In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 13-957V
Filed: June 4, 2020
PUBLISHED

DONNA BALDWIN, Petitioner, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	Special Master Horner Dismissal; Ruling on the Written Record; Influenza (flu) Vaccine; Ventricular Fibrillation; Cardiac Arrest

Andrew Donald Downing, Van Cott & Talamante, PLLC, Phoenix, AZ, for petitioner.
Lisa Ann Watts, U.S. Department of Justice, Washington, DC, for respondent.

DECISION[1]

On December 5, 2013, petitioner, Donna Baldwin, filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10-34 (2012), alleging that she suffered ventricular fibrillation and cardiac arrest caused-in-fact by her October 13, 2011 influenza ("flu") vaccination. 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; 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*

[1] Because this decision contains a reasoned explanation for the special master's action in this case, it will be posted on the United States Court of Federal Claims' website in accordance with the E-Government Act of 2002. *See* 44 U.S.C. § 3501 note (2012) (Federal Management and Promotion of Electronic Government Services). **This means the decision will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), petitioner has 14 days to identify and move to redact medical or other information the disclosure of which would constitute an unwarranted invasion of privacy. If the special master, upon review, agrees that the identified material fits within this definition, it will be redacted from public access.

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).

In many cases, however, the vaccine recipient may have suffered an injury not of the type covered in the Vaccine Injury Table. In such instances, an alternative means exists to demonstrate entitlement to a Program award. That is, the petitioner may gain an award by showing that the recipient's injury was "caused-in-fact" by the vaccination in question. § 300aa-13(a)(1)(B); § 300aa-11(c)(1)(C)(ii). In such a situation, of course, the presumptions available under the Vaccine Injury Table are inoperative. The burden is on the petitioner to introduce evidence demonstrating that the vaccination actually caused the injury in question. *Althen v. Sec'y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005); *Hines v. Sec'y of Health & Human Servs.*, 940 F.2d 1518, 1525 (Fed. Cir. 1991).

The showing of "causation-in-fact" must satisfy the "preponderance of the evidence" standard, the same standard ordinarily used in tort litigation. § 300aa-13(a)(1)(A); *see also Althen*, 418 F.3d at 1279; *Hines*, 940 F.2d at 1525. Under that standard, the petitioner must show that it is "more probable than not" that the vaccination was the cause of the injury. *Althen*, 418 F.3d at 1279. The petitioner need not show that the vaccination was the sole cause but must demonstrate that the vaccination was at least a "substantial factor" in causing the condition, and was a "but for" cause. *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999). Thus, the petitioner must supply "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury[,]" with the logical sequence being supported by "reputable medical or scientific explanation, *i.e.*, evidence in the form of scientific studies or expert medical testimony." *Althen*, 418 F.3d at 1278; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). A petitioner may not receive a Vaccine Program award based solely on his or her assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In what has become the predominant framing of this burden of proof, the *Althen* court described the "causation-in-fact" standard, as follows:

> Concisely stated, Althen's burden is to show by preponderant evidence that the vaccination brought about her injury by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between

> vaccination and injury. If Althen satisfies this burden, she is "entitled to recover unless the [government] shows, also by a preponderance of the evidence, that the injury was in fact caused by factors unrelated to the vaccine."

Althen, 418 F.3d at 1278 (citations omitted). The *Althen* court noted that a petitioner need not necessarily supply evidence from *medical literature* supporting petitioner's causation contention, so long as the petitioner supplies the *medical opinion* of an expert. *Id*. at 1279-80. The court also indicated that, 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." *Id*. at 1280.

In this case, petitioner has alleged that a flu vaccination caused her to suffer ventricular fibrillation and cardiac arrest. Since these conditions are not listed on the Vaccine Injury Table relative to the flu vaccine, petitioner must satisfy the above-described *Althen* test for establishing causation-in-fact.

II. Procedural History

Petitioner initiated this case on December 5, 2013, and subsequently filed medical records marked as Exhibits 1-22 and an affidavit marked as Exhibit 23. (ECF Nos.1, 4, 8.) She later filed additional medical records marked as Exhibit 24 and a Statement of Completion on April 14, 2014. (ECF Nos. 9-10.) The petition alleged that six days after her flu vaccination petitioner experienced a ventricular fibrillation and cardiac arrest caused by her vaccination. (ECF No. 1.) At that time, she was represented by attorney John Caldwell and the case was assigned to Special Master Laura Millman.

Special Master Millman questioned whether petitioner had a reasonable basis to proceed with the case but allowed petitioner the opportunity to consult an expert. (ECF No. 7.) Petitioner initially filed an expert report by cardiologist Robert Waugh, M.D., on August 18, 2014. (ECF No. 18; Ex. 25.) However, Special Master Millman subsequently required a supplemental report better addressing the required showings under the above-described *Althen* test. (ECF No. 20.)

Petitioner filed a supplemental report by Dr. Waugh on March 5, 2015.[2] (ECF No. 32; Ex. 29.) Dr. Waugh relied in significant part on a study by Lanza et al., which discussed vaccine-induced changes in platelet activity leading to a transient increase in the risk of cardiovascular events. (Ex. 29; Gaetano A. Lanza et al., *Inflammation-Related Effects of Adjuvant Influenza A Vaccination on Platelet Activation and Cardiac Autonomic Function*, 269 J. INTERNAL MEDICINE 118 (2011) (Ex. 28).) However, the study only addressed a period of forty-eight hours post-vaccination. (Lanza et al.,

[2] The report was initially filed on February 13, 2015 as Exhibit 27, but that copy was struck and refiled as Exhibit 29. (ECF Nos. 29, 31.)

supra, at Ex. 28.) Special Master Millman required a further supplemental report addressing how the Lanza article supported petitioner's claim arising six days post-vaccination. (ECF No. 30.) Petitioner filed additional medical records marked as Exhibits 30-34 in May of 2015 and then a second supplemental report by Dr. Waugh in June of 2015. (ECF Nos. 35, 38; Ex. 35.)

Respondent filed both his Rule 4 report recommending against compensation and a responsive expert report by cardiologist Lawrence Sperling, M.D., on September 30, 2015. (ECF No. 41; Exs. A-D.) Petitioner filed a responsive report by Dr. Waugh on January 6, 2016, along with additional supporting literature. (ECF No. 44; Exs. 36-38.)

On January 11, 2016, Special Master Millman held a status conference in which she discussed with the parties two articles, Christian et al., and Tsai et al., which she filed as Court Exhibits I and II respectively. (ECF Nos. 45-46.) Special Master Millman felt these two studies cast doubt on petitioner's reliance on the previously-filed Lanza article. (ECF No. 46.) Special Master Millman provided respondent an opportunity to file a supplemental expert report and instructed Dr. Sperling to address the two court exhibits. (ECF No. 47.) Respondent filed a supplemental report by Dr. Sperling on April 27, 2016. (ECF No. 50; Exs. E-G.)

An entitlement hearing was subsequently set, but then cancelled due to Dr. Waugh's retirement. (ECF Nos. 51-53.) Petitioner was ordered to file a report by a different expert or dismiss the case. (ECF No. 54.) However, petitioner's counsel (Mr. Caldwell) withdrew from the case before any expert report was filed. (ECF Nos. 54-61.)

On September 12, 2017, Special Master Millman held a status conference with petitioner, now proceeding *pro se*. (ECF No. 63.) Petitioner indicated that she was seeking another attorney, but also indicated that she wished to change her theory of the case. Petitioner wished to allege vaccine-caused peripheral neuropathy, Guillain-Barre syndrome ("GBS"), and positive findings of cytomegalovirus ("CMS") and Epstein Barr virus ("EBV"). (*Id*.) Special Master Millman indicated that additional medical records substantiating these allegations would be needed, but that petitioner should first determine whether she will proceed with different counsel.[3] (*Id*.)

On October 2, 2017, petitioner's current counsel, Andrew Downing, moved to be substituted as counsel in this case. (ECF No. 65.) Petitioner subsequently filed an expert report by cardiologist Robert Stark, M.D., with supporting medical literature. (ECF No. 69; Ex. 39-42.) The report opined that petitioner experienced vaccine-caused ventricular fibrillation and did not address any of the conditions alleged by petitioner during the September 12, 2017 status conference. (Ex. 39.)

Special Master Millman required respondent to file a supplemental report by Dr. Sperling responsive to Dr. Stark's report, but respondent advised that Dr. Sperling was

[3] These claims were not further pursued and were never substantiated. In her motion for a ruling on the record, petitioner requested a finding in her favor based only on her ventricular fibrillation and cardiac arrest. (ECF No. 92.)

no longer able to participate in the case. (ECF Nos. 70-71.) Respondent filed an expert report by cardiologist Shane LaRue, M.D., on March 5, 2018, and later filed the supporting literature on April 2, 2018. (ECF No. 72; Exs. H-I; ECF No. 75; Exs. J-R.)

Petitioner filed a supplemental report by Dr. Stark responsive to Dr. LaRue's report in March of 2018 and Dr. LaRue filed a supplemental report with supporting literature in July of 2018. (ECF No. 74; Ex. 43; ECF No. 77; Exs. S-V.) Thereafter, updated medical records and an additional statement of completion were filed between August and October of 2018. (ECF Nos. 79-84.)

On November 2, 2018, Special Master Millman advised that she would be retiring and that a hearing would be set in the case once it was reassigned to another special master. (ECF No. 85.) Subsequently, the case was reassigned to me on June 7, 2019, upon Special Master Millman's retirement. (ECF No. 87.)

During a status conference held June 14, 2019, I requested that the parties consult their experts and report back with proposed hearing dates. (ECF No. 88.) However, the parties requested a follow-up status conference and reported that they wished to have this case resolved on the written record. (ECF No. 91.) Petitioner's counsel confirmed that petitioner had no further evidence she wished to file.[4] (*Id*.)

Petitioner filed a motion for a ruling on the written record on September 23, 2019. (ECF No. 92.) Respondent filed a response on November 22, 2019 and petitioner filed her reply on February 5, 2020. (ECF Nos. 93, 97.) Additionally, petitioner filed a limited second supplemental expert report from Dr. Robert Stark in response to my order of December 3, 2019.[5] (ECF No. 96.) Accordingly, this case is now ripe for a decision.

III. Factual History

a. Medical Records

i. Before Vaccination

Petitioner has a history of various medical issues including muscle spasm, chronic fatigue syndrome, obesity, anemia, hypothyroidism, hyperlipidemia and dyslipidemia, hypertension, anxiety, depression, and diabetes. (Ex. 2, p. 25; Ex. 3, p. 3; Ex. 5, p. 2; Ex. 6, p. 2, 8; Ex. 9, pp. 3, 14; Ex. 14, p. 2.) Toward the end of 2009, petitioner had a cardiology consult for complaints of palpitations, dyspnea, and atypical

[4] Although the decision to proceed based on the written record was based on the agreement of the parties, I note that I have also separately determined that the parties have had a full and fair opportunity to present their cases and that it is appropriate to resolve this case without a hearing. *See* Vaccine Rule 8(d); Vaccine Rule 3(b)(2); *Kreizenbeck v. Secretary of Health & Human Services*, 945 F.3d 1362, 1366 (Fed. Cir. 2020) (noting that "special masters must determine that the record is comprehensive and fully developed before ruling on the record.").

[5] Along with the second supplemental expert report, petitioner filed two additional articles that were previously cited in Dr. Stark's first supplemental expert report but not previously filed.

chest pain. (Ex. 5, p. 2.) Upon initial evaluation, Dr. Jeffrey Howard noted that petitioner had a "low cardiovascular risk profile," but wanted to conduct a stress test and monitor petitioner to rule out any significant arrhythmias. (*Id*. at 3.) Overall, Dr. Howard interpreted petitioner's stress echocardiography and event monitoring diagnostic testing as normal, revealing no evidence of ischemia, and therefore, further diagnostic testing was unwarranted. (*Id*. at 9; 12.) However, petitioner's November 20, 2009 echocardiogram indicated that petitioner had "concentric LV hypertrophy" and "minimal to mild mitral regurgitation," but the impression was that petitioner's echocardiogram was favorable and "no stress induced regional wall motion abnormalities" were detected. (*Id*. at 10.)

Throughout 2010, petitioner sought treatment at Healing Innovations and frequently had visits to address various medical issues including elevated blood pressure, headaches, vertigo, fatigue, ear pain, and upper respiratory infection (URI). (*See* Ex. 6, pp. 2-29.) Petitioner reported having heart palpitations, rapid heart beating, and/or chest pain or pressure in various visits. (*Id*. at 7, 30, 43; Ex. 12, p. 5.) Significantly, in June 2010 on two different occasions, petitioner went to the Emergency Department at Dekalb Medical for dizziness and chest pain, but was discharged on the same day. (Ex. 13, pp. 95-97, 132-33.) Petitioner's electrocardiogram ("ECG") on June 9, 2010, was abnormal compared to her June 1, 2010 ECG, indicating "possible anterior infarct." (*Id*. at 90, 128.)

In early 2011, petitioner underwent left knee surgery and suffered an episode of laryngospasm while in recovery. (Ex. 2, p. 25; Ex. 4, p. 9; Ex. 24, p. 12.) In March 2011, petitioner underwent an internal medicine exam for disability evaluation. (Ex. 12.) On April 4, 2011, petitioner was referred to Dr. Roger S. Blair for a neurological evaluation for her chronic muscle spasms. (*Id*.) Petitioner reported to Dr. Blair that she suffered from twitches, trembling, and spasms, and had trouble swallowing. (*Id*.) Upon a review of systems, petitioner also complained of shortness of breath and heart palpitation. (*Id*. at 28.) Petitioner was referred to an otolaryngologist for her laryngospasm. (*Id*. at 30.) A month later, petitioner also had a neurological evaluation from Dr. Hung Huy Nguyen. (Ex. 11, pp. 2-4.) During that visit, petitioner reported family history[6] of myocardial infarction. (*Id*. at 3.) Upon physical examination, petitioner's heart had regular rhythm and no murmur. (*Id*.) Additionally, in April 2011, petitioner had an ultrasound guided aspiration biopsy on her left thyroid nodule. (Ex. 24, pp. 147-150,158.)

ii. Vaccination and Cardiac Event

Petitioner was administered the flu vaccine on October 13, 2011. (Ex. 21, p. 19.) Six days later, on October 19, 2011, petitioner suddenly began experiencing chest pain at home and had a "VFib arrest in the ambulance" en route to the hospital. (Ex. 24, p. 195.) Petitioner said that she started having some chest pain after sitting down from

[6] Petitioner stated specifically that her maternal grandfather had heart disease. (Ex. 33, p. 4.) Petitioner provided further family history during her cardiology consult in 2018, where she informed her doctor that her mother, paternal grandfather, and maternal grandfather all had heart issues. (Ex. 49, p. 3.)

"rushing around the house for towels to clean up" a water leak and described the pain as a dull pressure type pain in the center of the chest that radiated into her left shoulder and through her back. (*Id.* at 195; Ex. 9, p. 8.) Petitioner lost consciousness in the ambulance and was successfully resuscitated. (Ex. 24, p. 195.) Petitioner was admitted to Texas Health Harris Methodist Hospital HEB for ventricular fibrillation ("VF") cardiac arrest. (Ex. 24, p. 170; Ex. 2, p. 20.) Petitioner was "actively vomiting upon arrival and has blood in it," and reported that she felt like she was drowning." (Ex. 24, p. 187.) Petitioner reported no prior cardiac history and normal results from a prior catherization. (*Id*.) Upon physical examination after being admitted into the intensive care unit (ICU), petitioner had regular rate and rhythm and no murmur, rub or gallop was noted. (*Id*. at 9-10.)

Petitioner had a consultation with Dr. Srinivas Paranandi, cardiologist, on the same day she was admitted. (Ex. 9, p. 13-20.) Dr. Paranandi noted that petitioner was emotionally upset from her bathroom repairs, felt tightness in her chest, and experienced nausea and diaphoresis before eventually passing out. (*Id*. at 13.) Upon assessing the rhythm strips, he confirmed that petitioner was "in ventricular fibrillation and received successful defibrillation with restoration of sinus rhythm." (*Id*.) Petitioner's "EKG was nonacute and no significant ST segment deviations were noted," and her CT angiography study was also nonacute. (*Id*. at 14.) Dr. Paranandi noted that petitioner had some degree of aspiration but did not develop asystole after defibrillation. He further indicated that petitioner's echocardiogram showed no significant valvular or wall-motion abnormalities and upon evaluating coronary risk factors, Dr. Paranandi noted no prior documented history of coronary disease or peripheral vascular disease, but noted petitioner being morbidly obese with a history of hypertension, hyperglycemia, and hyperlipidemia. (*Id*.) Additionally, petitioner told Dr. Paranandi that during a cardiac assessment two years prior, petitioner was told that one of her heart valves "may not be closing well." (*Id*.)

Dr. Paranandi assessed petitioner's diagnostic testing on the day of her cardiac episode and wrote that her "[c]hest x-ray showed some patchy infiltrates, possibly due to aspiration," "[c]ardiac enzymes are negative for myocardial injury so far," and "echocardiogram showed ejection fraction of 70 percent and moderate left ventricular hypertrophy." (*Id*. at 16.) Dr. Paranandi's impression was that petitioner had "an out-of-hospital ventricular fibrillation arrest, requiring cardiopulmonary arrest and defibrillation to restore sinus rhythm, and since that time has remained stable, without further recurrence of arrhythmias." (*Id*.) Moreover, he indicated that the exact reason for her episode was unclear and since petitioner had normal left ventricular function, it appeared that petitioner did not have dilated cardiomyopathy issues. However, Dr. Paranandi suggested petitioner undergo a cardiac catherization to consider underlying coronary disease. (*Id*. at 16-17.) Petitioner underwent a diagnostic catheterization procedure on the same day and her results were unremarkable. (Ex. 2, p. 20; Ex. 9, p. 18-19; Ex. 24, p. 209.)

Dr. Paranandi referred petitioner to Dr. Scott L. Greenberg for further cardiology consultation and management. (Ex. 24, p. 206.) Dr. Greenberg noted that petitioner's

VF was of unclear etiology and that petitioner "does not have significant electrolyte disorder, cardiomyopathy, or obstructive coronary artery disease." (*Id*. at 207.) Additionally, Dr. Greenberg indicated that petitioner had "mild elevation of her troponin," which he attributed to the rapid heartbeat and cardiac arrest, "as opposed to a primary myocardial infarction." (*Id*.) However, Dr. Greenberg thought that petitioner was "absolutely at high risk for recurrent ventricular fibrillation" and that petitioner would benefit from having a defibrator implanted. (*Id*.) Petitioner then had an implantable cardioverter defibrillator ("ICD") inputted by Dr. Greenberg. (Ex. 9, p. 21.) After two days in the hospital, petitioner was overall stable since being revived and thus, Dr. Gary Duc Nguyen discharged petitioner on October 21, 2011. (Ex. 2, p. 20; Ex. 9, p. 17.)

iii. Post Vaccination

On October 27, 2011, a week after being discharged and two weeks after receiving her flu vaccination, petitioner visited her primary care physician at Hurst Family Medical for a follow-up and requested a referral to see Dr. Paranandi. (Ex. 2, p. 3.) The next day, petitioner visited Dr. Paranandi at the Cardiac & Vascular Center of North Texas for an outpatient follow-up. (Ex. 9, p. 3; Ex. 15, p. 6.) Dr. Paranandi stated that petitioner had presented with an out of hospital VF, was successfully resuscitated, and petitioner's rhythm strips did in fact indicate VF documentation. Dr. Paranandi stated that petitioner's catherization "showed normal coronary arties and ejection fraction of 65% to 70%, and mild hypokinesis of the mid-anterior region." (Ex. 9, p. 3.) Dr. Paranandi summarized petitioner's echocardiogram results, stating that it "showed ejection fraction of 70%, mild left ventricular hypertrophy, less than 1+ mitral and tricuspid insufficiency, and PA systolic pressure of 40mmHg." (*Id*.) Dr. Paranandi could not rule out a coronary spasm event, but believed that petitioner did not have a myocardial infarction. (*Id*.) At this appointment, petitioner reported having "'cattle prod' sensations in the chest." Dr. Paranandi examined petitioner's ICD, which showed she did not require or receive any ICD therapies, and the device itself was functioning well. (*Id*.) Therefore, Dr. Paranandi consulted Dr. Greenberg, who confirmed that petitioner's "'phantom shocks' could be related to anxiety." (*Id*.) About a week later, on November 2, 2011, petitioner returned to Hurst Family Medical for another follow-up following her VF cardiac arrest. Petitioner was noted to be "doing well," and she requested a referral to see Dr. Greenberg. (Ex. 2, p. 2.)

Petitioner had another outpatient follow-up visit with Dr. Paranandi on December 5, 2011. (Ex. 15, p. 3; Ex. 19, p. 57.) Petitioner reported, "chills every night, palpitations causing anxiety." (Ex. 15, p. 6.) Dr. Paranandi indicated as part of petitioner's history that "she had a[n] emotional argument with her husband prior to [VF arrest] and it was felt that she probably had a coronary spasm event, since there was a small area of mild focal disease in the mid anterior region." (Ex. 15, p. 3; Ex. 19, p. 57.) Petitioner's ICD was examined and appeared to be functioning well. "There were 3 brief episodes of NSVT[7] but no therapies were required or delivered." (Ex. 15, p. 3; Ex. 19,

[7] NSVT means nonsustained ventricular tachycardia. Neil M. Davis, *Medical Abbreviations: 26,000 Conveniences at the Expense of Communication and Safety*, p. 256 (12th ed. 2005).

p. 57.) Dr. Paranandi noted that petitioner appeared to be doing well and did not have any significant arrhythmias requiring her ICD to discharge. (Ex. 15, p. 4.) Dr. Paranandi advised petitioner to obtain a sleep study[8] and lower GI evaluation[9] for her ongoing issues through her PCP. (*Id*.)

On March 2, 2012, petitioner visited Dr. Greenberg for an ICD evaluation. (Ex. 14, p. 2.) Petitioner denied any chest pain, and upon physical examination, petitioner had regular rhythm and heart sounds. (*Id*. at 3.) Petitioner's ICD was functioning normally and she had not had any arrhythmias. (*Id*.) A couple of days thereafter, petitioner visited Dr. Paranandi for a follow-up and device check. (Ex. 19, p. 37-45.) Dr. Paranandi indicated that there were no results for any recent diagnostic studies, and he felt that petitioner, from the cardiac standpoint, was stable, but needed to be assessed for obstructive sleep apnea. (*Id*. at 40.)

On April 6, 2012, petitioner went to the emergency room ("ER") thinking she experienced ICD discharge, however no ICD discharge nor ICD malfunction was detected, and petitioner had a normal physical examination at the emergency room. (Ex. 19, p. 28; Ex. 22, pp.129-35; Ex. 24, p. 623, 626.) However, petitioner's EKG reflected that petitioner had poor R wave progress across the precordium and "[c]ompared with prior ECG, T wave amplitude in anterior leads has decreased, NS T wave changes laterally no longer present." (Ex. 22, p. 134.) Petitioner was discharged on the same day and it was confirmed that there was no evidence of petitioner's ICD firing or malfunctioning. (*Id*. at 134-35.) Petitioner was instructed to monitor her blood pressure at home and given detailed risk modification counseling including diet and activity instructions. (*Id.* at 6, 23, 31-32, 40.) Petitioner was evaluated by Dr. Paranandi on April 10, 2012, following her visit to the ER. (Ex. 19, p. 28.) Under the review of systems, petitioner was "positive for chest pain, palpitations and leg swelling." (*Id*. at 31.) Petitioner's EKG showed "sinus rhythm, no acute changes or prior infarctions, left posterior fascicular block pattern." (*Id*. at 28.) Dr. Paranandi reassured petitioner that there were no ICD shocks noted, reinforced that petitioner needed an assessment for obstructive sleep apnea, and concluded that petitioner was clinically doing well and was stable from the cardiovascular standpoint. (*Id*.)

Petitioner had another follow-up visit with Dr. Paranandi on June 12, 2012. (Ex. 16.) Again, petitioner's ICD was functioning well and there were no discharges. (*Id*. at 2.) Petitioner denied chest pains but had mildly chronic shortness of breath. (*Id*.) Petitioner also reported occasional brief palpitations and emotional distress for various reasons. (*Id*. at 4, 28.) Petitioner's EKG during this visit showed "sinus rhythm, poor R-wave progression through precordial leads, and no acute changes or definite infarction." (*Id*. at 2.) Petitioner's follow-up visit was otherwise unremarkable, and Dr. Paranandi noted that petitioner was clinically doing well and was stable from a cardiovascular standpoint. (*Id*. at 5.) Petitioner visited Dr. Paranandi for further outpatient follow-ups

[8] Petitioner underwent a sleep study in October 2012. (Ex. 17; Ex. 19, pp. 50-56.)

[9] Petitioner had a colonoscopy in February 2010. (Ex. 8.)

every three months. (Ex. 19, pp. 3, 11, 28, 37.) Petitioner's ICD continued to function well, and no intervention was ever needed. However, additional brief runs of NSVT not requiring ICD shocks were noted. (Ex. 19, pp. 7, 15, 23; Ex. 45, p. 285.)

On October 4, 2012, petitioner had a sleep medicine consultation with Dr. Jack D. Gardner, which led to an initial nocturnal polysomnogram study a couple of weeks thereafter. (Ex. 17, pp. 8-12.) Dr. Gardner's initial impression was that petitioner had mild respiratory disturbance and petitioner's polysomnogram revealed some apnea/hypopnea. (*Id*. at 11.) Petitioner was diagnosed with "Sleep Related Breathing Disorder/ Snoring." (*Id*. at 8.) Additionally, petitioner underwent a Home Sleep Test on October 24, 2012 and was found with "[o]bstructive sleep apnea with overall relatively low AHI but increased respiratory disturbance in REM sleep. Occasional oximetry artifact was noted." (*Id*. at 5.) In his analysis of petitioner's Home Sleep Test results, Dr. Gardner discussed petitioner's clinical history including hypertension, diabetes, and symptoms of nocturnal waking up choking, and concluded that petitioner "has a high pre-test probability of having mild OSA." (*Id*.)

Throughout 2013 and 2014, about every three months, petitioner visited Dr. Paranandi for further follow-up evaluations to monitor her ICD and any cardiovascular changes. (Ex. 22, pp. 75, 87,103; Ex. 45, pp. 65, 116, 153, 284.) During petitioner's visit on July 19, 2013, Dr. Paranandi reported that petitioner had an echocardiogram on June 29, 2013, which showed that petitioner had mild left ventricular hypertrophy with normal chamber dimensions. (Ex. 22, p. 87-88.) Dr. Paranandi also noted during this visit that petitioner underwent a sleep study and "apparently there was some sleep apnea found." (Ex. 45, p. 285.)

Petitioner was referred to Dr. Valery Lipenko at THP Neurology Specialists of North Texas for a neurology consultation and was subsequently examined for numbness and tingling in her arms, legs, hands, feet, lips, and tongue on April 29, 2014. (Ex. 45, pp. 202-08.) Petitioner reported that "in 2011 she went into v-fib and had to be resuscitated and her mobility problems increased after this event." (*Id*. at 202.) Upon a review of systems, petitioner reported chest pain, palpitations, and leg swelling. (*Id*. at 204.) Upon physical examination, petitioner had normal heart rate, regular rhythm, normal pulses, and no gallop or murmur. (*Id*. at 205.) Dr. Lipenko assessed petitioner with possible underlying sleep apnea and underlying small fiber neuropathy, as well as numbness and tingling, chronic headache, fatigue, facial numbness, and difficulty with memory. (*Id*. at 206.)

Dr. Lipenko subsequently referred petitioner to physical therapy, and on May 5, 2014, petitioner underwent an initial assessment at Texas Health Harris Methodist Hospital. (Ex. 47, p. 88.) Petitioner reported that she suffered a cardiac arrest in October 2011 and "[s]ince that episode she has noticed more of a decline in her health." (*Id*.)

About a month later, on June 5, 2014, petitioner visited Dr. Stevan Cordas, D.O, M.P.H. (Ex. 30, pp.1-2; Ex. 45, pp. 265-66.) Petitioner reported that in "October 2011 she had incorrectly provided influenza injection at Walgreens and then developed a flu-

like syndrome almost immediately. Three or four days later she had ventricular fibrillation.[10] Subsequently, she saw her cardiologist who put a pacemaker defibrillator and she report[ed] that she no longer had any activation of the defibrillator." (Ex. 30, p. 1.) Dr. Cordas reviewed his records from a prior examination of petitioner in March 2011, before her cardiac event and noted that petitioner had preexisting rheumatologic problems in weightbearing joints. Dr. Cordas was aware that petitioner was pursuing a vaccine-injury related claim. (*Id*.) Dr. Cordas stated, "[n]o symptoms typical of anaphylactic or anaphylactoid reactions related to the vaccine, so I cannot directly link the cardiac arrest to the flu shot." (*Id*. at 2.)

Regarding petitioner's body pain, Dr. Cordas referred petitioner to see Dr. Jay Roop, whom she visited on June 19, 2014. (Ex. 31, p. 13.) Petitioner reported, "prior to Oct 2011 I did not have v-fib or chronic pain with severe mobility issues. The noticeable increase was after my v-fib." (*Id*. at 11.) Petitioner underwent osteopathic manipulative treatment from Dr. Roop, who opined that petitioner needed ongoing evaluation and treatment due to the nature of her chronic pain. (*Id*. at 13.) Petitioner continued visiting Dr. Roop throughout 2014 and in 2015 to treat her knee pain with acupuncture and Hyalgan injections. (*See* Ex. 46.)

Petitioner visited Dr. Paranandi again on November 10, 2014 for an outpatient follow-up visit. (Ex. 45, pp. 65-72.) Petitioner reported that she went to the emergency room on September 27, 2014, with epigastric and lower retrosternal chest discomfort and discomfort in the interscapular area. (*Id*. at 65.) However, "workup was negative, and no acute coronary syndrome was noted." (*Id*.) Petitioner's diagnostic testing revealed normal results. (*Id*. at 69-70.) Dr. Paranandi stated that petitioner's ICD was functioning well and was stable from the cardiovascular standpoint. (*Id*. at 71.)

In sum, petitioner had frequent medical appointments and testing subsequent to her cardiac episode in October 2011. Petitioner was later diagnosed with benign paroxysmal vertigo and chronic Epstein-Barr Virus. (Ex. 31, p. 5, 9.) Petitioner was also managing and treating her diabetes, thyroid issues, and abdominal pain. (Ex. 45, pp. 11, 63, 91, 302.) Petitioner continued to have issues with her knee and leg swelling, where she started using a cane to ambulate, and started having problems with her memory. (Ex. 34, p. 14; Ex. 49, p. 1; *see* Ex. 50.) During some follow-up appointments, petitioner reported experiencing chest pain, palpitations, and/or shortness of breath and such symptoms were noted under a review of systems, but upon physical examination, petitioner had normal heart rate and regular rhythm. (Ex. 32, p. 17; Ex. 33, p. 12; Ex. 50, p. 5.) On March 6, 2015, petitioner went to the emergency department due to shortness of breath and chest discomfort, but was ultimately discharged on the same day since petitioner was feeling better and her workups were essentially negative. (Ex. 34, pp. 14-23; Ex. 44, p. 143.)

On November 6, 2015, petitioner had a neuropsychological evaluation with Dr. Andrew Houtz. (*Id*. at 121.) During this visit, petitioner again reported that in 2011, while being transported to a hospital for a heart attack, petitioner "went into cardiac

[10] This is incorrect. As described above, the ventricular fibrillation occurred six days after the vaccination.

defibrillation and 'died.'" (*Id*.) Petitioner also reported that when she "'came back to life' she was unable to breathe and immediately began to vomit/cough up blood from 'her lungs.' Since this event, [petitioner] reported that her 'body doesn't feel right.'" (*Id*.) After conducting testing, Dr. Houtz concluded that "[b]ased on these test results, there is little evidence to suggest cognitive dysfunction from a neuropsychological perspective" and that petitioner did not meet the diagnostic criteria for a dementing disorder or mild cognitive impairment. (*Id*. at 125.) Dr. Houtz recommended a conservative treatment approach including specific sleep hygiene activities and relaxation training exercises. (*Id*. at 125-127.)

On June 2, 2016, petitioner had an evaluation for her bilateral knee pain with Dr. Robert Schmidt. (Ex. 48, at 1.) During this visit, petitioner reported that she received a flu shot and had a reaction which resulted in a brief episode of ventricular fibrillation. (*Id*. at 2.) Petitioner also stated that she had a pacemaker in place and that she had some memory loss associated with her cardiac episode. (*Id*.) Dr. Schmidt recommended moving forward with a right knee replacement for petitioner and depending on petitioner's recovery from the procedure, left knee surgery was to follow. (*Id*. at 4.) On August 10, 2016, petitioner underwent total right knee arthroplasty and was subsequently discharged three days after. (*Id*. at 18.) Thereafter, petitioner returned to Dr. Schmidt for post-surgery follow-ups. (*Id*. at 36, 48)

From 2014 to 2017, petitioner went to THP Neurology Specialists of North Texas for follow-ups for her history of depression, numbness and tingling, headaches, chronic fatigue, memory loss, and brain fog. (*See, e.g.*, Ex. 47, pp. 2, 12, 22, 37, 44, 51, 74.) Petitioner complained of having chest pain, palpitations, leg swelling, and shortness of breath several times upon a review of systems during various visits to see her neurologist. (*See*, *e.g.*, *id*. at 5, 15, 25, 39, 46, 54, 84.) On physical examination, petitioner consistently had normal results. (*See*, *e.g.*, *id*. at 5, 16, 26, 40, 47, 54.) Petitioner's past medical and surgical history included ventricular fibrillation, cardiac arrest, and pacemaker placement. (*Id*. at 13, 38, 44, 82.) However, for petitioner's October 13, 2016 and November 6, 2017 visits, petitioner's past medical history included a "cardiac arrest (HCC) due to an overdose of the flu vaccine" and "dementia mild, since cardiac arrest." (*Id*. at 3, 22-23.) Petitioner continued seeking treatment at the THP Neurology Specialists of North Texas for follow-up exams relating to her memory loss and overall neurologic health in 2018 and 2019. (Ex. 52, p. 9, 32, 62.) Petitioner felt that her memory was the same. (*Id*. at 62.)

Additionally, throughout 2015 to 2018, petitioner continued visiting Dr. Paranandi for follow-ups and ICD evaluations. (Ex. 44; Ex. 45, pp. 168-75; 181-201, 355-62.) Petitioner was doing well from a clinical standpoint and her ICD was functioning well. (*Id*. at 140.) Petitioner's stress test was essentially unremarkable and showed that petitioner had normal myocardial perfusion without evidence for ischemia or infraction. (*Id*. at 96.) Petitioner's chest exam on August 5, 2016 showed petitioner had normal heart size and her ICD was in good position. (Ex. 44, p. 122.) Petitioner's echocardiogram from September 11, 2017 showed mild to moderate left ventricular hypertrophy with ejection fraction of 65% and grade 1 diastolic dysfunction was noted.

(*Id*. at 144.) During multiple routine ICD evaluation visits, petitioner complained of shortness of breath. (*Id*. at 74, 140, 147.) During petitioner's chest exam on September 17, 2017, petitioner complained of having a cough and congestion. Dr. Robert Glyoyna reported that "[n]o significant change is evident since" petitioner's chest exam on August 5, 2016. (*Id.* at 110.) Physical examination of petitioner on March 13, 2018, revealed normal results. Further, her ICD was functioning well, and petitioner had no complaints. (*Id*. at 147.) Overall, petitioner was stable from a cardiovascular standpoint. (*Id.* at 141.)

On June 15, 2018, petitioner saw Dr. Robert Dale Anderson for a cardiology consult regarding her ICD. (Ex. 45, p. 343; Ex. 49, p. 1.) Petitioner, again, expressed that she felt her arrest in October 2011 was related to her flu shot. (Ex. 49 at 1.) Petitioner's activity was limited by knee problems and obesity. Petitioner had occasional shortness of breath and orthostatic dizziness but denied any recent chest pain and palpitations. (*Id*.) Dr. Anderson concluded that petitioner's left ventricle function and coronary arteries were normal and encouraged petitioner to continue with weight loss diet. (*Id*. at 5, 15.) Petitioner had another visit regarding her ICD and high blood pressure at Dr. Anderson's office on December 13, 2018. (Ex. 51, p. 1.) Petitioner requested decreasing some of her medications and her cardiologist changed her prescription for metoprolol. (Ex. 51, p. 1,5; Ex. 53, p. 20.)

According to petitioner's most recent ICD report, dated May 2, 2019, the last shock delivered was when the device was first implanted on October 20, 2011. (Ex. 51, p. 22.) Petitioner continued managing her various health problems, but the records do not reflect any significant cardiovascular events resembling petitioner's cardiac episode in 2011.

b. Expert Opinions

Over the course of this case, a total of eleven expert reports were filed by four different cardiology experts over a span of six years. As described above, petitioner initially filed a report by Dr. Waugh. He exchanged views with respondent's initial expert, Dr. Sperling, and his opinion evolved over the course of his four reports. Subsequently, however, both experts ceased participating in the case and additional cardiology experts, Drs. Stark (for petitioner) and LaRue (for respondent), were retained to offer further opinions and to be available for a potential entitlement hearing. Dr. Stark's basis for opining in the case differed from Dr. Waugh's opinion and Dr. LaRue filed reports responsive to both Dr. Stark and Dr. Waugh. Drs. Stark and LaRue both submitted multiple reports. In light of this history, each expert report will be separately discussed in chronological order.

i. <u>Dr. Waugh's Initial Opinion</u>

Petitioner presented an expert opinion from Dr. Robert A. Waugh, then an attending cardiologist at Durham Veterans Administration Hospital and at Duke Clinical Research Institute. (Ex. 25, p.1.) Dr. Waugh received his medical degree at University

of Pennsylvania School of Medicine in 1962 and was licensed as a physician in North Carolina. Additionally, he was board certified in internal medicine and cardiovascular disease. (Ex. 26.) In his first report, Dr. Waugh opined that

> Given the proximate temporal sequence of events with an influenza vaccination on 10/13/2011 and an episode of significant emotional distress shortly thereafter on 10/19/2011 in combination with the absence of significant coronary artery disease by coronary angiography on 10/19/2011, it is plausible that Mrs. Baldwin suffered an episode of coronary spasm on 10/19/2011 (possibly left anterior descending coronary artery) leading to myocardial ischemia and an acute coronary syndrome (ACS) with classic symptoms thereof.

(Ex. 25, p. 3.) He further indicated, however, that "[t]he exact pathophysiologic sequence at the cellular level between vaccination, coronary artery spasm & her emotional distress, remains to be elucidated but coronary artery spasm is a well-recognized phenomenon and, I believe, played a major role in her sequence of events." (*Id*.)

In his second report, he noted the difficulty in identifying vaccine-related injuries epidemiologically due to their rarity, but cited a 2011 study appearing in the Journal of Internal Medicine by Lanza et al, "Inflammation-related effects of adjuvant influenza A vaccination on platelet activation and cardiac autonomic function," as evidence of a mechanism whereby petitioner's flu vaccination may have combined with her ongoing risk factors and emotional stress on the day of the episode to become a substantial factor in bringing about her cardiac event.[11] (Ex. 29, p. 1 (citing Lanza et al., *supra*, at Ex. 28).)

Dr. Waugh cited the Lanza study for the proposition that "significant correlations were found between C-reactive protein (CRP) (a response to inflammation) and heart rate variability." (Ex. 29, p. 2.) He opined that "[t]his suggests a pathophysiological link between inflammation and cardiac autonomic dysregulation which, in turn, increases the risk of cardiovascular events." (*Id*.) Indeed, the Lanza authors concluded that:

> In this study, we have shown that influenza A vaccination in patients with type II diabetes included, together with the expected inflammatory reaction, an increase in platelet activation and a cardiac sympathovagal imbalance. Overall, the vaccine-induced changes in platelet activity and autonomic

[11] The Lanza study examined twenty-eight patients with type II diabetes over the course of four days. (Lanza et al., *supra*, at Ex. 28, pp. 1-2.) On day one, patients underwent 24-hour ambulatory electrocardiogram ("ECG") monitoring and had a blood sample collected. On day two, the patients were administered an influenza A vaccination. On day three, patients had a second 24-hour ECG recording. Additional blood samples were drawn 24 and 48-hours after the vaccination. (*Id*.) C-reactive protein ("CRP"), interleukin-6 levels, monocyte-platelet aggregates ("MPAs") and monocyte/platelet receptor expression were measured. (*Id*. at 1.) A control group of twelve patients underwent the same testing but did not receive any vaccination. (*Id*. at 3.)

nervous activity may transiently increase the risk of cardiovascular events in vaccinated patients.

(Lanza et al., *supra*, at Ex. 28, p. 7.)

Dr. Waugh stressed that, although petitioner had ongoing cardiac risk factors of obesity, hypertension, and type II diabetes, her heart function had been unremarkable, and she had no notable findings on angiography or ventriculography following her cardiac event. (Ex. 29, p. 2.) He noted that her lab results were unremarkable, except for expected elevated troponin levels, and that she had no cardiac enzymes for myocardial injury. For these reasons, he opined that a transient heart rate variability brought on by inflammation was likely. (*Id*.)

Dr. Waugh acknowledged that the Lanza study ended after only 48-hours but suggested that the autonomic nervous system dysfunction identified in the study would persist for as long as the inflammation was present. (Ex. 29, p. 2.) He therefore opined that a six-day interval would be plausible. (*Id*.)

In his third report, Dr. Waugh stressed that he was not relying on the Lanza study's findings regarding increased platelet activation as leading to a clotting risk. (Ex. 35, pp. 1-2.) There is no indication in petitioner's history that she had a clotting injury such as stroke, embolism or heart attack. (*Id*. at 2.) Rather, he was relying on the Lanza study's finding of sympathovagal imbalance, which is an indication of disturbed autonomic function evidenced by increased CRP. (*Id*.) Dr. Waugh noted that, although the Lanza study concluded after only 48 hours, the study found progressively higher CRP levels at 24 and 48-hours post-vaccination, suggesting that CRP levels had not yet peaked. (*Id*.) He also noted that it takes the body a week or more to clear the wild flu virus. (*Id*. at 1.)

ii. Dr. Sperling's Responsive Opinion

Respondent provided a competing opinion from Dr. Laurence S. Sperling, cardiologist and current professor at Emory University School of Medicine and director of The Center for Heart Disease Prevention. Dr. Sperling is board certified in internal medicine and cardiovascular disease. Dr. Sperling previously served as a special cardiovascular consultant to the Center for Disease Control and Prevention. (Ex. A, p. 1.) Dr. Sperling received his medical degree from Emory University School of Medicine in 1989 and is licensed to practice in Georgia. (Ex. B.) Dr. Sperling opined that petitioner "experienced a ventricular fibrillation arrest that in all probable likelihood was due to uncharacteristic physical exertion and profound emotional distress," highly unlikely to be related to her October 13, 2011 vaccine from six days earlier. (Ex. A, p. 5.)

Dr. Sperling concurred with petitioner's treating cardiologists that petitioner had probable coronary artery spasm and elevated heart rate. (Ex. A, p. 3.) However, Dr. Sperling emphasized that "[t]he increase in physical activity in a physically disabled

person coupled with emotional distress in all likelihood resulted in an increase in heart rate and abnormalities in vasomotor tone." (*Id*.) Moreover, Dr. Sperling believed that petitioner's pre-existing conditions, i.e., her morbid obesity, type II diabetes, and hypertension, predisposed her to acute cardiovascular events and underlying endothelial dysfunction. Dr. Sperling further opined that "[t]he elevated pulse secondary to physical exertion and emotional distress provided an extra cardiac burden that may have resulted in coronary artery spasm and then an unstable cardiac rhythm." (*Id*.)

Dr. Sperling contested Dr. Waugh's reliance on the Lanza article to suggest that influenza vaccine can cause petitioner's cardiac problem and hospitalization. (*Id*. at 4.) Dr. Sperling contrasted petitioner's cardiac event occurring approximately 140 hours after vaccination with the cases in the Lanza study, where inflammatory markers were measured at 24 and 48 hours post vaccination. (*Id*.) Dr. Sperling, instead, asserted that "the overwhelming preponderance of scientific data supports the influenza vaccine as being cardioprotective." (*Id*.) Dr. Sperling cited two articles that involved over 26,000 patients in total, showing no adverse cardiovascular effect following flu vaccination. Dr. Sperling highlighted that the Udell article noted a significant decrease in cardiovascular events in patients that were considered of high cardiovascular risk and that the Smeeth article concluded that the "mild transient inflammation induced by vaccination does not appear to translate into a detectable increase in the risk of vascular events." (*Id*. (citing Jacob A. Udell et al., *Association Between Influenza Vaccination and Cardiovascular Outcomes in High-Risk Patients: A Meta-Analysis*, 310 (16) JAMA 1711 (2013) (Ex. C); Liam Smeeth et al., *Risk of Myocardial Infraction and Stroke After Acute Infection or Vaccination*, 351 N. ENG. J. MED. 2611 (2004) (Ex. D).) Therefore, Dr. Sperling posited that petitioner's sympathetic nervous system was engaged secondary to physical and emotional exertion, not her flu vaccination. (Ex. A, p. 4.)

iii. Dr. Waugh's Response

Dr. Waugh responded to Dr. Sperling's report, stressing that "there was nothing wrong with [petitioner's] heart," and that despite having a history of "multiple *putative* cardiac risk factors, none has proved to have caused her any harm." (Ex. 36, p. 1 (emphasis original).) Dr. Waugh attributed petitioner's pre-vaccine symptoms (rapid heartbeat and dizziness) to her orthostatic hypertension and her hypertension to her diabetes. (*Id*.) However, Dr. Waugh stated that, on the day of her cardiac event, "the only variable not previously present was the influenza vaccine." (*Id*. at 2.) Dr. Waugh reiterated that petitioner's pre-vaccine objective testing never identified any heart problems. (*Id*. at 1.) Dr. Waugh noted that although there was a reference in petitioner's admission notes for the day of the cardiac arrest that petitioner had a tentative coronary artery spasm, no objective test found evidence of such spasm and in fact coronary artery spasm is a chronic condition, a condition petitioner did not have. (*Id*. at 2.) Therefore, Dr. Waugh disagreed with Dr. Sperling's reliance on the notation regarding coronary artery spasm to counter his causation theory. Dr. Waugh further criticized the articles relating flu vaccines to cardiovascular protection as overly generalized as to apply to petitioner's specific factors in this case. (*Id*.)

Considering petitioner's significant medical history filled with various stressful events, Dr. Waugh found merely conjecture Dr. Sperling's theory that petitioner's plumbing problems on the day of her cardiac arrest were enough to have caused such a profound emotional shock that would cause the cardiac arrest. (*Id*. at 1.) Dr. Waugh indicated that the "type of heart dysregulation that is emotionally driven is called Takotsubo cardiomyopathy, which has distinctive markers on MRI and X-ray." (*Id*.)

Dr. Waugh, again, cited to the Lanza study as directly applicable in this case. (*Id*. at 2.) He explained the data collected showed a trend of *increasing* numbers, not peaked numbers, of inflammatory markers when measured at 24 hours and 48 hours. (*Id*.) And therefore, Dr. Waugh opined that it is "logical to conclude that inflammatory markers remained manifest four days later when perhaps the added stressor of trying to stop a leaky sink was the final straw leading to the event." (*Id*.)

iv. Dr. Sperling's Further Response

In response, Dr. Sperling agreed that a "definitive etiology in regard to [petitioner's] acute cardiovascular event is not possible to determine," but it is highly unlikely that petitioner had an acute episode of Takotsubo cardiomyopathy (TTC) as Dr. Waugh suggested in his supplemental report.[12] (Ex. E, p. 1.) However, he emphasized a point made in the Sharkey article that Dr. Waugh cited, which claimed that "'in 85% of cases, TTC was triggered by an emotionally or physically stressful event that preceded onset of symptoms by minutes to hours.'" (*Id*. at 3 (citing Scott W. Sharkey, John R. Lesser & Barry J. Maron, *Cardiology Patient Page: Takotsubo (Stress) Cardiomyopathy*, 124:18 CIRCULATION e460 (2011) (Ex. 38)).) Additionally, to rebut Dr. Waugh's assertion that petitioner experienced far more significant stressors than plumbing problems, Dr. Sperling added that the Sharkey article also found "it is not understood why a specific stressful event will on one occasion trigger [TTC] whereas at another time a similar circumstance (even more stressful) does not." (Ex. E, p. 4 (citing Sharkey, Lesser & Maron, *supra*, at Ex. 38).)

Again, Dr. Sperling reemphasized that the Lanza article does not support petitioner's causation theory since there was no data beyond 48 hours and no indication that the numbers would continue to rise thereafter. (Ex. E, p. 3.) Instead, Dr. Sperling stated, citing to the Tsai and Christian articles, that "the available studies have failed to document any significant increase in markers of inflammation, specifically CRP, beyond 3 days post vaccine." (*Id*. (citing M.Y. Tsai et al., *Effect of Influenza Vaccine on Markers of Inflammation and Lipid Profile*, 145 J. LAB. CLINICAL MED. 323 (2005) (Court Exhibit I); Lisa Christian et al., *Inflammatory Responses to Trivalent Influenza Virus Vaccine Among Pregnant Women*, 29 VACCINE 8982 (2011) (Court Exhibit II)).) Moreover, Dr. Sperling found the Singh study more relevant than the Lanza article to petitioner's case. The study "explored defibrillator firing in patients who are at high risk for malignant arrhythmias" and found that there was a "trend towards an increase of number of ICD

[12] Dr. Waugh did not actually suggest petitioner experienced TTC. He cited TTC as a basis for distinguishing what petitioner experienced from cardiac events caused by emotional shock. (Ex. 36, p. 1.)

therapies in unvaccinated patients as compared to vaccinated patients." (Ex. E, p. 3 (citing Sheldon M. Singh, Russel J. de Souza & Ramanan Kumareswaran, *Increased Defibrillator Therapies During Influenza Season in Patients Without Influenza Vaccines*, 31 J. OF ARRYTHMIA 210 (2015) (Ex. G)).) Additionally, the study found that after receiving the flu vaccine, even the patients who were at high risk for developing arrhythmias did not experience an increase in malignant arrythmias. (Ex. E, pp. 3-4.) Therefore, Dr. Sperling opined that the flu vaccine, "despite being temporally related," did not play any role in petitioner's cardiac event on October 19, 2011. (*Id*.)

v. Dr. Stark's Initial Opinion

Following Dr. Waugh's retirement and a change of counsel, petitioner provided an opinion from Dr. Robert M. Stark to further support her petition. Dr. Stark graduated from Harvard Medical School in 1970 and is board certified in internal medicine and cardiology. (Ex. 40.) He is now an adjunct professor at New York Medical College and attending physician at Greenwich Hospital in Connecticut. (*Id*. at 1.) Dr. Stark additionally is an instructor for the American Heart Association's course in Advanced Cardiac Life Support (ACLS). (Ex. 39, p.1.)

Dr. Stark opined that petitioner's flu vaccination triggered an antigen-antibody reaction that inflamed the arteries and such inflammation "can cause the platelets in smaller blood vessels to clump together and stick to the vessel wall," causing petitioner's cardiac arrest. (Ex. 39, pp. 2-3.) Dr. Stark explained that spontaneous ventricular fibrillation could be a result of ischemic coronary disease, microvascular disease, or arrhythmogenic focus in the myocardium. However, given petitioner's negative stress test, normal cardiac catherization, and lack of any previous cardiac problems (arrythmias, coronary artery lesions, fainting), Dr. Stark ruled out coronary artery disease and any pre-existing arrhythmogenic focus. (*Id*.) He stated, "[n]ot until [petitioner] received a flu vaccine (six days before) did she have her first episode of chest pain, collapse and cardiac arrest." (*Id*. at 2.)

vi. Dr. LaRue's Responsive Opinion

Following Dr. Sperling's departure from this case, respondent filed an expert report from Dr. Shane R. LaRue, responding to both Dr. Waugh's and Dr. Stark's expert opinions. Dr. LaRue graduated from Medical College of Wisconsin in 2005 and received a master's degree in population health sciences at Washington University of School of Medicine in 2012. (Ex. I.) He is board certified in internal medicine, advanced heart failure and transplant cardiology, adult echocardiography, and cardiovascular diseases. (*Id*. at 2.) Dr. LaRue is now an assistant professor in the cardiovascular division at Washington University School of Medicine. (*Id*.)

Based on petitioner's medical records, Dr. LaRue provided an alternative theory of causation, opining that "it is far more likely that [petitioner] experienced [ventricular fibrillization] arrest due to her underlying risk and acute trigger." (Ex. H, p. 5.) He noted that both petitioner's pre and post cardiac arrest electrocardiograms indicated

poor r-wave progression (PRWP) in the precordial leads and this suggests that petitioner has left ventricular hypertrophy (LVH) or an abnormality in the mid-anterior wall of her left ventricle. (*Id*. at 4.) Dr. LaRue additionally emphasized that petitioner's ECG indeed documented that petitioner has LVH and "LVH has a well-established association with ventricular arrhythmias." (*Id*.) He cited to the McLenachan and Haider articles that found hypertensive individuals with LVH experienced higher frequencies of ventricular premature contractions and that LVH was a risk factor for sudden death, respectively. (Ex. H, p. 4 (citing James M. McLenachan et al., *Ventricular Arrythmias in Patients with Hypertensive Ventricular Hypertrophy*, 317 N. ENGLAND J. MED. 787 (1987) (Ex. K); Agha Haider et al., *Increased Left Ventricular Mass and Hypertrophy are Associated with Increased Risk for Sudden Death*, 31 J. AM. COLLEGE Cardiology 1454 (1998) (Ex. L)).) Additionally, Dr. LaRue opined, citing to the Gami study, that petitioner's obstructive sleep apnea (OSA) increased her risk for ventricular arrythmia and sudden cardiac death. (Ex. H, pp. 4-5 (citing Apoor S. Gami et al., *Obstructive Sleep Apnea and the Risk of Sudden Cardiac Death: A Longitudinal Study of 10,701 Adults*, 62 J. AM. COLLEGE CARDIOLOGY 610 (2013) (Ex. M)).) Lastly, he opined that the emotional and physical stress significantly contributed to petitioner's cardiac event. (EX. H, p. 5.) Dr. LaRue cited to the Lampert study that found elevated levels of anger and anxiety and mild-to-moderate physical activity significantly preceded ICD shocks. (*Id*. (citing Rachel Lampert, *Emotional and Physical Precipitants of Ventricular Arrythmia*, 106 CIRCULATION 1900 (2002) (Ex. N)).) Dr. LaRue further provided studies that test the relationship between influenza vaccine and ventricular arrhythmias, suggesting that flu vaccinations have been associated with lower rates of cardiovascular morbidity and mortality. (Ex. H, p. 5.)

Moreover, Dr. LaRue assessed the opinions of Drs. Waugh and Stark. Dr. LaRue suggested that Dr. Waugh's multiple reports contained contradictions and further emphasized that Dr. Waugh essentially offered two different etiologies (coronary artery spasm and emotional stress), both unrelated to flu vaccine, to explain ventricular arrhythmias. (*Id*. at 6.) Dr. LaRue then stressed that the "influenza vaccine has been studied in numerous trials, in hundreds of thousands of people, and no reports of associated ventricular arrythmias," and in fact, there are "specific recommendations to vaccinate people with coronary artery disease and hear failure, two populations that have an increased risk of ventricular arrythmias." (*Id*. at 7.)

Dr. LaRue disagreed with Dr. Waugh's opinion that "there was nothing wrong with [petitioner's] heart." (*Id*. at 8.) Addressing the medical record, Dr. LaRue noted that petitioner's ECG demonstrated mild LVH and petitioner "experienced at least 13 episodes of ventricular tachycardia or non-sustained ventricular tachycardia … over the 11 months immediately following her arrest." (*Id*.)

Regarding the temporal relationship, Dr. LaRue noted that the Lanza article did not indicate when inflammation peaked and when it normalized, and it is "not logical to think inflammation would persist an indefinite time after a vaccine," especially when medical literature indicates that inflammation resolved by day five. (*Id*. at 7-9.) Moreover, Dr. LaRue opined that petitioner was not in a pro-thrombotic state due to her

vaccination at the time of her cardiac arrest. (*Id*. at 8.) According to Dr. LaRue, Dr. Waugh conflated the "body's response to influenza infection and vaccination with an inactivated influenza virus" and therefore, "the time course of one does not allow meaningful inference in the other." (*Id*.)

In turn, Dr. LaRue cited to the Christian article, which measured levels of CRP for one week after vaccination and found significant increases in CRP one and two days after vaccination, but no difference after one week. (*Id*.) Additionally, Dr. LaRue referenced the Posthouwer study that measured both CRP and thrombin activation after influenza and pneumococcal vaccines. He indicated that the Posthouwer study found that CRP peaked at two or three days after vaccination but returned to baseline on the fourth and fifth day. (*Id*. at 7-8 (citing Dirk Posthouwer et al., *Influenza and Pneumococcal Vaccination as a Model to Assess C-Reactive Protein Response to Mild Inflammation*, 23 VACCINE 362 (2004) (Ex. R)).)

Dr. LaRue also rebutted Dr. Stark's opinion, emphasizing that petitioner's cardiac catherization report did not "suggest [petitioner] had an acute myocardial infarction of a small vessel such as slow coronary flow or a vessel that was occluded." (Ex. H, p. 4.) He again pointed to petitioner's ECGs (indicating poor r wave progression) and post arrest ventricular arrhythmias. (*Id*. at 9.) Specifically, he indicated petitioner "may well have had an arrhythmogenic focus that had not yet manifest, and finally did so in the setting of the catecholamine surge of her stressful situation." (*Id*.) Further, Dr. LaRue again pointed to the Posthouwer study, which showed CRP and thrombin activation to return to baseline at day four and five post-vaccination, to rule out the possibility of petitioner's cardiac arrest being caused by clotting due to vaccine-induced inflammation. (*Id*.) Additionally, he found the two articles Dr. Stark cited inapplicable to petitioner's case. (*Id*.) The first article pertained to vasculitis involving cerebral and coronary arteries and the second article described bodily responses to influenza infection, which were all not related to petitioner's conditions and theory of causation. (*Id*.)

Ultimately, Dr. LaRue concluded that, considering the lack of reported cases relating flu vaccine to ventricular arrhythmias or sudden cardiac death, petitioner's flu vaccine, more likely than not, did not contribute to petitioner's ventricular fibrillation arrest. (*Id*. at 10.) He opined that petitioner more likely experienced a ventricular fibrillation from her underlying hypertensive heart disease and obstructive sleep apnea and was triggered by the immediately preceding emotional event. (*Id*.)

vii. Dr. Stark's Response

Dr. Stark addressed Dr. LaRue's theory that petitioner's obstructive sleep apnea and mild ventricular hypertrophy caused her cardiac arrest in his responsive expert report. (Ex. 43.) First, Dr. Stark conceded that petitioner's combined cardiac risk factors (overweight, diabetes, hypertension, hypercholesterolemia) could lead to coronary obstructions, but petitioner's testing results (electrocardiograms, stress test, catherization) in fact showed petitioner was "free of any obstructions." (*Id*. at 1.) Next, Dr. Stark refuted Dr. LaRue's suggestion that petitioner's sleep apnea was a causal

factor by emphasizing that "sleep apnea patients face their risk for arrhythmia when their blood oxygen levels decline to low levels during night time sleep" whereas petitioner had her cardiac arrest when she was not asleep and during the day. (*Id*.) Suggesting that "virtually all patients with hypertension have some degree of left ventricular hypertrophy," Dr. Stark opined that petitioner's mild left ventricular hypertrophy "is a negligible contributor to potential ventricular fibrillation or cardiac arrest." (*Id*. at 2.)

Regarding petitioner's post-vaccination cardiac condition, Dr. Stark explained that the "detected and recorded 13 episodes of ventricular tachycardia," could indicate that there was an arrhythmogenic focus, but most likely, the focus came from the area that was damaged when petitioner had her cardiac arrest. (*Id*. at 3.) He further asserted that the fact of these post-arrest arrhythmias "in no way proves that [petitioner] was already prone to arrythmias prior to her 10/13/11 flu immunization and subsequent cardiac arrest." (*Id*.)

Dr. Stark expanded on his theory of post-influenza immunization inflammation and clotting by referencing the Falsey study, which had data showing increasing numbers of HI antibody titers occurring over 28 days, suggesting that "antigen-antibody mediated inflammatory responses [can] extend substantially beyond the hypothetical five day period following immunization that was outlined by Dr. LaRue." (*Id*. at 2-3 (citing Ann Regina Falsey et al., *Randomized, Double-Blind Controlled Phase 3 Trial Comparing the Immunogenicity of High-Dose and Standard-Dose Influenza Vaccine in Adults 65 Years of Age and Older*, 200(2) J. INFECTIOUS DISEASE 172 (2009) (Ex. 53)).) Additionally, Sanofi Pasteur reported that 21.4 to 35.6% of patients receiving the "earlier formulations of Fluzone" had local and systemic inflammatory reactions within the first three to seven days. (Ex. 43, p. 2 (citing Sanofi Pasteur, *372 Fluzone High-Dose* (March 29, 2019) (Ex. 55)).) Dr. Stark also found the Christian study testing inflammation following flu vaccinations in pregnant woman not applicable to petitioner's case and further that Dr. LaRue's analysis of post-immunization clotting ability, based on the Posthouwer article, was limited to thrombin activation while a "myriad of potential measures of clotting abilities" exist that "might well explain the change in clotting ability that has been observed following influenza immunization." (Ex. 43, pp. 2-3.)

viii. Dr. LaRue's Further Response

Respondent filed a supplemental expert report from Dr. LaRue, addressing the points raised in Dr. Stark's supplemental report. (Ex. S.) Dr. LaRue clarified that his prior discussion of petitioner's cardiovascular risk factors was not to suggest that petitioner suffered a coronary obstruction, but instead that petitioner had risks for cardiac arrythmias and sudden cardiac death. (*Id*. at 1.) Moreover, Dr. LaRue noted that there is an increased risk of obstructive sleep apnea induced arrythmias during the daytime due to the "increase in sympathetic drive," although the greatest risk remained during nighttime. (*Id*.) In regard to Dr. Stark's opinion that mild LVH has negligible arrhythmic risk, Dr. LaRue noted the McLenachan trial, finding higher incidence of ventricular arrythmias in hypertensive patients with LVH than without, and the Haider

study, finding higher incidence of sudden cardiac death in patients with LVH than those without. (*Id*. at 1-2 (citing McLenachan et al., *supra*, at Ex. K).) Both studies did not find severity of LVH to be a criterion. (Ex. S, pp. 1-2.) Additionally, Dr. LaRue stressed the significance of petitioner's recorded mid-anterior hypokinesis and poor r wave progression. (*Id*. at 2.)

Regarding inflammation, Dr. LaRue agreed with Dr. Stark that inflammatory mediators exert an effect on vascular endothelium, but in this case, there is no evidence indicating that petitioner experienced significant inflammation six days post-vaccination. (*Id*.) Dr. LaRue next assessed the Falsey article, opining that "[p]resence of antibody 28 days after vaccination is evidence only of immunity, not of ongoing inflammation." (*Id*. at 2.) Regarding clotting, Dr. LaRue agreed that many measures of clotting ability exists; however, "[t]hrombin is required both for the initiation and the propagation of the clotting cascade and thus elevation in prothrombin fragment is seen in a hypercoagulable state," showing that the Posthouwer article is indeed relevant to this case. (*Id*. at 2-3.) Regarding petitioner's post-arrest ventricular tachycardia, Dr. LaRue disagreed with Dr. Stark that the focus originated from the area damaged from petitioner's cardiac arrest. He stated such damage would be evident in an electrocardiogram, but petitioner's ECG both pre- and post-arrest were essentially undifferentiated. (*Id*. at 3.)

ix. Dr. Stark's Final Response

On February 5, 2020, Dr. Stark provided a further supplemental expert opinion focusing on two points. (Ex. 54.) Dr. Stark addressed petitioner's "proven absence of underlying coronary artery disease, and the well-known mechanism of immune and inflammatory reactions in causing coronary events." (*Id*. at 1.) Again, Dr. Stark stressed that although petitioner had several risk factors for coronary atherosclerosis, petitioner did not have coronary atherosclerosis. (*Id*.) Dr. Stark opined that the "absence of significant coronary disease makes the alternative of coronary inflammation far more likely" and that petitioner's known diabetes predisposes her to small vessel inflammation and spasm. (*Id*.) Dr. Stark emphasized that systemic inflammation from recent flu immunization can cause a cardiac event, where the mechanism is "virtually identical to that for influenza infection." (*Id*.) Again, Dr. Stark highlighted the Lanza study, showing that for diabetic patients, there were "significant rises in inflammatory markers as well as platelet activation and adrenergic activation which are known to predispose to heart attack." (*Id*. at 2.)

IV. Discussion

a. *Althen* Prong One

Under *Althen* prong one, petitioner must provide a "reputable medical theory," demonstrating that the vaccine received can cause the type of injury alleged. *Pafford v. Sec'y of Health & Human Servs.*, 451 F.3d 1352, 1355–56 (Fed. Cir. 2006) (citations omitted). Such a theory must only be "legally probable, not medically or scientifically

certain." *Id.* at 549. 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. *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 [her] theory. While it does not require medical or scientific certainty, it must still be 'sound and reliable.'" *Boatmon v. Sec'y of Health & Human Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019) (quoting *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548-49 (Fed. Cir. 1994)).

Petitioner explains that her "medical theory centers upon the inflammation caused by the antigen-antibody complex triggered by the influenza immunization." (ECF No. 92, p. 7.) She suggests that her theory can be separated into two parts - first, the influenza vaccine causes inflammation, and, second, that inflammation can in turn cause a ventricular fibrillation cardiac event. (*Id*. at 8.) Upon my review, I find that petitioner has at best demonstrated only the first part of her two-part theory. The first part of petitioner's theory – that influenza vaccination causes inflammation (to at least some degree) – is not meaningfully disputed. Both parties, as well as the previously-assigned special master, have filed literature finding increased levels of inflammatory markers following influenza vaccination, most notably C-reactive protein ("CRP"). (Lanza et al., *supra*, at Ex. 28; Posthouwer et al., *supra*, at Ex. R; Christian et al., *supra*, at Court Exhibit I.) The second part of petitioner's theory, however, is not persuasively addressed.

With regard to that second part of her theory, petitioner acknowledges that "what is so difficult about Mrs. Baldwin's injury is that the exact mechanism of how and why she experienced a ventricular fibrillation event will never be elucidated to a level of scientific certainty." (ECF No. 92, p. 8.) Nonetheless, she posits that her experts – Drs. Waugh and Stark – "discuss various mechanisms as to how inflammation can cause ventricular fibrillation." (*Id*.) In that regard, Dr. Waugh and Dr. Stark propose two different mechanisms of injury. Dr. Waugh suggests that petitioner experienced an arrhythmia brought on by "chemical-electrical instability" in the heart. (Ex. 29, p. 2; Ex. 35, p.1.) Dr. Stark proposes that petitioner's inflammation led to a prothrombotic state which led to small blockages in microcirculation. (Ex. 39, pp. 1-2.)

Dr. Waugh was the first to opine. Apart from his *ipse dixit*, Dr. Waugh's theory of causation is supported by a single study. As described above, in Lanza et al., twenty-eight patients with type II diabetes were monitored in connection with administration of adjuvant influenza A vaccines. (Lanza et al., *supra*, at Ex. 28.) The purpose of the study was to investigate "the effect of an inflammatory stimulus (influenza A vaccine) on platelet activation and cardiac autonomic function." (*Id*. at 1.) The study authors noted that "the exact relation between [autonomic nervous system] activity and inflammation in the clinical setting remains unclear." (*Id*. at 2.) They further noted, in particular, that "how inflammatory stimuli affect sympathovagal balance in humans has not been explored." (*Id*.) Accordingly, the authors characterize their study as the first of its kind. (*Id*. at 4.)

Measurements of CRP and interleukin-6 (to assess inflammation) as well as monocyte-platelet aggregates (MPAs) and monocyte/platelet receptor expression (to assess platelet activation) were taken before and after vaccination. Electrocardiograms were recorded and heart rate variability (HRV) was assessed. (*Id*. at 1.) The authors concluded that "[s]ignificant correlations were found between CRP levels and HRV parameters, suggesting a pathophysiological link between inflammation and cardiac autonomic regulation." (*Id*.) This, they posited, suggests that there may be a transient increase in the risk of cardiovascular events. (*Id*.) The Lanza study is intriguing as a piece of preliminary investigation. However, it falls well short of supporting petitioner's theory that post-vaccination inflammation can lead to ventricular fibrillation and/or cardiac arrest.

First, the study findings are not all favorable to petitioner's theory. Although Dr. Waugh mostly stressed the relationship between CRP and HRV, both Dr. Waugh, and later Dr. Stark, indicated that the pro-thrombotic state discussed in Lanza is significant to their theories. (Ex. 35, p. 1; Ex. 39, p. 2.) As Dr. Waugh explained it, the correlation between elevated CRP and reduced HRV "predicts" destabilized autonomic function (Ex. 35, p. 2), but the chemical-electrical instability that leads to arrhythmia is a result of the pre-clotting or pro-thrombotic state. (Ex. 35, p. 1.) Specifically, he opined that "[f]lu shots also produce platelet activation which in turn leads to autonomic nervous system (ANS) sympathetic-parasympathetic imbalance favor adrenergic predominance." (Ex. 29, p. 2.) However, the Lanza study failed to find any correlation between increased platelet activation (as measured by MPA) and either CRP levels or HRV variables. (Lanza et al., *supra*, at Ex. 28, p. 4.) The authors explained that "[t]he increase in MPA, however, did not show any significant correlation with HRV changes, suggesting that the acute cell-mediated inflammatory response to viral vaccine did not result in appreciable effects on cardiac autonomic activity. Accordingly, no significant relation was found between HRV changes and platelet activation in this context."[13] (*Id*. at 7.)

Second, even focusing on the CRP/HRV correlation found within the study, to the extent Dr. Waugh suggested that CRP merely "predicts" HRV changes (Ex. 35, p. 2), he effectively suggests (and I agree) that the Lanza study shows only a correlation between elevated CRP and reduced HRV and not a causal relationship. In that regard, the Lanza authors noted that they conducted a separate study (not a part of this record) which found that administration of beta-blockers not only improved HRV, but also reduced CRP levels. This, they explained, "suggest[s] that the improvement of sympathovagal balance by beta-blockers may translate into anti-inflammatory effects." (Lanza et al., *supra*, at Ex. 28, p. 6.) Thus, although the authors posit a

[13] Nonetheless, petitioner stresses in her brief language from the Lanza study indicating that increased MPA formation may suggest development of a pro-thrombotic state which, in turn, has previously been associated with acute coronary events. (ECF No. 92, p. 11 (quoting Lanza et al., *supra*, at Ex. 28, p. 5).) However, petitioner has not presented the cited literature purporting to associate MPAs with acute coronary events. Moreover, the Lanza study did not describe what type of acute coronary events had previously been associated to a pro-thrombotic state. Additionally, as noted above, the Lanza study itself found no relation between platelet activation and the HRV changes that are the basis for petitioner's theory and her reliance on this article.

"pathophysiological link" between inflammation and cardiac sympathovagal balance, they cautioned that this link is "complex" and indirectly suggest that the nature (and directionality) of the relationship has not necessarily been established.

Finally, the Lanza study is very small, with only twenty-eight patients, and does not report, nor is it likely sufficiently powered to detect, any actual clinical manifestation of the cardiovascular risk the authors posit. In that regard, respondent has cast some doubt on the clinical significance of these findings by noting that larger scale meta analyses have found that the flu vaccine is likely to be cardio protective. That is, the risk of adverse cardiac events has been shown to be decreased among those vaccinated against influenza.[14] (*See*, *e.g.*, Udell et al., *supra*, at Ex. C; Rohit S. Loomba et al., *Influenza Vaccination and Cardiovascular Morbidity and Mortality: Analysis of 292 383 Patients*, 17 J. CARDIOVASCULAR & THERAPEUTICS 277 (2012) (Ex. P)). Moreover, Dr. Sperling also cited the Singh study, which explored defibrillator firing in patients who are at high risk for malignant arrhythmias and found that there was a "trend towards an increase of number of ICD therapies in unvaccinated patients as compared to vaccinated patients." (Ex. E, p. 3 (citing (Ex. G) (Singh).) Those findings do not negate the Lanza study, but they do suggest, especially in conjunction with the above-discussed points, that the results should be interpreted with caution. Moreover, respondent's expert, Dr. Sperling persuasively explained that "[a]lthough C-reactive protein (CRP) is an often used inflammatory marker it is quite non-specific and impacted by lifestyle and behavior factors, [and] medications. Importantly, stress, anxiety, and depression can also increase levels of CRP." (Ex. E, p. 3.) He opined that the Lanza study is too small and controls for too few of these variables to be broadly applicable. (*Id*.)

Given these limitations, it is also worth noting that Dr. Waugh himself characterized his theory as one "plausible" mechanism among "a number of possible mechanisms" for petitioner's cardiac event. (Ex. 29, p 2.) Standing alone, this would be insufficient to meet petitioner's burden. *See, e.g.*, *Boatmon*, 941 F.3d at 1360 (stating that "[w]e have consistently rejected theories that the vaccine only 'likely caused' the injury and reiterated that a 'plausible' or 'possible' causal theory does not satisfy the standard.").

Of course, petitioner also submitted an additional opinion by Dr. Stark. Dr. Stark posited a relationship between post-vaccination inflammation and the pro-thrombotic effect. (Ex. 39, p. 2.) He stated that "the inflammatory state, caused by flu vaccine-

[14] Of course, petitioner is not obligated to come forward with epidemiological evidence. *Andreu*, 569 F.3d at 1380. Moreover, Dr. Waugh addressed the difficulty of detecting adverse events following vaccination due to their rarity. (Ex. 29, p. 1.) Additionally, it must be noted that the perceived cardio protective effect of the influenza vaccine evidenced in epidemiological studies may likely result from a reduction in the rate of influenza infection among vacinees rather than from characteristics of the vaccine itself. Indeed, petitioner has filed literature by Gurevich, et al., discussed below, that suggests influenza infection is associated with an increase in deaths from cardiovascular disease. (Ex. 42.) This makes assessment of the true significance of this epidemiological evidence very difficult. Accordingly, epidemiologic evidence of a cardio protective effect from the influenza vaccine, though relevant, is not in itself dispositive.

induced antigen-antibody inflammation, can cause the platelets in smaller blood vessels to clump together and stick to the vessel wall. This can result in small blockages in the coronary microcirculation." (*Id*. at 1-2.) He also suggested, more generally, that the flu vaccine can "inflame the arteries (arteritis) including the coronary arteries." (*Id*.)

Like Dr. Waugh, Dr. Stark also relied on the Lanza study. (Ex. 54.) He suggested that the study "demonstrated significant rises in inflammatory markers (c-reactive protein and interleukin) as well as platelet activation and adrenergic activation which are known to predispose to heart attack." (Ex. 54, p. 2.) Dr. Stark's reliance on this study is unpersuasive for all the same reasons discussed above regarding Dr. Waugh's opinion. In further support of his opinion, however, Dr. Stark also submitted a review article by Gurevich et al. and a case report by Ritter et al. (O. Ritter et al., *Myocardial Infarction After Influenza Vaccination*, 92 Z. KARDIOL 962 (2003) (Ex. 41); Victor S. Gurevich, Vladimir M. Pleskov & Margarita C. Levaya, *Autoimmune Nature of Influenza Atherogenicity*, 1050 ANN. N.Y. ACAD. SCI 410 (2005) (Ex. 42).) Neither of these submissions significantly support either Dr. Stark's or Dr. Waugh's opinions or suggest any greater weight should be attributed to the Lanza study.

Gurevich et al., reviewed prior studies, including both population-based studies and clinical trials, suggesting that influenza *infection* was associated with increased death rates from cardiovascular disease. (Gurevich, Pleskov & Levaya, *supra*, at Ex. 42, p. 1.) The authors propose that influenza infection may contribute to progression of atherosclerotic vascular injury through an autoimmune process. (*Id*. at 6.) Significantly, however, in a section devoted to reviewing influenza vaccination, the authors acknowledge that prior studies have observed a negative association between influenza vaccination and myocardial infarction. (*Id*.) Importantly, the authors also noted that a prior study showed that patients with a high risk of thrombosis were among those populations that received greater protective benefit from vaccination. (*Id*.) This would appear to cut directly against Dr. Stark's theory. Although Gurevich et al. stressed that influenza vaccination has been associated with autoimmune reactions *generally*, they cited no evidence to support any relationship between the influenza *vaccination* and adverse cardiac events. Rather, they indicated only that the proposed relationship is "worthy of attention."[15] (*Id*.)

Ritter et al., noted a case report of a single female patient who experienced secondary vasculitis and myocardial infarction after influenza vaccination. (Ritter et al., *supra*, at Ex. 41, p. 1.) Though entitled to some weight, a single case report, by its very nature, cannot rule out chance occurrence.[16] Notably then, despite suggesting a causal

[15] As noted in footnote 14, above, the cardio protective effect of the flu vaccine should be viewed cautiously; however, the fact remains that the findings cited in the Gurevich paper relative to vaccination do not ultimately support petitioner's claim regardless of whether it detected a true cardio protective benefit or merely failed to detect the type of adverse event proposed by petitioner.

[16] "[C]ase reports 'do not purport to establish causation definitively, and this deficiency does indeed reduce their evidentiary value'…. [but] 'the fact that case reports can by their nature only present indicia of causation does not deprive them of all evidentiary weight.'" *See Paluck v. Sec'y of Health & Human*

relationship, the authors of this case report concede that "[t]he connection between viral infection or vaccination and vasculitis or development of coronary sclerosis is not clear." (*Id*. at 6.) Moreover, in conclusion they stress that "the influenza vaccine is considered safe," especially in high risk patients, and that "[t]here is evidence that . . . vaccination reduces the incidence of myocardial ischemia." (*Id*. at 7.) In light of this context provided by the authors, as well as the limitations of case reports generally, I assign very little weight to this article.[17]

Dr. Stark explained that in his view "the presence of continuing inflammation following influenza immunization is a key factor in this case." (Ex. 43, p. 2.) In that regard, he relied on post-marketing information from the high dose "Fluzone" vaccine as well as a clinical trial examining the immunogenicity of high dose vaccines to standard dose vaccines as evidence of "an antigen-antibody mediated inflammatory response" capable of acting on the endothelium of blood vessels. (Ex. 43, pp. 2-3 (citing Exs. 55-56).) Critically, however, petitioner received a "Fluvirin" vaccine (Ex. 21, p. 19), not a high dose "Fluzone" vaccine. Accordingly, Dr. Stark's reliance on data from high dose vaccines is inapposite. Moreover, the clinical trial cited by Dr. Stark concluded that "[t]here was a statistically significant increase in the level of antibody response induced by [high dose] influenza vaccine, compared with that induced by [standard dose] vaccine, without an attendant increase in the rate or severity of clinically relevant adverse reactions." (Ex. 56, p. 1.) Such a conclusion would seem to weigh against any suggestion of a correlation between the antigen-antibody mediated inflammatory response cited by Dr. Stark and the type of cardiovascular adverse reaction alleged by petitioner.

For all these reasons, I find that petitioner has failed to provide preponderant evidence of a medical theory causally connecting her influenza vaccine to ventricular fibrillation or any other acute cardiac event. *Althen*, 418 F.3d at 1278. Petitioner is not required to come forward with either medical literature or epidemiological evidence and any evaluation of such evidence is completed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Andreu*, 569 F.3d at 1380. Nonetheless, I am not persuaded that petitioner's theory is supported by preponderant evidence. That is, neither Dr. Waugh nor Stark, either separately or in combination, have substantiated their opinions with sound and reliable scientific evidence. *Boatmon*, 941 F.3d at 1359. Suspicion of causation does not meet the preponderance standard.[18] *See, e.g.*, *W.C. v. Sec'y of*

Servs., 104 Fed. Cl. 457, 475 (2012) (quoting *Campbell v. Sec'y of Health & Human Servs.*, 97 Fed. Cl. 650, 668 (2011).

[17] An additional issue is presented by this paper in that the original publication was in German. (Ritter et al., *supra*, at Ex. 41.) Petitioner's counsel confirmed that the accompanying translation was produced by him using online tools and is not a professional translation. (ECF No. 88.) Respondent's counsel does not concede the accuracy of the translation; however, the parties opted not to pursue a more formal translation. (ECF No. 91.)

[18] In *Halverson v. Secretary of Health & Human Services*, another special master ruled that a high dose influenza vaccine caused petitioner's cardiac arrest and ultimately her death based in part on Dr. Stark's opinion. No. 15-227V, 2020 WL 992588 (Fed. Cl. Spec. Mstr. Feb. 4, 2020). Notably, that special master

Health & Human Servs., 704 F.3d 1352 (Fed. Cir. 2013). Thus, petitioner has failed to satisfy *Althen* prong one.

b. *Althen* Prong Two

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; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *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 do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. *See*, Section 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, 746 n.67 (2009) ("there 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").

Here, there is no dispute that petitioner had a sudden VF cardiac arrest and was successfully resuscitated. However, none of petitioner's treating physicians attributed her condition to her flu vaccination and in fact, petitioner's cardiologists consistently factored the physical and emotional stress that immediately preceded petitioner's cardiac arrest into their causal assessments. (*See, e.g.*, Ex. 9, pp. 3, 13; Ex. 24, p. 205.) In fact, Dr. Cordas explained that he could not associate petitioner's October 13, 2011 flu vaccination to her October 19, 2011 cardiac arrest, noting that "[n]o symptoms typical of anaphylactic or anaphylactoid reactions related to the vaccine, so I cannot directly link the cardiac arrest to the flu shot." (Ex. 30, p. 2.) Moreover, none of petitioner's treating physicians noted any concerns of inflammation as a factor in

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found that the Lanza and Gurevich articles did support that petitioner's claim. However, the facts and expert presentation in *Halverson* are distinguishable from this case. First, the *Halverson* decedent had significant co-morbidities not present in this case that additionally contributed to her death. Second, the *Halverson* decedent received a high dose influenza vaccination and petitioner's theory in that case was specific to that vaccine. Respondent's expert in that case, Dr. Rose, acknowledged that the high dose vaccine at issue contained four times the amount of a purified protein called hemagglutinin as other influenza vaccines. Since hemagglutinin is known to cause clumping of red blood cells, petitioner's experts opined that the high dose vaccine coupled with the decedent's significant comorbidities, including an upper respiratory infection and pre-existing heart dysfunction, were sufficient to cause a fatal occlusion within her already restricted and stressed arteries.

causing petitioner's cardiac arrest and there is no objective testing to show that petitioner experienced an inflammatory process (as suggested by petitioner's theory) immediately before or after her cardiac arrest.

Petitioner's claim is also hampered by her experts' failure to consistently articulate what effect they believe petitioner's influenza vaccine had on her heart to ultimately lead to petitioner's ventricular fibrillation. In his first report, Dr. Waugh opined that petitioner experienced "an episode of coronary spasm," noting it to have been "possibly in the left anterior descending coronary artery," which resulted in acute coronary syndrome and, but for emergency medical personnel, would have resulted in death from ventricular fibrillation. (Ex. 25, p. 3.) In his second and third reports, however, Dr. Waugh indicated that "there are a number of possible mechanisms for the cardiac event itself," before opining that "[t]he case speaks to a transient heart rate variability . . ." and later indicating that petitioner experienced an arrhythmia brought on by "chemical-electrical instability" in the heart. (Ex. 29, p. 2; Ex. 35, p.1.) In his fourth report, Dr. Waugh explicitly contradicted his earlier report and disclaimed his initial opinion that petitioner had experienced a coronary artery spasm, instead contending that petitioner suffered an "inflammatory electrical malfunction . . . that would not affect coronary arteries." (Ex. 36, p. 1.) Thus, after a reversal, Dr. Waugh ultimately opined before departing the case that petitioner's influenza vaccine caused some unspecified change in petitioner's heart rate by acting on her autonomic nervous system. Dr. Stark, however, though he was consistent in stating his own opinion, brought the discussion back full circle by again implicating petitioner's coronary artery. That is, while he did not invoke any spasm of the artery, Dr. Stark opined that petitioner experienced a ventricular fibrillation and cardiac arrest caused by inflammatory and prothrombotic effect in the coronary artery which led to "small blockages in the coronary microcirculation." (Ex. 39, p. 2.)

However, there is no clear indication from the records that petitioner had any significant inflammation or prothrombotic reaction. As respondent pointed out, "[i]t was not until after the filing of this petition (several years after vaccination) that petitioner reported having a 'flu-like syndrome almost immediately' following vaccination." (ECF No. 93, n.7.) The only available evidence potentially addressing this point are petitioner's prothrombin times (a test measuring clot formation). Upon admission to the hospital after suffering her cardiac event, petitioner's prothrombin time was 13.1 seconds, where the targeted range is 11.6-14.5 seconds. (Ex. 24, p. 190.) Petitioner's time was 14.6 seconds when it was collected the following day, on October 20, 2011 at 4:36am. (*Id*. at 218.) None of the experts or treating physicians attributed any significance to these findings, which appear to be within normal limits, and petitioner has not pointed to any other clinical evidence suggesting petitioner was in a prothrombotic state at that time.

Respondent argues that petitioner's claim is "essentially premised upon a post hoc ergo propter hoc ('after this, therefore because of this') line of reasoning that implicates the flu vaccine as causal based on the mere coincidence that her ventricular event followed vaccination." (ECF No. 93, p. 2.) Respondent points to petitioner's

history of heart palpitations, hypertension, poor r-wave progression, left ventricular hypertrophy, and obstructive sleep apnea, arguing that petitioner was predisposed for a ventricular arrest. Combined with her conditions, the "emotional upset and uncharacteristic physical exertion," are far more likely to have caused petitioner's cardiac event. (*Id*. at 2, 15-16.)

Petitioner argues, however, that the only varying factor present at the time of her cardiac episode was her vaccination. Petitioner indicated that her heart was overall normal despite having multiple risk factors for poor cardiac health, including obesity, hypertension, and diabetes and argued that "there is no evidence whatsoever that these risk factors led to changes in her heart that could be blamed for her event." (ECF No. 92, p. 13.) In his final report, Dr. Stark stressed that petitioner had no coronary atherosclerosis despite her cardiac risk factors. (Ex. 54.) Nonetheless, Dr. Waugh also acknowledged previously that petitioner's cardiac risk factors likely played at least some part in her cardiac event, identifying petitioner's vaccination only as a "tipping point." He opined that "[g]iven her ongoing risk factors, as well as the stress the day of the event, the flu shot was likely the tipping point without which the event would not have occurred." (Ex. 29, p.1.)

In that regard, petitioner's medical records support Dr. LaRue's view that petitioner had underlying left ventricular hypertrophy ("LVH") or an abnormality in the mid-anterior wall of her left ventricle as evidenced by petitioner's ECGs before and after suffering her VF arrest. (Ex. 13, p. 90, 128; Ex. 22, p. 134; Ex. H, p. 5-7.) Further, respondent provided medical literature establishing an association between LVH and ventricular arrythmias. (*See*, *e.g.*, McLenachan, *supra*, at Ex. K; Haider, *supra*, at Ex. L; Thomas Kahan & Lennart Bergfeldt, *Left Ventricular Hypertrophy in Hypertension: Its Arrhythmogenic Potential*, 91 HEART 250 (2005) (Ex. T); Saurav Chatterjee et al., *Meta-Analysis of Left Ventricular Hypertrophy and Sustained Arrhythmias*, 114 AM. J. CARDIOLOGY 1049 (2014) (Ex. U).) While Dr. Stark dismissed LVH as a causal factor because petitioner had documented *mild* LVH, Dr. LaRue contended that based on the literature, severity was not a factor in concluding that the presence of LVH significantly increases one's chances of suffering arrythmias.

Here, notwithstanding petitioner's experts' opinions that petitioner's risk factors alone did not cause petitioner's cardiac episode, the medical records reflect that petitioner did have mild LVH and poor r wave progression. (Ex. 13, p. 127; Ex. 19, p. 41.) And significantly, the majority of the experts in this case, as well as petitioner's treating physicians, considered petitioner's emotional and physical stress on that day as a factor or trigger to petitioner's cardiac arrest. Although Dr. Waugh later noted that "[t]o suggest that [petitioner] had a profound emotional shock as the result of a leaky sink that would lead an otherwise fairly cardio-healthy woman to suffer cardiac arrest is a stretch," he had previously acknowledged, as noted above, that in his opinion the flu vaccine had acted only as a "tipping point" in light of petitioner's "ongoing risk factors, as

well as the stress of the day of the event."[19] (Ex. 29, p 1.) Additionally, the Sharkey article on which Dr. Waugh relied acknowledged that stressful events do trigger cardiac episodes despite the fact that it is not generally understood why a specific stressful event would trigger a cardiac episode in one instance but not another. (Ex. 36, p. 1; Sharkey, Lesser, & Maron, *supra*, at Ex. 38, p. 3.)

In light of all of the above, and in consideration of the record as a whole, including the opinions of Drs. Waugh and Stark, the competing opinions of Drs. Sterling and LaRue, all the medical literature filed, and the opinions and findings of petitioner's treating physicians, I do not find that there is preponderant evidence that the flu vaccine substantially contributed to petitioner's cardiac event on October 19, 2011. Instead, I find respondent's alternative explanation, supported by the contemporaneous medical records, more persuasive in explaining the sequence of events that led to petitioner's cardiac event, unrelated to her flu vaccination.

For these reasons, I find that petitioner failed to provide preponderant evidence of a logical sequence of cause and effect showing that petitioner's flu vaccination caused her cardiac event. Accordingly, petitioner has failed to satisfy *Althen* prong two.

c. *Althen* Prong Three

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *Bazan v. Sec'y of Health & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must also coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Human Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. den'd after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 503 Fed. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Human Servs.*, No. 11–355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

Dr. Waugh opined that flu vaccine can cause inflammation and that the potential for an adverse cardiac event persists for as long as the inflammation is present. (Ex. 29, p. 2.) Respondent has argued that petitioner's cardiac event, having occurred six days after her vaccination, is beyond what could be considered a medically-accepted timeframe for the type of mechanism petitioner proposes based on the available evidence regarding how long the markers of that inflammation persist. (ECF No. 41, p. 10 (arguing Dr. Waugh has no basis to extend the 48-hour study period from the Lanza article to six days).) However, Dr. LaRue has cited literature showing that, although the

[19] It is also worth noting that, contrary to Dr. Waugh's characterization of the day in question, petitioner reported to her physicians not merely a leaky sink, but also an "emotional argument" with her husband just prior to her cardiac event. (Ex. 19, p. 57.)

inflammation cited by petitioner may peak earlier, it remains measurable for at least five days after vaccination. (Ex. S, p. 2 (citing Posthouwer et al., *supra*, at Ex. R.) That same article notes that prior studies have measured the highest increases in the first week post-vaccination, but also that non-significant elevations have been measured up to 28 days post-vaccination. (Ex. R, p. 364.)

Additionally, the previously-assigned special master filed two studies as Court Exhibits I and II that showed that CRP levels were elevated in the days following vaccination, but returned to baseline at seven days post vaccination. Specifically, the Tsai study measured CRP levels at one, three, and seven days post-vaccination. (Tsai et al., *supra*, at Ex. II.) According to the authors, the CRP levels were elevated on day one, further elevated on day three, then back to baseline upon measurement at day seven. (*Id.* at 326.) The authors characterized day three as the "peak;" however, since measurements were not taken on days four through six, an actual peak was not measured. The Christian study measured CRP at one, two, and seven days post-vaccination. (Christian et al, *supra*, at Ex. I.) That study similarly found a significant increase on day one, followed by a further increase on day two, and then a return to baseline upon measurement on day seven. (*Id*. at 4-5.)

Although this literature does not pinpoint when inflammation completely diminishes post-vaccination, the articles collectively support the idea that inflammation continues to rise for at least three days post-vaccination, may remain present for at least five days after vaccination, and is not confirmed absent, or back to baseline, until day seven. Therefore, by establishing that her cardiac event occurred six days post-vaccination, petitioner has provided preponderant evidence establishing a temporal relationship between the flu vaccine and her cardiac arrest, especially in light of the Federal Circuit holding in *Paluck v. Secretary of Health & Human Services,* which cautions against setting "hard and fast deadline[s]" for onset. *See*, 786 F.3d 1373, 1383-84 (Fed. Cir. 2015) (stating that "[t]he special master further erred in setting a hard and fast deadline" for onset and noting that the medical literature filed in the case "do not purport to establish any definitive timeframe for onset of clinical symptoms.").

However, even though petitioner met *Althen* prong three based on the six-day onset period, a temporal relationship alone cannot establish causation.[20] *Veryzer v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 344, 356 (2011) (explaining that a "temporal relationship alone will not demonstrate the requisite causal link and that petitioner must posit a medical theory causally connecting the vaccine and injury."). Thus, petitioner's failure to meet prongs one and two means that petitioner cannot be compensated. *Hibbard v. Sec'y of Health & Human Servs.*, 698 F.3d 1355, 1364-65

[20] Also significant to this point is the fact that petitioner's emotional stress on the day of her hospitalization can also be temporally related to her cardiac event. Dr. Sperling emphasized a point made in the Sharkey paper submitted by Dr. Waugh that stress-related cardiomyopathy occurs within minutes to hours of a stressful event in 85% of cases. (Ex. E, p. 3 (citing Sharkey, Lesser & Maron, *supra*, at Ex. 38).) For all the reasons discussed above, preponderant evidence that the timing of petitioner's cardiac event is consistent with petitioner's theory does not necessarily suggest preponderant evidence that the theory is the best explanation of the course of events.

(Fed. Cir. 2012) (holding the special master did not err in resolving the case pursuant to Prong Two when respondent conceded that petitioner met Prong Three).

V. Conclusion

For all the reasons discussed above, after weighing the evidence of record within the context of this Program, I do not find by preponderant evidence that petitioner's injury was caused by her October 13, 2011 influenza vaccination as alleged. Therefore, this case is dismissed.[21]

IT IS SO ORDERED.

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

[21] In the absence of a timely-filed motion for review of this Decision, the Clerk of the Court shall enter judgment accordingly.