that Mr. Adams would have been experiencing prior to his PEA would have been perfusing. (Tr. 97.) Dr. LaRue opines that from a physiological perspective, Dr. Rashtian’s theory is missing a step, noting he doesn’t “understand how a person would go from having [ventricular tachycardia] that was perfusing, . . . and then go back into a normal rhythm and then arrest from a PEA mechanism. (*Id.*) While

he acknowledges that some patients may remain alert for a time while experiencing ventricular tachycardia, Dr. LaRue opines that “there is not a conceivable mechanism for a person to transition from alert with ventricular tachycardia to collapsed with PEA in the presence of EMS personnel.” (Ex. D, p. 1.) Rather, when left unattended, ventricular tachycardia may degenerate to asystole, and asystole is not a rhythm that is classified as a cause of PEA. (*Id.* at 1-2; Tr. 87-88.)

Dr. LaRue opines that there is no scientific evidence of a causal relationship between the flu vaccine and PEA arrest. (Ex. A, p. 7; Tr. 102-103.) He cites a systemic review of flu vaccine literature by Beyer et al., which makes no mention of PEA arrest as a complication of flu vaccination. (Ex. A, p. 7 (citing W.E.P. Beyer et al., *Immunogenicity and Safety of Inactivated Influenza Vaccines in Primed Populations: A Systematic Literature Review and Meta-Analysis*, 29 *VACCINE* 5785 (2011) (Ex. A, Tab 3)).) He further cites a study that looked at VAERS reports over a 23-month period and found no mention of pulseless electrical activity arrest in association with the flu vaccine. (Ex. A, p. 7 (citing Penina Haber et al., *Post-Licensure Surveillance of Quadrivalent Inactivated Influenza (IIV4) Vaccine in the United States, Vaccine Adverse Event Reporting System (VAERS), July 1, 2013–May 31, 2015*, 34 *VACCINE* 2507 (2016) (Ex. A, Tab 4)).) Moreover, Dr. LaRue indicates that a study filed by petitioner concluded that the flu vaccine was associated with a reduced risk of primary cardiac arrest. (Tr. 101-02 (discussing David S. Siscovic et al., *Influenza Vaccination and the Risk of Primary Cardiac Arrest*, 152 *AM. J. EPIDEMIOLOGY* 674 (2000) (Ex. 16)).)

In this case, Dr. LaRue disagrees with Dr. Rashtian’s proposed theories that the stress of the flu vaccine, acute leukocytosis, acute inflammatory reaction, and possible delayed anaphylaxis caused Mr. Adams’s stable substrate to become unstable and induce his ultimate PEA arrest. (Tr. 99-101; Ex. A, p. 8.) First, he notes that there is no evidence to suggest that Mr. Adams suffered from any kind of inflammation reaction as a result of vaccination. (Ex. A, p. 8; Tr. 99.) There was no localized inflammation at the vaccination site, and Mr. Adams’s elevated white blood count is to be expected following any kind of cardiac arrest and prolonged resuscitation. (Ex. A, p. 8 (citing Ex. 5, pp. 241-43, 260); Tr. 99-100.) With respect to anaphylaxis, Dr. Rashtian notes that an anaphylactic reaction generally occurs within minutes to hours of exposure, not 12 hours after exposure. (Tr. 100; Ex. A, p. 7.) While he acknowledges that a delayed anaphylactic reaction may be possible, Dr. LaRue notes that further investigation and research are necessary to determine whether the specific timing in this case could be consistent with a delayed anaphylactic reaction. (Tr. 112-13.) Additionally, he stresses that there is no clinical evidence suggestive of an anaphylactic reaction, noting that there was no wheezing, no vocal fold swelling, and no shortness of breath at the time of intubation. (*Id.* at 100; Ex. A, p. 7 (discussing Ex. 5, p. 268).) Furthermore, none of Mr. Adams’s treating physicians suggested that Mr. Adams suffered an anaphylactic reaction, and Dr. LaRue highlights that his treating physicians specifically documented that an anaphylactic reaction was unlikely given the lack of clinical evidence. (Tr. 100-01; Ex. A, p. 7.)

In sum, Dr. LaRue notes that Mr. Adams experienced a PEA arrest of unknown etiology which ultimately led to his death. (Ex. D, p. 2; Tr. 82-83, 106.) He stresses that Mr. Adams's PEA arrest "was unequivocally not related to any ventricular arrhythmia and thus does not represent any exacerbation of his prior ventricular arrhythmias." (Ex. A, p. 10; Ex. D, p. 2.) Dr. LaRue opines that there is no evidence to suggest or support the conclusion of that Mr. Adams's flu vaccine played any role in his clinical course and ultimate death. (Ex. A, p. 10; Ex. D, p. 2; Tr. 82-83, 106-07.)

V. Analysis

a. *Althen* prong one (theory of causation)

Under *Althen* prong one, petitioner must provide a "reputable medical theory," demonstrating that the subject vaccine can cause the type of injury alleged. *Pafford*, 451 F.3d at 1355-56 (quoting *Pafford v. Sec'y of Health & Human Servs.*, No. 01-0165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004)). Such a theory must only be "legally probable, not medically or scientifically certain." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548-49 (Fed. Cir. 1994). Petitioner may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. See *Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1378-79 (Fed. Cir. 2009) (citing *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1325-26 (Fed. Cir. 2006)). However, "[a] petitioner must provide a 'reputable medical or scientific explanation' for [their] theory." *Boatmon*, 941 F.3d at 1359 (quoting *Moberly*, 592 F.3d at 1322). "While it does not require medical or scientific certainty, it must still be 'sound and reliable.'" *Id.* (quoting *Knudsen*, 35 F.3d at 548-49).

Both parties' experts agree that Mr. Adams died as a result of PEA cardiac arrest. (Tr. 43, 75-76, 96, 106.) However, PEA arrest is only a final result, the moment when blood stops circulating as a result of "myriad" underlying causes. (*Id.* at 48, 89.) Thus, Dr. Rashtian's theory of causation is intended to explain what occurred prior to the ultimate PEA arrest. (*Id.* at 43.) During the course of the hearing, he discussed a number of theoretical propositions as underlying his theory of causation, often returning to the notion that what he proposes is multifactorial. (*E.g.*, *id.* at 131-33.) However, two shortcomings are dispositive with respect to *Althen* prong one. First, Dr. Rashtian is unpersuasive in seeking to explain how the flu vaccine could be the initiating cause of the cardiologic effects he is positing. He is vague with respect to the relevant immunology as a general matter and admits to being unable to substantiate the flu vaccine as a cause of ventricular arrhythmia in particular. Second, even if Dr. Rashtian had succeeded in suggesting that a post-vaccination process could result in a loss of cardiac equilibrium leading to ventricular arrhythmia, he has not preponderantly established that such an arrhythmia would result in the PEA cardiac arrest at issue in this case.

Dr. Rashtian opines as a general matter that vaccines, including the flu vaccine, can aggravate and worsen underlying conditions. (Tr. 37-38.) However, Dr. Rashtian is

not trained in immunology (*Id.* at 135), and his opinion was either vague or inconsistent with respect to the nature of the immune reaction potentially at issue. Initially he cited a post-vaccination release of cytokines. (*Id.* at 37-38.) Later, he suggested that “nobody can say for sure that . . . [Mr. Adams] didn’t have an anaphylactic reaction.” (*Id.* at 38.) He then backtracked on the proposed anaphylactic reaction, indicating his theory would remain operative “whether it was delayed anaphylactic or a systemic reaction or cytokine reaction.” (*Id.* at 45.) Later, on cross examination, he stated that he was relying on “a combination of a nonsustained ventricular tachycardia, delayed anaphylactic [reaction], a delayed severe vasovagal reaction, some immune reaction, acute pulmonary edema, any of those, or in combination.” (*Id.* at 65.) Ultimately, in response to the undersigned’s questions, Dr. Rashtian suggested that, absent an anaphylactic response, an “appropriate” cytokine response to vaccination would be sufficient to disturb Mr. Adams’s cardiac equilibrium given his underlying susceptibility. (*Id.* at 74-75.)

However, even acknowledging that the vaccine can induce some inflammatory immune response, mere invocation of a vaccine’s intended immune response is not in and of itself sufficient to carry petitioner’s burden under *Althen* prong one. See *Elvira ex rel. D.E. v. Sec’y of Health & Human Servs.*, No. 17-531V, 2024 WL 4966035, at *20 (Fed. Cl. Spec. Mstr. Nov. 6, 2024); *Vanore v. Sec’y of Health & Human Servs.*, No. 21-0870V, 2024 WL 3200287, at *18 (Fed. Cl. Spec. Mstr. May 31, 2024); *Kalajdzic ex rel. A.K. v. Sec’y of Health & Human Servs.*, No. 17-792V, 2022 WL 2678877, at *23-24 (Fed. Cl. Spec. Mstr. June 17, 2022), *mot. for rev. denied*, 2024 WL 4524777 (Fed. Cl. Oct. 18, 2024), *aff’d*, No. 2023-1321, 2024 WL 3064398 (Fed. Cir. June 20, 2024); *Cordova v. Sec’y of Health & Human Servs.*, No. 17-1282V, 2021 WL 3285367, at *17 (Fed. Cl. Spec. Mstr. June 23, 2021). There must be some additional evidence linking the vaccine’s immune response to the pathology of petitioner’s actual condition. For example, the Chief Special Master has observed:

I have on many occasions considered theories asserting a vaccine-caused, cytokine-driven process led to injury, but have repeatedly deemed such theories wanting, absent evidence connecting the process (no matter how scientifically plausible it might be) with additional proof sufficient to render it “more likely than not” that the immune processes outlined could be rendered pathogenic by introduction of a vaccine. Otherwise, such a theory only attempts to transmute the expected reaction to a vaccine into pathology.

M.R. v. Sec’y of Health & Human Servs., No. 16-1024V, 2023 WL 4936727, at *27 (Fed. Cl. Spec. Mstr. June 30, 2023) (citing *Dean v. Sec’y of Health & Human Servs.*, No. 13-808V, 2017 WL 2926605, at *17 (Fed. Cl. Spec. Mstr. June 9, 2017); see also *Kaltenmark v. Sec’y of Health & Human Servs.*, No.17-1362V, 2023 WL 8870299, at *28 (Fed. Cl. Spec. Mstr. Nov. 27, 2023) (the undersigned observing that, “[e]ven where there is some reason to suspect a condition may be cytokine mediated, this does not automatically lead to the conclusion that vaccines can cause the injury merely because vaccines produce some cytokine elevations”).

Here, despite being unable to implicate any specific immune process, Dr. Rashtian nonetheless opined without any apparent support that the flu vaccine can cause or trigger ventricular arrhythmia, which he opined is the primary concern for patients with Mr. Adams's substrate. (Tr. 58, 72-73.) He stated that "any stress or injury or anything – stress can initiate and trigger a ventricular tachycardia in a patient who has the appropriate substrate and appropriate treatment triggers." (*Id.* at 58.) However, he agreed that patients with a substrate similar to that of Mr. Adams – a lower ejection fraction of 41 to 50 percent along with frequent PVCs and nonsustained ventricular tachycardia – are at higher risk for a future cardiac event across the board. (*Id.* at 70-71.) He acknowledged that a number of factors can affect this substrate and that it is "not uncommon" for the trigger to remain unknown. (*Id.* at 71, 73; Ex. 8, p. 4.) Moreover, he acknowledged that the literature he has cited does not implicate the flu vaccine as a cause of ventricular arrhythmia (Tr. 60-62 (discussing Al-Khatib et al., *supra*, at Ex. 11; Compton, *supra*, at Ex. 12)), and conceded that "[y]ou're not going to find" articles that "say that [a] vaccine caused the ventricular tachycardia" (*Id.* at 62). Dr. Rashtian has otherwise himself opined that the flu vaccine is cardio-protective. (*Id.* at 50-52; Ex. 8, p. 6.) In his first report indicated that "as a cardiologist/cardiac electrophysiologist, I am a passionate advocate of administering yearly flu vaccines to older patients and to patients with underlying cardiac structure abnormality, to reduce their risk of cardiac events and total mortality and even reduce the total number of cardiac arrests per year." (Ex. 8, p. 6.)

In general, petitioners have not been able to preponderantly establish that the flu vaccine can cause acute cardiovascular events. *Goff v. Sec'y of Health & Human Servs.*, No. 17-259V, 2025 WL 431582 (Fed. Cl. Spec. Mstr. Jan. 13, 2025) (stroke), *mot. for rev. denied*, 2025 WL 2081262 (Fed. Cl. July 9, 2025); *Druery v. Sec'y of Health & Human Servs.*, No. 17-1213V, 2023 WL 5094088 (Fed. Cl. Spec. Mstr. July 11, 2023) (heart attack), *mot. for rev. denied*, 169 Fed. Cl. 557 (2024); *Singleton v. Sec'y of Health & Human Servs.*, No. 17-1474V, 2023 WL 3595653 (Fed. Cl. Spec. Mstr. May 23, 2023) (stroke); *Baldwin v. Sec'y of Health & Human Servs.*, No. 13-957V, 2020 WL 4197937 (Fed. Cl. Spec. Mstr. June 4, 2020) (cardiac arrest), *mot. for rev. denied*, 151 Fed. Cl. 431 (2020); *Schultz v. Sec'y of Health & Human Servs.*, No. 16-539V, 2020 WL 1039161 (Fed. Cl. Spec. Mstr. Jan. 24, 2020) (stroke); *Sokol v. Sec'y of Health & Human Servs.*, No. 16-1631V, 2020 WL 553842 (Fed. Cl. Spec. Mstr. Jan. 9, 2020) (stroke); *Hayward v. Sec'y of Health & Human Servs.*, No. 15-005V, 2018 WL 2772495 (Fed. Cl. Spec. Mstr. May 4, 2018) (cerebral vascular accident); *but see Irwin v. Sec'y of Health & Human Servs.*, No. 16-1454V, 2024 WL 863690 (Fed. Cl. Spec. Mstr. Jan. 23, 2024) (stroke); *Halverson v. Sec'y of Health & Human Servs.*, No. 15-227V, 2020 WL 992588 (Fed. Cl. Spec. Mstr. Feb. 4, 2020) (cardiac arrest).

Even if petitioner had been successful in identifying the flu vaccine as a likely cause of ventricular arrhythmia, Dr. LaRue would still be persuasive in disputing the relevance of ventricular arrhythmia as an underlying cause of PEA arrest. Dr. LaRue explained that ventricular tachycardia and PEA are "big picture, the two different kinds or types of cardiac arrest." (Tr. 89-90.) In his supplemental report, Dr. Rashtian asserted that "I am certain that Dr. LaRue agrees that any ventricular arrhythmia cardiac

arrest that is shockable will degenerate to PEA arrest if not attended to and treated promptly.” (Ex. 21, p. 3.) In response, however, Dr. LaRue explained that unattended ventricular tachycardia degenerates to asystole, which is a complete absence of cardiac rhythm due to the absence of electrical activity in the heart. (Ex. D, pp. 1-2; Tr. 87-88.) PEA arrest, by contrast, is effectively the inverse, constituting “a heterogenous group of organized rhythms without sufficient mechanical contraction of the heart to produce a palpable pulse or measurable blood pressure.” (Ex. D, p. 2 (citing Elmer, *supra*, at Ex. D, Tab 1).) Ultimately, Dr. Rashtian conceded during the hearing that “[u]sually a ventricular tachycardia does not terminate in the PEA . . . It degenerates to – usually to a systolic cardiac arrest.” (Tr. 47.) And, during cross examination, Dr. Rashtian discussed the fact that, despite presenting literature touching on the diagnosis and management of ventricular arrhythmias, these papers do not describe PEA as a potential consequence of ventricular tachycardia. (*Id.* at 60-64 (discussing Al-Khatib et al., *supra*, at Ex. 11; Compton, *supra*, at Ex. 12).)

As Dr. LaRue explains it, the only way that sustained ventricular tachycardia could be followed by PEA would be for a resuscitative shock to bring the cardiac rhythm back to normal, but in so doing nonetheless find that the heart could not pump. (Tr. 90.) While not impossible, he opined that “generally speaking, it’s not likely or doesn’t happen.” (*Id.*) And, as applied to this specific case, because Mr. Adams was conscious and talking prior to his PEA, this would mean that any ventricular tachycardia would have been perfusing, meaning blood was still supplied to the heart and the brain. (*Id.* at 97-98.) Thus, there is a “missing step” physiologically in terms of how blood could have been perfusing during the ventricular tachycardia but then stop perfusing and result in PEA arrest only after the heart rhythm had returned to normal. (*Id.*) Despite acknowledging that what he proposed was “not common” (*Id.* at 60-61), and despite opining that Mr. Adams’ hypothesized tachycardia resolved prior to the arrival of paramedics (*Id.* at 45, 130), Dr. Rashtian never addressed this point by Dr. LaRue. Instead, Dr. Rashtian simply reasoned that, because Mr. Adams experienced ventricular tachycardia on separate occasions both before and after the cardiac arrest, it is probable that he also suffered the same tachycardia in the context of his PEA. (*Id.* at 59.) However, despite submitting two reports, testifying at the hearing, and having the last word in the form of his rebuttal testimony, Dr. Rashtian never explained how ventricular tachycardia would degenerate to PEA.

For all these reasons, petitioner has not met her preponderant burden of proof under *Althen* prong one.

b. *Althen* prong two (logical sequence of cause and effect)

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; *Andreu*, 569 F.3d at 1375-77; *Capizzano*, 440 F.3d at 1326-27; *Grant v. Sec’y of Health & Human Servs.*, 956 F.2d 1144, 1147-48 (Fed. Cir. 1992). Medical records are generally viewed as particularly trustworthy evidence. *Cucuras v. Sec’y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). However, medical

records and/or statements of a treating physician's views do not *per se* bind the special master. See § 300aa-13(b)(1) (providing that “[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court”); *Snyder v. Sec’y of Health & Human Servs.*, 88 Fed. Cl. 706, 745 n.67 (2009) (“[T]here is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted.”). A petitioner may support a cause-in-fact claim through either medical records or expert medical opinion. § 300aa-13(a). The special master is required to consider all the relevant evidence of record, draw plausible inferences, and articulate a rational basis for the decision. *Winkler*, 88 F.4th at 963 (citing *Hines*, 940 F.2d at 1528).

In this case, there is no evidence from Mr. Adams's medical records to suggest that any of his treating physicians would support the causal relationship petitioner is alleging. In fact, the medical records tend to suggest they would not agree. First, none of the treating physicians recorded any opinion endorsing Mr. Adams's vaccination as a causal factor in his condition despite the fact that this was repeatedly questioned by his family. (*E.g.*, Ex. 23, pp. 135, 161, 228.) Second, the medical records indicate that, to the contrary, during a multidisciplinary evaluation in the intensive care unit, “[the] patient[s] family [was] informed that it is unlikely the flu shot can cause PEA arrest.”¹¹ (*Id.* at 195.) Thus, Mr. Adams's treating cardiologist opined that the cause of his PEA was “unclear.” (*Id.* at 188; see also Tr. 106 (Dr. LaRue similarly opining).) Third, whereas Dr. Rashtian's expert opinion incorporates a potential anaphylactic reaction into his explanation, Mr. Adams's treating physicians did not think that an anaphylactic injury was likely, having observed no wheezing, no shortness of breath, and normal vocal cords upon intubation.¹² (Ex. 23, pp. 167, 195; see also Tr. 99-101 (Dr. LaRue agreeing).)

Of course, as noted above, petitioner can also substantiate a logical sequence of cause and effect based on expert opinion. § 300aa-13(a). Here, however, Dr. Rashtian's opinion effectively amounts to speculation. During the hearing, Dr. Rashtian stressed that both he and Dr. LaRue agree that Mr. Adams died as a result of PEA arrest (Tr. 43), and that “when the paramedic arrived, he was in a PEA cardiac arrest. We don't know exactly what was going on that one hour before or the day before . . .” (*Id.* at 38). Upon questioning from the undersigned, Dr. Rashtian confirmed during the

¹¹ In the interest of completeness, I also note that the same statement goes on to state that they “cannot totally rule out” that Mr. Adams's condition is due to adverse effects of the vaccine. (Ex. 23, p. 195.) However, when reading the statement as a whole, it is clear that the overarching point is that the proposed causal relationship is *unlikely*.

¹² Specifically, the medical record indicates that they “cannot completely exclude” an anaphylactic reaction but indicates that “it is difficult to say that this was anaphylactic reaction.” (Ex. 23, p. 167.) Again, as indicated above, while this record is nuanced, the overarching point is that an anaphylactic reaction was considered *unlikely*. See *supra* note 11. Moreover, to the extent an anaphylactic reaction was considered, the treating physicians suggested that it would not necessarily have been due to the vaccination, noting that Mr. Adams had also started a new medication. (Ex. 23, p. 167; see also Ex. 4, p. 7 (newly prescribing empagliflozin at the same medical encounter at which the vaccine at issue was administered).)

hearing that it is “absolutely correct” that once Mr. Adams entered PEA arrest, his theory could neither be proven nor disproven. (*Id.* at 69-70.) In that regard, Dr. LaRue persuasively explained that the symptoms Mr. Adams experienced prior to his PEA (elevated temperature and shortness of breath) were nonspecific and that PEA has many causes. (*Id.* at 89, 95-96.) Moreover, Dr. LaRue explained that clinically it is not unusual for the cause of PEA to remain unknown when it occurs outside of a hospital setting, as it did in this case. (*Id.* at 121-22.) Although Dr. Rashtian sought to reason out what probably happened to the decedent prior to his PEA arrest, he acknowledged multiple times during the hearing that the nature of what happened prior to the arrival of the paramedics simply remains unknown. (*Id.* at 38, 46-47, 59.)

Moreover, Dr. Rashtian’s opinion relies on a jumble of potential contributing factors, none of which are especially well substantiated. During the hearing, Dr. Rashtian stated that he’s “not giving one direct cause.” (Tr. 134.) Instead, he’s “trying to give medical probability of what led before the PEA. And I’m saying the combinations of vaccine reaction, cytokine release, runs of a ventricular arrhythmia, possible anaphylactic reaction to them, all in combination” contributed. (*Id.*) However, none of these specific factors are preponderantly supported. Although Dr. Rashtian opined that petitioner’s symptoms of shortness of breath and elevated temperature are consistent with hypotension, which can in turn be consistent with anaphylaxis, he also indicated that these symptoms can otherwise be consistent with tachycardia. (*Id.* at 38-39, 45-46, 127-28.) Ultimately, Dr. LaRue is persuasive in opining that the symptoms are nonspecific. Apart from these two nonspecific symptoms, there is no clinical evidence to support the presence of a vaccine reaction, cytokine release, ventricular arrhythmia, or anaphylaxis, either individually or in combination. To be sure, the fact that these purported events would have occurred before the arrival of paramedics means that the dearth of confirmed clinical findings could not necessarily have been avoided. However, whereas specific findings to support petitioner’s theory are lacking, Dr. Rashtian otherwise agreed that that any number of factors can affect the type of substrate that Mr. Adams had and that “if you look at 1,000 patients like him, by virtue of him having the lower ejection fraction and the PVCs, he would be [at] higher risk of a future cardiac arrest.” (*Id.* at 70-71; *see also* Ex. 8, p. 4.) Even if the decedent had experienced a tachycardic event, Dr. Rashtian opined that it would not be uncommon for the underlying cause to remain a mystery. (Tr. 72-73.)

And, finally, the one key causal factor that remains clear throughout Dr. Rashtian’s opinion – the role of ventricular tachycardia – is unpersuasive. As discussed under *Althen* prong one, Dr. Rashtian’s suggestion that PEA arrest would result from ventricular tachycardia is in itself unlikely. Thus, it is noteworthy that the treating physicians suggested that arrhythmia had been ruled out. (Ex. 23, p. 167.) In particular, Mr. Adams’s treating cardiologist made note of the fact that “[n]o VT or V. fib was noted [by paramedics] and patient did not require cardioversion during CPR.” (*Id.* at 176.) Furthermore, the treating cardiologist explained that Mr. Adams had not complained of any chest pain or palpitations prior to his PEA, concluding that “[h]e did not have chest pain or cardiac arrhythmias before the PEA.” (*Id.* at 176, 188.) Therefore, contrary to Dr. Rashtian’s opinion, the treating cardiologist concluded that

“[t]he cause of PEA cardiac arrest is not clear.” (*Id.* at 188.) While Dr. Rashtian relies in part on Mr. Adams’s prior history of nonsustained ventricular tachycardia to infer the underlying cause of his PEA, Dr. LaRue is more persuasive in noting that in the hours leading up to PEA, Mr. Adams did not complain of the symptoms he had previously attributed to his tachycardia. (Tr. 95-96.)

Given the temporal proximity, it is, of course, easy to see why Mr. Adams’s family initially focused on his vaccinations as a potential cause of his PEA cardiac arrest and ultimate death. Moreover, when they approached his physicians about that concern, they received measured responses indicating that, while the proposed causal connection was unlikely, it “cannot [be] completely exclude[d]” or “cannot [be] totally rule[d] out.” (Ex. 23, pp. 167, 195.) Thus, it is hardly a surprise that petitioner filed this action. Throughout the course of this proceeding, the parties’ experts have likewise been measured in their assessments. Both experts have stressed that it is impossible to know what happened prior to the point at which Mr. Adams first received paramedic attention. (Tr. 38, 46-47, 59, 95-96, 118-21.) Importantly, however, it is petitioner who bears the preponderant burden of proof, and the Federal Circuit has explained that “[a]lthough probative, neither 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.” *Althen*, 418 F.3d at 1278 (citing *Grant*, 956 F.2d at 1149). While the treating physicians reasonably suggested that it is difficult, if not impossible, to prove a negative, the fact that they could not rule out or completely exclude Mr. Adams’s vaccination as a cause is not tantamount to preponderant evidence supporting that causal relationship. And, while the cause of Mr. Adams’s PEA cardiac arrest remains unknown, for all the reasons discussed above, petitioner’s expert’s explanation of events is not preponderantly supported.

For all these reasons, petitioner has not met her preponderant burden of proof under *Althen* prong two.¹³

¹³ There has been ambiguity in this case with respect to whether the case should be addressed under *Athen* or *Loving*. In her pre-hearing brief, petitioner framed the case as one of significant aggravation, contending with respect to the first three *Loving* prongs that Mr. Adams had pre-existing PVC and nonsustained ventricular tachycardia with a stable substrate, which was significantly aggravated by the vaccination leading to an acutely unstable arrhythmia inducing sudden cardiac death. (ECF No. 87, pp. 16-17.) However, in respondent’s pre-hearing brief, he argues that “[t]o the extent that Mr. Adams had a cardiac arrest, this injury occurred in its entire[t]y after vaccination. While Mr. Adams had predisposing factors for the cardiac arrest, there is insufficient evidence that those risk factors materially changed after vaccination.” (ECF No. 86, p. 14 n.7.) During the hearing, petitioner’s expert, Dr. Rashtian, described the vaccination at issue as “both a trigger and aggravation.” (Tr. 37.) He described the vaccine as creating an immune response that Mr. Adams could not tolerate due to his underlying condition. (*Id.* at 37-39.) I note, however, that it is immaterial whether the alleged logical sequence of cause and effect is evaluated as part of a vaccine-triggered injury under *Althen* prong two or as part of a significant aggravation under *Loving* prong five. The analysis herein indicates that, regardless of whether Mr. Adams’s pre-existing PVC and nonsustained ventricular tachycardia are viewed as a condition to be aggravated or merely as potential predisposing factors, there is inadequate evidence to implicate any arrhythmia as the cause of his PEA cardiac arrest.

c. *Althen* prong three (proximate temporal relationship)

The third *Althen* prong requires establishing a “proximate temporal relationship” between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1278. A petitioner must offer “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 & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008).

In this case, petitioner relies on the notion that “[r]esearch supports that significant inflammatory reactions can arise within hours of vaccination, particularly in patients with pre-existing conditions. This aligns with the understanding of how vaccines can influence cardiovascular stability.” (ECF No. 87, p. 21.) However, neither Dr. Rashtian’s testimony nor the literature he cited support these points.

Though petitioner has failed to develop the record on this point, it is not necessarily controversial that a cytokine response would begin within approximately 12 hours of vaccination as a general matter. However, petitioner has not substantiated that this fact alone is dispositive of timing appropriate for a causal inference relative to the actual injury at issue. For example, in *Hayward*, a case involving a stroke occurring about 12 hours post-vaccination, the special master noted that petitioner’s immunology expert had explained that proinflammatory cytokines begin to increase within six hours of vaccination, but found that the record as a whole supported the conclusion that it would take longer than half a day for such cytokines to increase to pathologic levels and further that relevant literature otherwise supported a longer latency between inflammatory triggers and cerebral vascular accidents. 2018 WL 2772495, at *8, *19. Here, petitioner and her expert essentially take for granted that the timing of onset is appropriate for a causal inference.

But in any event, for the reasons discussed under *Althen* prong one, Dr. Rashtian has not meaningfully addressed how a transient cytokine response to vaccination would result in either ventricular tachycardia specifically or PEA arrest. Thus, this shortcoming prevents petitioner from meeting her burden of proof under *Althen* prong three. The explanation for what is a medically acceptable timeframe must coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one’s requirement). *de Bazan*, 549 F.3d at 1352; *Shapiro v. Sec’y of Health & Human Servs.*, 101 Fed. Cl. 532, 542 (2011), *mot. for recons. denied after remand*, 105 Fed. Cl. 353 (2012), *aff’d*, 503 F. App’x 952 (Fed. Cir. 2013); *Koehn v. Sec’y of Health & Human Servs.*, No. 11-355V, 2013 WL 3214877, at *26 (Fed. Cl. Spec. Mstr. May 30, 2013), *aff’d*, 773 F.3d 1239 (Fed. Cir. 2014).

Additionally, Dr. Rashtian raised for the first time during the hearing that the decedent may have experienced a delayed anaphylactic reaction. (Tr. 38-39.) As with the proposed cytokine response more generally, Dr. Rashtian has not actually substantiated that 12 hours post-vaccination is the appropriate timing for onset of such a reaction. Although Dr. LaRue agreed in general that a delayed anaphylactic reaction

can occur, he did not agree, in the absence of relevant research, that the specific timing in this case would be appropriate for such a reaction. (*Id.* at 112-13.) But in any event, he did not agree based on the decedent's clinical presentation that an anaphylactic reaction is implicated. (*Id.* at 99-101, 122-23.) Because I have credited Dr. LaRue's opinion that anaphylaxis is not implicated under *Althen* prong two, his opinion likewise refutes the notion that the appropriate timing for an anaphylactic injury is informative under *Althen* prong three.

For all these reasons, petitioner has not met her preponderant burden of proof under *Althen* prong three.

VI. Conclusion

As noted at the outset, I offer my sincerest condolences to petitioner for her loss. However, for all of the reasons described above, I find that petitioner has not met her burden of proof. Therefore, pursuant to § 300aa-12(d)(3)(A) and Vaccine Rule 10, this decision concludes that petitioner is not entitled to an award of compensation. Absent a timely motion for review, the Clerk is directed to enter judgment dismissing this case for insufficient proof in accordance with Vaccine Rule 11(a).

IT IS SO ORDERED.

s/Daniel T. Horner
Daniel T. Horner
Special Master