In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: September 2, 2025

VANESSA K. DRAKE and LANCE DRAKE, as parents and legal representatives of a minor child, T.B.D.,	*	
Petitioners,	*	No. 17-1836V
v.	*	Special Master Nora Beth Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Entitlement; Table Injury; Diphtheria-Tetanus-Acellular Pertussis ("DTaP") Vaccine; Cardiac Arrest; Encephalopathy.
Respondent.	*	

Armond Marcarian, Marcarian Law Firm, P.C., Woodland Hills, CA, for Petitioners.
Alyssa M. Petroff, U.S. Department of Justice, Washington, DC, for Respondent.

RULING ON ENTITLEMENT[1]

I. INTRODUCTION

On November 27, 2017, Vanessa K. Drake and Lance Drake, as parents and legal representatives of minor child T.B.D., ("Petitioners") filed a petition under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"), 42 U.S.C. § 300aa-10 et seq.

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

AUTHENTICATED U.S. GOVERNMENT INFORMATION GPO

(2018).[2] Petitioners allege T.B.D. suffered "injuries including ventricular tachycardia, ventricular fibrillation, hypoxic ischemic brain damage, and cardiomyopathy" caused-in-fact by the diphtheria-tetanus-acellular pertussis ("DTaP"), inactivated poliovirus ("IPV"), *Haemophilus influenzae* type B ("Hib"), Rotavirus, pneumococcal conjugate ("Prevnar 13"), and hepatitis B ("Hep B") vaccinations administered to T.B.D. on December 15, 2015. Petition at Preamble (ECF No. 1). On January 5, 2021, Petitioners filed an amended petition adding acute hypersensitivity myocarditis and a Table injury of acute encephalopathy to the claimed injuries.[3] Amended ("Am.") Petition at Preamble (ECF No. 38). Respondent argued against compensation, stating that "this case is not appropriate for compensation under the terms of the [Vaccine Act]." Respondent's Report ("Resp. Rept.") at 2 (ECF No. 11).

After carefully analyzing and weighing the evidence presented in this case in accordance with the applicable legal standards,[4] the undersigned finds that Petitioners have proved their Vaccine Injury Table claim by preponderant evidence. The undersigned also finds that in the alternative, Petitioners have satisfied their burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005) for their causation-in-fact claim. Accordingly, Petitioners are entitled to compensation.

II. ISSUES IN DISPUTE

The parties first dispute the nature of T.B.D.'s injury, including the diagnosis of his injury. Joint Submission, filed Dec. 13, 2022, at 1 (ECF No. 65).

The parties also dispute (1) whether the vaccines administered to T.B.D. on December 15, 2015 caused T.B.D.'s alleged injuries; (2) whether T.B.D. suffered injuries listed in the Vaccine Injury Table; and (3) whether T.B.D.'s reported injuries satisfy the Vaccine Injury Table requirements. Joint Submission at 2. As set forth by Respondent, "[t]o avail themselves of the

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[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to -34 (2018) ("Vaccine Act" or "the Act"). All citations in this Ruling to individual sections of the Vaccine Act are to 42 U.S.C.A. § 300aa.

[3] "The amended petition alleges T.B.D. suffered Table encephalopathy or, alternatively, T.B.D.'s encephalopathy and cardiac arrest were caused in fact by DTaP alone, or in combination with the other vaccines he received on December 15, 2015." Petitioner's Brief Re: Ruling on the Record ("Pet. Br."), filed Feb. 16, 2023, at 1 (ECF No. 74); see also Am. Petition at ¶¶ 31-33.

[4] Although this Ruling discusses some but not all of the medical literature in detail, the undersigned reviewed and considered all of the medical records and all of the medical literature submitted in this matter. See Moriarty v. Sec'y of Health & Hum. Servs., 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though [s]he does not explicitly reference such evidence in h[er] decision."); Simanski v. Sec'y of Health & Hum. Servs., 115 Fed. Cl. 407, 436 (2014) ("[A] Special Master is 'not required to discuss every piece of evidence or testimony in her decision.'"), aff'd, 601 F. App'x 982 (Fed. Cir. 2015).

presumption of causation awarded under a Table claim, [P]etitioners must demonstrate that T.B.D.'s condition met the legal Table definition of an encephalopathy." Resp. Response to Pet. Br. ("Resp. Response"), filed Mar. 27, 2023, at 15 (ECF No. 76) (citing 42 C.F.R § 100.3(c)(2)(i)).

In the alternative to their Table claim, Petitioners also assert a causation-in-fact claim alleging T.B.D.'s injuries were caused-in-fact by the vaccinations he received on December 15, 2015. Pet. Br. at 23. The parties disagree about whether Petitioners have provided preponderant evidence of causation for all three Althen prongs. See Joint Submission at 2; Pet. Br. at 24-29; Resp. Response at 25-28, 31-38; Althen, 418 F.3d at 1280.

III. BACKGROUND

A. Procedural History

Petitioners filed their petition and medical records[5] on November 27, 2017. Petition; Pet. Exhibits ("Exs.") 1-20. Respondent filed his Rule 4(c) report arguing against compensation on May 2, 2018. Resp. Rept. at 2.

On October 3, 2018, Petitioner filed an expert report from Dr. Anthony Chang. Pet. Ex. 23. Respondent filed an expert report from Dr. Richard E. Ringel on March 29, 2019. Resp. Ex. A. On October 7, 2019, the case was reassigned to the undersigned. Notice of Reassignment dated Oct. 7, 2019 (ECF No. 22). This matter was initially set for an entitlement hearing on December 2, 2020, but it was postponed so Petitioners could file an amended petition alleging a Table encephalopathy injury. Order dated Jan. 23, 2020 (ECF No. 33); Order dated Oct. 8, 2020 (ECF No. 34).

Petitioners filed a supplemental expert report from Dr. Chang on December 30, 2020. Pet. Ex. 27. On January 5, 2021, Petitioners filed an amended petition. Am. Petition. On April 26, 2021, Respondent filed a supplemental expert report from Dr. Ringel and an expert report from Dr. Peter M. Bingham. Resp. Exs. F-G. On September 21, 2021, Petitioners filed an expert report from Dr. Perry Lubens. Pet. Ex. 30. Respondent filed a supplemental expert report from Dr. Bingham on October 1, 2021. Resp. Ex. I.

The entitlement hearing was rescheduled for April 18, 2023. Prehearing Order dated Dec. 6, 2021 (ECF No. 61). On September 29, 2022, the parties indicated they wish to proceed with a ruling on the record in lieu of an entitlement hearing to resolve this case. Ruling on the Record Order dated Sept. 30, 2022, at 1 (ECF No. 62). The entitlement hearing was cancelled, and a briefing schedule was set. Id.

On February 16, 2023, Petitioners filed their motion for a ruling on the record. Pet. Br. Respondent filed a response on March 27, 2023, and Petitioners filed a reply on May 26, 2023. Resp. Response; Pet. Reply Re: Ruling on the Record ("Pet. Reply"), filed May 26, 2023 (ECF No. 79).

[5] Petitioner continued to file medical records throughout the course of litigation.

On February 6, 2024, the undersigned held a status conference to address the additional information needed for her to reach a decision regarding entitlement. Order dated Feb. 6, 2024, at 1 (ECF No. 81). The undersigned recommended convening a one-day hearing where the parties' neurologist experts testify to the injuries T.B.D. suffered by virtue of his cardiac arrest as compared to that caused by the periventricular white matter abnormality. Id. at 2. A hearing was set for September 26, 2024. Order dated April 24, 2024 (ECF No. 87).

On September 17, 2024, the September 26 hearing was cancelled and the parties agreed to submit additional expert reports from their neurologist experts in lieu of a hearing. Order dated Sept. 17, 2024, at 2 (ECF No. 89). On October 21, 2024, Petitioners filed a supplemental expert report from Dr. Lubens. Pet. Ex. 39. On November 20, 2024, Respondent filed a supplemental expert report from Dr. Bingham. Resp. Ex. Q. Petitioner did not file a responsive expert report.

This matter is now ripe for adjudication.

B. Summary of Medical Records

1. Pre-Vaccination History

T.B.D. was born premature, at thirty-six weeks on October 22, 2015. Pet. Ex. 3 at 10; Pet. Ex. 4 at 5-6. He weighed six-pounds and six-ounces. Id. at 5. T.B.D.'s antenatal genetic testing was negative. Pet. Ex. 1 at 10-19, 29-40. A newborn metabolic screen was normal. Pet. Ex. 4 at 16. T.B.D. received his first Hep B vaccine on October 23, 2015. Id. at 22. He was discharged from the hospital in good health. Id. at 8-13.

On November 5, 2015, T.B.D. had a two-week well-child visit with his pediatrician, Dr. Jennifer Hartstein. Pet. Ex. 5 at 38. He presented with a "bump on right occipital." Id. at 39. Dr. Hartstein noted it was a "soft cystic mass that fe[lt] almost like two cysts." Id. Everything else was normal. Id. at 39-40. The plan was to "[m]onitor cystic mass – possible two lymph nodes or dermoid." Id. at 40.

T.B.D. was seen for "thrush" on November 16, 2015. Pet. Ex. 5 at 35. History of present illness documented that Petitioners had "noticed increasing fussiness over past few days" that seemed primarily at night and questioned whether it was associated with increased spit up and drooling. Id. The rest of the examination was normal. Id. at 35-36. The assessment was fussy infant. Id. at 37.

On December 15, 2015, T.B.D. returned to Dr. Hartstein for his two-month check-up. Pet. Ex. 5 at 31. His physical examination, including the cardiovascular assessment,[6] was

[6] T.B.D.'s cardiovascular examination showed a regular heart rate and rhythm, no murmurs, and 2+ femoral pulses bilaterally. Pet. Ex. 5 at 32. T.B.D.'s previous visits up to this date also showed the same normal cardiovascular findings. Id. at 46 (October 26, 2015), 42 (October 30, 2015), 40 (November 5, 2015), 36 (November 16, 2015).

normal. Id. at 32. Developmental delay was noted but "suspected normal and temporary related to prematurity 36 weeks." Id. at 33. T.B.D. received his first Pentacel (DTaP-Hib-IPV), second Hep B, first Prevnar 13, and first Rotavirus vaccines at 3:13 p.m.-3:14 p.m. Id. at 32-33.

2. Post-Vaccination Event

Approximately 23 hours later, on December 16, 2015, the Los Angeles Fire Department received a call at 1:56 p.m., and arrived at T.B.D.'s residence at 2:08 p.m., for "possible [apparent life threatening event ("ALTE")]" with a chief complaint of "choking." Pet. Ex. 6 at 1. The narrative history from the Los Angeles Fire Department indicated:

> [o]riginal call came as a possible ALTE, [T.B.D.] had choked on milk and the mother was unable to clear [T.B.D.'s] airways . . . . [T.B.D.] had agonal resp[irations] and slow pulse on scene. When [T.B.D.] was re evaluated in back of rescue, [T.B.D.] had lost pulse and resp[irations]. CPR was started, chest compressions and [bag-valve-mask] while enroute to hospital. Had to suction airway several times while enroute. [T.B.D.] was found to be in fine v[entricular] fib[rillation] at this time.

Id. at 3.

Upon arrival to Glendale Adventist Medical Center on December 16, T.B.D. was "unresponsive," had "[n]o cardiac activity," and had "[v]omitus in the airway." Pet. Ex. 7 at 11-12. T.B.D. was evaluated by Dr. Evelyn Wong for "cardio-respiratory arrest" with onset "just prior to arrival." Id. at 11. Associated diagnoses were "[c]ardiopulmonary arrest; [m]etabolic acidosis; [h]ypothermia." Id. Per T.B.D.'s mother, she was breast feeding him when he "cried out and went pale at [1:55 p.m.]" Id. "Witnessed arrest" was documented. Id. Dr. Wong noted T.B.D. "had his first set of immunizations yesterday and was doing fine. Mom was . . . breast-feeding and [T.B.D.] suddenly grabbed the mom's hair and became unresponsive; [T.B.D.] did not vomit." Id. Compressions and mouth to mouth were attempted during which T.B.D. "cried initially but became unresponsive again. [Emergency medical services ("EMS")] was called and found [T.B.D.] in full arrest." Id. No preceding symptoms were noted. Id. Risk factors included premature birth. Id. Differential diagnoses included "[c]ardiac arrest, cardiac dysrhythmia, seizure disorder, hypoglycemia, intracranial hemorrhage, [and] [a]spiration." Id. at 12. T.B.D. was stabilized, intubated, and placed on a respirator with sedative medication at 4:04 p.m. Id. at 12-14.

3. Hospitalization

Later that afternoon, T.B.D. was transferred to Children's Hospital Los Angeles ("CHLA") where he remained until January 19, 2016. Pet. Ex. 7 at 14; Pet. Ex. 8 at 5, 558. On admission, he remained "intubated without any pressors." Pet. Ex. 8 at 10. Physical examination noted T.B.D. was "[i]ntubated with minimal spontaneous movements" and

"pinpoint pupils" that were "minimally responsive." Id. at 8. He was started on amiodarone[7] for persistent episodes of supraventricular tachycardia ("SVT")[8] and intermittent ventricular tachycardia ("VT").[9] Id. at 10, 237. There were concerns for heart failure, however, it was "reassuring that prior to [the] episode[,] [T.B.D.] [was] asymptomatic and growing appropriately with no appreciable murmurs." Id. at 10, 239. T.B.D. was extubated on December 18, 2015. Id. at 43.

Given concern for heart failure, an echocardiogram was performed as well as laboratory tests for cardiac markers, B-Type Natriuretic Peptide ("BNP")[10] and troponins.[11] Pet. Ex. 8 at 10. It was anticipated the cardiac markers would be "mildly elevated given cardiac arrest with chest compressions." Id. The cardiac arrest was suspected to be "likely related to an arrythmia." Id. at 238, 242. Other less likely causes of the cardiac arrest included "primary pulm[onary] disease[,] . . . seizures, [and] aspiration." Id. at 10. During his hospitalization, T.B.D. was primarily followed by cardiology and neurology. See generally Pet. Ex. 8.

i. Cardiology Assessments

Pediatric cardiologist Dr. Michael Silka evaluated T.B.D. on December 17, 2015. Pet. Ex. 8 at 83. T.B.D. remained intubated and sedated. Id. An echocardiogram was "notable for

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[7] Amiodarone hydrochloride is "a potassium channel blocking agent" that is "administered orally or by intravenous infusion in the treatment and prophylaxis of ventricular arrhythmias." Amiodarone Hydrochloride, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=2253 (last visited Aug. 13, 2025).

[8] Supraventricular tachycardia is "any regular tachycardia in which the point of stimulation is located above the bundle branches, either in the sinus node, atria, or atrioventricular junction; it may also include those arising from large reentrant circuits encompassing both atrial and ventricular sites." Supraventricular Tachycardia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=112013 (last visited Aug. 13, 2025).

[9] Ventricular tachycardia is "an abnormally rapid ventricular rhythm with aberrant ventricular excitation (wide QRS complexes), usually in excess of 150 per minute." Ventricular Tachycardia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=112015 (last visited Aug. 25, 2025).

[10] BNP or brain natriuretic peptide is "a hormone . . . stored mainly in the myocardium of the cardiac ventricles. Blood levels of BNP are elevated in hypervolemic states such as congestive heart failure and hypertension." Brain Natriuretic Peptide, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=97135 (last visited Sept. 2, 2025).

[11] Troponins "are proteins that exist in skeletal and cardiac muscles that regulate the calcium-dependent interaction of myosin with actin for muscle contractions." Troponins, Mosby's Manual of Diagnostic and Laboratory Tests 451 (6th ed. 2018). Troponins are "biomedical marker for cardiac disease" with elevated troponins associated with "acute coronary syndromes" including myocarditis. Id. at 452-53.

relatively normal function and mild-moderate concentric [left ventricular hypertrophy]."[12] Id. at 83-84. On physical examination two days later, T.B.D. exhibited "normal baby movements." Id. at 92.

Cardiac magnetic resonance imaging ("MRI") with and without contrast and MRI Velocity Imaging done December 21, 2015 showed "moderate asymmetric hypertrophy of the interventricular septum"[13] with "normal myocardial perfusion . . . . With a baby this small it is impossible to exclude myocarditis." Pet. Ex. 8 at 639-40. T2 imaging "was poor quality and nondiagnostic." Id. at 639.

Complete blood count ("CBC") with differential drawn December 16, 2015 revealed elevated neutrophils (55.4%; reference range of 25.0-45.0), decreased lymphocytes (25.4%; reference range of 46.0-66.0), and elevated monocytes (16.9%; reference range of 2.0-8.0). Pet. Ex. 8 at 615. CBC showed normal eosinophils (1.4%; reference range of 0.0-3.0) on December 16, 2015; however, eosinophils were elevated (5.2%; reference range of 0.0-3.0) on December 26. Id.

Serial testing of BNP and Troponin I showed the following elevated results which were monitored until they normalized during T.B.D.'s hospitalization:

Troponin, I	12/16/2015	9.99	[Reference Range is 0.00-0.05 ng/mL]
	12/17/2015	12.20	
	12/18/2015	6.99	
	12/19/2015	3.80	
	12/20/2015	2.96	
	12/22/2015	0.53	
	12/23/2015	0.30	
	12/24/2015	0.11	
	12/26/2015	<0.05	
BNP	12/16/2015	258	[Reference Range is 0-100 pg/mL]
	12/18/2015	774	
	12/19/2015	1430	
	12/20/2015	1220	
	12/21/2015	1090	

[12] Left ventricular hypertrophy is a "hypertrophy of the myocardium of a ventricle of the heart, due to chronic pressure overload; it is manifest electrocardiographically by increased QRS complex voltage." Ventricular Hypertrophy, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=81659 (last visited Aug. 25, 2025).

[13] Interventricular septum is "the wall that separates the left ventricle from the right ventricle, consisting of a muscular and a membranous part." Interventricular Septum of Heart, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=105608 (last visited Aug. 25, 2025).

12/22/2015	937
12/23/2015	998
12/24/2015	358
12/26/2015	309
12/28/2015	208
01/04/2016	91

Pet. Ex. 6 at 600-07.

Blood for additional testing including a myocarditis panel,[14] comprehensive cardiomyopathy panel,[15] among other tests, was collected on December 21, 2015. Pet. Ex. 8 at 618- 620. Daily progress noted on December 29, 2015 documented that the myocarditis panel was negative, however the actual results do not appear in the medical records. See Pet. Ex. 8 at 54.

During T.B.D.'s hospitalization, his heart rhythm was continuously monitored and interpretations of his rhythm were periodically documented. Electrocardiogram ("EKG") abnormalities included SVT, ventricular hypertrophy, non-sustained VT, borderline prolonged QT interval, tachycardia, ventricular premature complexes, prolonged QT interval, and others. Pet. Ex. 8 at 347-49, 351-54, 362, 393, 421, 497. Other abnormalities included atrial fibrillation, pared ventricular premature complexes, right axis deviation, and consider RVH. Id. at 421. EKG also documented multiform premature ventricular contractions ("PVCs"). Id. at 437. On January 16, 2016, Dr. Silka noted "RBBB [right bundle branch block] morphology with no consistent P wave preceding." Id. at 216.

T.B.D.'s acute diagnosis was cardiac arrest from an arrhythmia. Pet. Ex. 8 at 92, 238. The cause of the arrythmia and cardiac arrest were "unclear." Id. at 242. On December 18, 2015, Dr. Grace Kung noted she was considering bloodwork for cardiomyopathy/myocarditis. Id. at 89. On December 20, 2015, pediatric cardiologist Dr. Sarah Badran noted T.B.D. had "significant improvement without anti-inflammatory treatment, will not give [intravenous immunoglobulin ("IVIG")]/steroids at this time." Id. at 96.

On December 22, 2015, Dr. Silka noted the MRI and echocardiogram together demonstrated "mild septal hypertrophy [consistent with] diagnosis of hypertrophic

[14] Although the actual study results are not in the medical records this procedure appears to test for viral causes of myocarditis. This understanding is supported by the records of geneticist Dr. Derek Wong who referenced the study as a "viral myocarditis panel," noting it was negative. See Pet. Ex. 10 at 87. It is also supported by the expert report from Dr. Ringel, and his opinion that the "viral panel" done about two weeks after T.B.D.'s arrest was negative. Resp. Ex. A at 4.

[15] Although the actual study results are not in the medical records, and so it is not entirely clear, this procedure appears to test for genes associated with cardiomyopathy. See, e.g., Pet. Ex. 8 at 114.

cardiomyopathy."[16] Pet. Ex. 8 at 99. Consultation for genetic testing was recommended for hypertrophic obstructive cardiomyopathy.[17] Id.

On December 23, 2015, Dr. Badran examined T.B.D. Pet. Ex. 8 at 109-10. She also noted the cardiac MRI showed "mild septal hypertrophy [consistent with] diagnosis of hypertrophic cardiomyopathy." Id. at 109. The next day, Dr. Badran documented "[p]resumptive diagnosis at this time with septal hypertrophy and [ventricular fibrillation] is hypertrophic cardiomyopathy." Id. at 114.

Serial echocardiograms were performed during hospitalization. Pet. Ex. 8 at 327-46. The first echocardiogram performed on December 16 showed "[m]ild concentric left ventricular hypertrophy" with "mild septal hypokinesis."[18] Id. at 328-30. On December 29, echocardiogram showed "[b]orderline decreased [left ventricular] systolic function, mild septal hypokinesis" and "abnormal left ventricular diastolic function." Id. at 339. A repeat echocardiogram on January 8, 2016 revealed "mild concentric ventricular hypertrophy" with "mild septal hypertrophy." Id. at 342.

ii. Neurology Assessments

On December 21, 2015, T.B.D.'s brain MRI revealed "restricted diffusion along the posterior aspects of the insular cortex[19] and posterior temporal lobe[20] on right," which was

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[16] Hypertrophic cardiomyopathy is "a condition, often of autosomal dominant inheritance, marked by ventricular hypertrophy, particularly of the left ventricle and often involving the interventricular septum, with diastolic dysfunction manifest as impaired ventricular filling; it is sometimes the cause of sudden cardiac death." Hypertrophic Cardiomyopathy, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=63272 (last visited Aug. 25, 2025).

[17] Hypertrophic obstructive cardiomyopathy is "a form of hypertrophic cardiomyopathy in which the location of the septal hypertrophy causes obstructive interference with left ventricular outflow." Hypertrophic Obstructive Cardiomyopathy, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=63273 (last visited Aug. 25, 2025).

[18] The echocardiograms also showed other findings, but these are not discussed as they are not relevant to the question of causation.

[19] Insular lobe, or insula, is "a portion of the cerebral cortex lying deep in the lateral sulcus, almost surrounded by the circular sulcus." Insula, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=25449 (last visited Aug. 26, 2025).

[20] Temporal lobe is "the lower lateral lobe of the cerebral hemisphere, lying below the posterior ramus of the lateral sulcus, lateral to the collateral sulcus, and merging behind with the occipital lobe on the lateral and inferior surfaces." Lobus Temporalis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=87306 (last visited Aug. 26, 2025).

"suggestive of infarction/hypoperfusion injury." Pet. Ex. 8 at 134, 637. Periventricular white matter changes were also observed, "suggestive of prior white matter injury." Id. at 134-35, 637.

T.B.D. was examined by the CHLA inpatient service neurology residents on December 22, 2015. Pet. Ex. 8 at 287. During the consultation, T.B.D. was "fussy but consolable." Id. at 285. Petitioners reported that T.B.D. started tracking two weeks ago and that he kept "hands fisted" bilaterally. Id. at 284. Neurologic examination was "significant for diffuse hypertonia with cortical fisting[21] and [bilateral lower extremity] hyper-reflexia." Id. at 286. "[T.B.D.] [was] able to drink from a bottle without pooling, choking, or coughing. He move[d] all extremities. He had increased tone in the appendicular muscles. He had persistent fisting (which his parents state[d] [was] present even before the [cardiac] arrest)." Id. at 287. Lastly, examination revealed T.B.D. had "clonus[22] on the right ankle [five to six] beats, extinguishable. On the left, his left [lower extremity] [was] swollen and discolored because of a clot." Id.

On December 22, Dr. Leigh Ramos-Platt (attending neurologist) wrote the "MRI of the brain demonstrate[d] abnormalities in the temporal lobe and insula. There were also changes seen in the periventricular white matter that seem to be of a different age." Pet. Ex. 8 at 287. The assessment was that T.B.D. "most likely had two separate insults to his brain resulting in his MRI findings." Id. at 286. The restricted diffusion was most likely acute/subacute and likely had occurred within 14 days. Id. "This is most likely a result of the cardiac arrest he suffered on admission." Id. at 286-87 ("[T]he the changes in the MRI were consistent with the event."). The periventricular white matter changes were likely more remote and occurred during the perinatal period. Id. at 286. "These findings could be due to his premature status, or due to periods of hypoxia secondary to his underlying cardiac condition." Id. Additionally, the periventricular white matter involvement was "likely responsible for [T.B.D.'s] increased tone." Id. "This increased tone was evident even prior to the arrest as his parents noted the persistent fisting prior to the arrest." Id. at 288. Dr. Ramos-Platt noted that periventricular white mater "changes can be seen in preemie[,] . . . [but] can also be seen with previous hypoperfusion that did not result in an arrest." Id. at 287-88.

Petitioners were informed that the "lesions in the temporal lobe/insular cortex can be a nidus for seizures/epilepsy." Pet. Ex. 8 at 286, 288. "Unfortunately, these are the same findings with arrhythmia. Neurology will have to work closely with cardiology regarding distinguishing which process is causing these symptoms in the future." Id. at 288. It was noted that cardiology was currently working up T.B.D.'s arrhythmia and that a preliminary read of T.B.D.'s cardiac MRI demonstrated "hypertrophic cardiomyopathy." Id. at 283. On December 22, Dr. Ramos-

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[21] "Adduction and flexion posturing of the thumb (also referred to as 'cortical thumb' sign) and fisting are normal findings in term newborns, but their persistence beyond [four] months of age, or the presence of a tightly fisted hand that does not open spontaneously, may be a sign of upper motor neuron injury." Frederico Poropat et al., Cortical Thumb Sign, 163 J. Pediatrics 605 (2013).

[22] Clonus is "a continuous rhythmic reflex tremor . . . set in motion by reflex testing." Clonus, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition ?id=10153 (last visited Aug. 22, 2025).

Platt concluded that given the MRI findings, she did "not feel that a seizure caused the cardiac arrhythmias. [T.B.D.] probably did have a seizure[] as a result of low perfusion to the brain." Id. at 288. It was noted that T.B.D. was already on anticoagulation for his deep vein thrombosis. Id.

On December 23, 2015, a video electroencephalogram ("EEG") demonstrated "no electrographic or electroclinical seizures," but "epileptiform discharges" were seen in the right temporal head region which were abnormal. Pet. Ex. 8 at 134-35.

A follow-up neurology consultation on December 28 was "significant for increase[d] [bilateral lower extremity] tone as well as primitive cross-adductor reflexes." Pet. Ex. 8 at 135-36. Attending neurologist Dr. Eugenia Ho discussed with Petitioners that the changes observed on T.B.D.'s EEG "can be seen in the acute phase of injury but if [T.B.D.] does not have clinical seizures, he does not require anticonvulsants." Id. at 136. The plan was physical therapy and occupational therapy, to follow up with cardiomyopathy workup, and to follow up with outpatient neurologist Dr. Brian Wong. Id. at 135.

iii. Genetic Assessments

A genetics consultation was conducted on December 23, 2015 "for a question of genetic syndromes/etiologies associated with hypertrophic cardiomyopathy." Pet. Ex. 8 at 271-72. Family history indicated T.B.D.'s paternal grandfather has an "enlarged heart" but that he has not had any heart complications. Id. at 273. Dr. Catherine Karimov wrote that hypertrophic cardiomyopathy was diagnosed in T.B.D. "based on measurements of the cardiac wall on echocardiogram." Id. at 282. Dr. Karimov assessed the different causes of cardiomyopathy from a genetics point of view, noting that "family history may suggest a familial hypertrophic cardiomyopathy." Id. at 281-82. She explained the familial type is "a disease in which the heart muscle becomes abnormally thick or 'hypertrophied' without any discernable cause. The condition maybe passed down through families and is believed to be a result of a defect or defects in genes that control heart muscle growth and function." Id. at 282. She discussed with Petitioners "the importance of obtaining a metabolic cause of hypertrophic cardiomyopathy and then if negative[,] [to] continue to establish the possible familial hypertrophic cardiomyopathy." Id. Additionally, it was requested that T.B.D.'s parents have echocardiograms and to obtain the echocardiograms previously performed on T.B.D.'s paternal grandfather. Id. at 273, 282.

iv. Discharge Notes

T.B.D. was discharged on January 19, 2016. Pet. Ex. 8 at 558. Echocardiogram done the prior day showed mild left ventricular hypertrophy with normal systolic function. Id. at 344-46, 559. Dr. Matthew Smith summarized his hospital course. Id. at 558. T.B.D. "presented . . . with ventricular fibrillation and cardiac arrest at home and was found to have ectopic atrial tachycardia and hypertrophic cardiomyopathy . . . . He was neurologically appropriate after resuscitation." Id. Brain MRI demonstrated hypoperfusion injury to parts of the temporal lobe. Id. T.B.D. was "found to have ectopic atrial tachycardia [] and rhythm stabilized on amiodarone infusion." Id. T.B.D. transitioned to propranolol but was brought back to the intensive care unit ("ICU") on December 26, 2016 and restarted on amiodarone. Id. at 246, 558. During his

hospital course, T.B.D. developed right lower extremity venous thrombosis associated with a peripherally inserted central catheter ("PICC") line[23] that was then removed, and he was started on Lovenox. Id. at 100, 120, 267, 558. At the time of discharge, he had been in normal sinus rhythm for about seven days with the last arrythmia on January 13, 2016. Id. at 558. "While on amiodarone he had a mild elevation of [thyroid-stimulating hormone]" which was to be followed on an outpatient basis. Id. Hypertrophic cardiomyopathy panel was "pending at time of discharge to hopefully determine the etiology of his condition." Id. Discharge diagnosis was hypertrophic cardiomyopathy. Id. Discharge medications included amiodarone, enoxaparin, and propranolol. Id. at 569.

4. Post-Hospitalization Care

On January 25, 2016, T.B.D. was evaluated by Dr. Silka. Pet. Ex. 10 at 140. Dr. Silka documented that since T.B.D.'s discharge, a screening test for mucopolysaccharidosis ("MPS")[24] came back positive and Petitioners were awaiting an appointment with genetics specialist Dr. Derek Wong for further evaluation. Id. Cardiac genetic screening for possible cardiomyopathy also remained pending. Id. at 141.

Dr. Silka noted that T.B.D.'s "cardiac arrest occurred [within] 24 h[ours] of his first [DTaP] ([two] month) immunization—although any cause/effect relationship would be speculative." Pet. Ex. 10 at 141. Diagnosis remained hypertrophic cardiomyopathy and "[h]istory of sudden cardiac arrest successfully resuscitated." Id. at 143. Dr. Silka noted T.B.D. appeared stable as to his cardiac status and that his overall course was "[i]mproving." Id.

T.B.D. presented to Dr. Hartstein on January 27, 2016 for follow-up from cardiac arrest. Pet. Ex. 5 at 28. Dr. Hartstein wrote T.B.D. was

> [d]oing well, [he] went to Dr. Silka on Monday—arrhythmia not explained by SVT alone—would be only seen in conjunction with cardiomyopathy. Minor hypertrophic cardiomyopathy—may be ignored if he was a healthy and normal

[23] A PICC line is a "a long catheter introduced through a vein in the arm, then through the subclavian vein into the superior vena cava or right atrium to administer parenteral fluids . . . or medications or to measure central venous pressure." Peripherally Inserted Central Catheter, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=63721 (last visited Aug. 18, 2025).

[24] Mucopolysaccharidoses are an "inherited deficiencies of enzymes involved in glycosaminoglycan breakdown" with common clinical manifestations of MPS including "neurodevelopmental delays and regression, joint contractures, organomegaly, stiff hair, progressive respiratory insufficiency . . . , cardiac valvular disease, skeletal changes, and cervical vertebral subluxation." Matt Demczko, Overview of Lysosomal Storage Disorders, Merck Manual, https://www.merckmanuals.com/professional/pediatrics/inherited-disorders-of-metabolism/overview-of-lysosomal-storage-disorders?query=mucopolysaccharidoses (last visited Aug. 20, 2025).

> [infant]. +polysaccharides in urine—awaiting genetic evaluation for MPS—will see [genetics specialist] Dr. Derek Wong . . . in early February.

Id.

Petitioners were with T.B.D. at all times "based on Dr. Silka's prediction that he has a 30% risk of another cardiac arrest." Pet. Ex. 5 at 29. Dr. Hartstein had an "[e]xtensive discussion" with Petitioners about vaccinations. Id. at 30. She documented that T.B.D.'s "cardiac arrest occurred one day after he received his [two] month vaccinations." Dr. Hartstein wrote that "Dr. Silka says ok to do [vaccines] with Tylenol for 24 h[ours] before and after." Id. Dr. Hartstein explained to Petitioners that "no one can reassure that the vaccinations won't decompensate [T.B.D.'s] heart rhythm" and recommended they hold off on vaccinations at the four-month visit pending genetic results. Id.

On February 1, 2016, T.B.D. was seen by pediatric physical therapist Nancy Egizil for a developmental evaluation. Pet. Ex. 5 at 53. T.B.D. was "current with his immunizations; however, it is noted that the cardiac arrest happened [two] days after vaccination." Id. She noted that T.B.D. presented with "delays in gross motor skills and adaptive behavior." Pet. Ex. 11 at 5. He had "limited movement repertoire and work against gravity. He [was] not yet making any transitional movements and ha[d] difficulty elevating his head when in prone and does not tolerate prone positioning." Id.

T.B.D. was evaluated by endocrinologist Dr. Anna Ryabets-Lienhard for abnormal thyroid function studies on February 10, 2016. Pet. Ex. 10 at 90-91. Based on a comprehensive evaluation, Dr. Ryabets-Lienhard assessed T.B.D.'s abnormal thyroid function to be amiodarone-induced. Id. at 124.

Dr. Derek Wong saw T.B.D. for a genetics evaluation on February 11, 2016. Pet. Ex. 10 at 83. Dr. Wong noted T.B.D.'s history of "cardiac arrest with post-ALTE [VT] and [SVT] and hypertrophic cardiomyopathy." Id. at 88. During his hospitalization, T.B.D. had metabolic studies which were mostly normal. Id. at 83-84. Cardiomyopathy panel was normal and the viral myocarditis panel was negative.[25] Id. at 87. Metabolic testing was "essentially normal," except that urine glycosaminoglycans were elevated which may suggest MPS. Id. at 86, 88. It was noted that subtypes of MPS could cause such elevations and "present with cardiomyopathy early in infancy, [but] there is also the possibility that this is a false positive test." Id. at 88. Accordingly, at that time, "the etiology of [T.B.D.'s] condition remain[ed] unknown." Id. Exome sequencing was recommended "to rule out all forms of MPS as well as other genes that could predispose to an [ALTE]." Id.

On February 15, 2016, T.B.D. returned for a cardiology follow-up with Dr. Silka. Pet. Ex. 10 at 78. Echocardiography done that day was normal; the "prior subtle appearance of moderate [left ventricular hypertrophy] not present today." Id. at 81. Dr. Silka noted that a

[25] Here, Dr. Wong references a "viral myocarditis panel" as normal. Pet. Ex. 10 at 87. This appears to be the same myocarditis panel study that was done during hospitalization. See, e.g., Pet. Ex. 8 at 54.

screening test for MPS was positive and further evaluation was pending. Id. at 78. He wrote that "[c]ardiac genetic screening for possible cardiomyopathy has been completed—with no mutation identified." Id. at 79. Dr. Silka again noted that T.B.D.'s cardiac arrest occurred "within 24 hours of his first [DTaP] ([two] month) immunization—although any cause/effect relationship would be speculative." Id. Dr. Silka concluded that if all other work-up was negative, he would "consider this a primary electrical disorder." Id. at 81.

Dr. Hartstein saw T.B.D. on February 22, 2016 for his four-month check-up. Pet. Ex. 5 at 23. Dr. Hartstein documented that at the "[l]ast echo—Dr. Silka said no cardiomyopathy." Id. at 24. Immunizations (Pentacel (DTaP-Hib-IPV), Hep B, and Prevnar 13) were discussed, but were declined by Petitioners due to the "cardiac arrest one day after [two] mo[nth] vaccines." Id. at 25-26. Dr. Hartstein "agree[d] with [Petitioners] that we cannot rule out vaccine as a potential etiology for T.B.D.'s cardiac arrest, now that the hypertrophic cardiomyopathy is resolved and genetic testing is so far negative. We plan to hold off on vaccines until further genetic testing [is] complete." Id. at 26.

On March 23, 2016, T.B.D. was seen for further genetic evaluation by Dr. Derek Wong. Pet. Ex. 5 at 64. Dr. Wong's assessment was that "[a]t this time, there is no evidence of a storage disorder on a clinical basis, and the cardiomyopathy . . . has resolved." Id. at 66. A lysosomal enzyme screening showed no abnormalities in the enzymes tested which included most of the relevant MPS diseases except for MPS subtype II. Id. at 65-66. And Dr. Wong noted it was "unlikely that [T.B.D.] has MPS II." Id. at 66. However, Dr. Wong also noted "[i]t is possible that [T.B.D.] has an MPS disorder and that it has not manifest[ed] yet, or that he has another condition that is giving him the positive test." Id.

On April 4, 2016, neurologist Dr. Brian Wong evaluated T.B.D. Pet. Ex. 5 at 9. He noted T.B.D.'s history of hypertrophic cardiomyopathy, ventricular fibrillation, and hospitalization in December 2015. Id. Dr. Wong also referenced prior MRIs which showed hyperintensities in the right insular region and the periventricular white matter abnormalities. Id. He explained to T.B.D.'s family that the "insular region was most likely damaged and [T.B.D. was] at risk of developing seizures due to his arrhythmia, and that the periventricular white matter discharge was most likely from a remote ante-/perinatal injury." Id. Neurologic assessment noted T.B.D. "has been without seizures and is developing well." Id. at 13. T.B.D.'s tone was mildly increased in the left upper extremity, but he did not have cortical fisting. Id.

At T.B.D.'s six-month check-up with Dr. Hartstein on April 25, 2016, chronic hypoxic-ischemic brain injury was listed as an active problem, and it was noted as "hypoperfusion injury of temporal lobe from cardiac arrest." Pet. Ex. 5 at 15-16. Dr. Hartstein's assessment was that T.B.D. was "doing remarkably well—mostly caught up with development, with the exception of a mild gross motor delay." Id. at 18. She wrote that

> [b]ecause [T.B.D.'s] been improving, [Petitioners] are concerned that [T.B.D.'s] arrest may have been associated with his [two]-month vaccinations since it happened the next day—therefore, they are electing to withhold vaccinations at this time. We had a lengthy discussion about this—there's no way that I can say whether or not his arrest was associated with his vaccinations. There's also a

> known risk of contracting and suffering from a vaccine-preventable illness. They elect to wait while the work-up is being completed and will consider doing immunizations within a controlled setting (while admitted), which is at the suggestion of Dr. Silka, his electrophysiologist.

Id. Dr. Hartstein submitted a Vaccine Adverse Event Reporting System ("VAERS") report the same day. Pet. Ex. 9 at 1-3. The adverse event was "cardiopulmonary arrest." Id. at 3. Dr. Hartstein called T.B.D.'s mother the following day "to let her know [she] submitted a VAERS report for [T.B.D.'s] cardiopulmonary arrest one day after his [two] month vaccines." Pet. Ex. 5 at 116. During the call, T.B.D.'s mother indicated she contacted a vaccine lawyer to discuss his case. Id.

On April 28, 2016, T.B.D. was seen for a cardiology follow-up with Dr. Silka. Pet. Ex. 5 at 5. Dr. Silka noted that all "cardiac genetic screening for possible cardiomyopathy has been completed—with no mutation identified." Id. "Clinically, he appears to be progressing well during his work with physical therapy. There are no new problems and he appears to be growing and developing as a normal infant." Id. Dr. Silka assessed T.B.D. as "clinically stable – no apparent events/arrhythmias." Id. at 7. Dr. Silka "had a long discussion with the family as testing continues with no apparent cause/basis for his initial [ventricular fibrillation] arrest. . . . We also discussed the need to proceed with his immunizations[] once the results of the exome are established." Id. at 8.

T.B.D. returned to Dr. Silka on June 27. Pet. Ex. 10 at 19. Dr. Silka noted that the whole exome clinical sequence testing was completed, and no abnormality was identified. Id. "Due to this result, Dr. Derek Wong has advised no further genetic investigation or repeat of the MPS screening." Id. Dr. Silka wrote that they "discussed results of testing to date and the POSSIBILITY that the arrhythmias were [due] to an untoward/unanticipated reaction to the [DTaP] vaccine. So far, [Petitioners] have declined the [second] dose of [DTaP]." Id. at 22.

On July 15, 2016, T.B.D. was seen by physical therapist Theodore Nguyen. Pet. Ex. 13 at 1-4. The visit summary noted that he still presented "with decreased trunk and lower extremity strength required for independent mobility." Id. at 2.

On October 28, 2016, T.B.D. was seen for his 12-month check-up by Dr. Hartstein. Pet. Ex. 5 at 101. Dr. Hartstein noted testing for "Brugada syndrome[26] — negative. No answer for his arrhythmia." Id. at 102. Dr. Hartstein noted T.B.D. had a gross motor function developmental delay. Id. at 103, 105. Immunizations were refused. Id. at 104. Dr. Hartstein's note stated "uncertain of the etiology of [T.B.D.'s] cardiac arrest, but because it happened 24 hours after receiving his first vaccines, cannot guarantee safety with subsequent vaccinations." Id. at 105.

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[26] Brugada syndrome is "an autosomal dominant ion channelopathy characterized by sudden, idiopathic ventricular fibrillation in an apparently healthy person . . . It is genetically heterogeneous, with the most common cause being mutation in the SCN5A gene." Brugada Syndrome, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=110340 (last visited Aug. 20, 2025).

T.B.D. was seen by neurology for follow-up on November 4, 2016. Pet. Ex. 10 at 2. Dr. Brian Wong stated T.B.D. "has been thriving since his last visit [six] months ago. He is caught up developmentally." Id. at 3. T.B.D. continued to be followed by cardiology, but "at this point does not have a working diagnosis." Id. T.B.D. was continuing in physical and occupational therapy once per week and was primarily working on ambulation. Id. Physical and neurological examination was normal. Id. at 3-4. Because T.B.D.'s development was on track and he was seizure free, no further workup was required, with neurology follow-up visits as needed. Id. at 8.

On January 18, 2017, Dr. Silka saw T.B.D. for a cardiology follow-up. Pet. Ex. 15 at 13. Dr. Silka determined there had been no arrhythmias for the past 12-month interval and that T.B.D.'s extensive work up was nondiagnostic. Id. at 16. T.B.D.'s parents remained concerned that vaccines may have caused his cardiac arrest. Id. Dr. Silka "discussed the National Vaccine Injury Program" with Petitioners, and agreed to "write them an objective statement of what is known and what is uncertain as regards [T.B.D.]" Id.; see also Pet. Ex. 21.

Dr. Hartstein noted that certain vaccines, including DTaP, were "[c]ontraindicated" at T.B.D.'s 15-month well-child visit on January 31, 2017. Pet. Ex. 5 at 98.

T.B.D returned to Dr. Hartstein on March 24, 2017 to evaluate his behavior of "squeezing hands with whimpering." Pet. Ex. 5 at 88. History indicated "one month [history of] holding his hands and pulling on them." Id. Dr. Hartstein was concerned that T.B.D. may have "paresthesias from the amiodarone" given the length of time T.B.D. had been on the medication. Id. at 88, 90. While T.B.D.'s cardiologist did not think amiodarone was the cause of the symptoms, the plan was to wean off the amiodarone. Id. at 90-91.

On May 2, 2017, Dr. Hartstein evaluated T.B.D. for his 18-month visit and wrote he "seem[ed] totally fine." Pet. Ex. 5 at 83-84. The problem list noted "[d]ilated cardiomyopathy secondary to mucopolysaccharidosis" had resolved and was ruled out by Dr. Derek Wong, and exome testing was negative. Id. at 85. No further genetics workup was recommended. Id. T.B.D. continued physical therapy for gross motor delay. Id. Immunizations were discussed but declined by Petitioners who requested a "letter of medical exemption." Id. at 86.

No new cardiac problems were reported at T.B.D.'s cardiology follow-up with Dr. Silka on May 8, 2017. Pet. Ex. 15 at 8. T.B.D. had discontinued amiodarone several months prior. Id. "Developmentally, he appear[ed] to be above average in terms of higher order cognitive function, based on the assessment of his physical therapist." Id. at 9. It was noted that "Dr. Hartstein ha[d] discussed [T.B.D.'s] immunization status with [] immunology and the decision has been made to not to proceed with further immunizations at this time." Id.

On May 22, 2017, T.B.D. presented to endocrinology for follow-up by Dr. Ryabets-Liehard. Pet. Ex. 15 at 1. Dr. Ryabets-Liehard noted that T.B.D.'s "[thyroid function tests] now have been normal for the past 12 mo[nths]." Id. "No known etiology for his cardiac issue at this time." Id. at 2.

An echocardiogram performed on November 5, 2018 showed normal cardiac size and function. Pet. Ex. 24 at 24, 26.

At a follow-up with cardiology on November 16, 2020, Dr. Silka noted T.B.D.'s lack of new cardiac symptoms and documented that he was no longer on cardiac medication. Pet. Ex. 26 at 3. Dr. Silka's assessment was "[status post] unexplained [out-of-hospital ventricular fibrillation], possibly (temporally) related to [two]-month vaccinations. No recurrences of arrhythmias since initial presentation [five] years ago. . . . Remaining uncertainty regarding the cause of the initial cardiac arrest once again discussed. Future progress in genetic testing may be basis for repeat genetic arrhythmia panel testing in next [two to three] years." Id. at 7.

While additional records have been filed, they are not relevant to the issues in dispute and are therefore not discussed.

C. Letters from Treating Providers

1. Dr. Michael J. Silka

On January 19, 2017, T.B.D.'s treating cardiologist (since December 16, 2015) authored a letter to "[w]hom it may concern." Pet. Ex. 21 at 1. He described T.B.D. as a healthy infant who received DTaP, IPV, Hib, Hep B, Rotavirus, and Prevnar 13 vaccines on December 15, 2015. Id. "Other than some mild fussiness post immunization, no adverse effects were noted by [Petitioners]." Id. But on December 16, T.B.D. "suddenly lost consciousness and became apneic." Id. T.B.D. was "documented to be in ventricular fibrillation and required several defibrillator shocks to restore sinus rhythm. He continued to have ventricular arrhythmias following admission to the CHLA ICU with eventual control using amiodarone and esmolol." Id. An "extensive array" of cardiac and genetic tests, including "whole exome analysis, provocative pharmacologic stimulation to assess for possible Brugada syndrome[,] and multiple event monitoring and cardiac imaging sequences," were performed and "the cause of [T.B.D.'s] cardiac arrest remains undefined." Id.

Dr. Silka concluded that at the time of writing his letter, "the cause of [T.B.D.'s] cardiac arrest one day post immunization remains undefined. Given the absence of any other plausible explanation, consideration of a possible adverse reaction to one of his immunizations remains a distinct possibility." Pet. Ex. 21 at 1.

2. Dr. Jennifer A. Hartstein

T.B.D.'s treating pediatrician, Dr. Hartstein, authored a letter on May 7, 2017, to serve as a "medical exemption for all vaccinations." Pet. Ex. 17 at 1. Dr. Hartstein wrote that T.B.D. "suffered from a full cardiac arrest with subsequent arrhythmia one day after his [two]-month vaccinations (DTaP-IPV-HiB, Hep B, [Prevnar 13], and [Rotavirus]). He was hospitalized for over a month and requires close cardiac follow-up." Id. Dr. Hartstein concluded it is "unknown what precipitated the cardiac arrest, but immunizations have been held at the recommendation of [T.B.D.'s] electrophysiologist to avoid a possible trigger for further arrhythmia." Id. She stated this was a "permanent exemption." Id.

D. Affidavits

1. T.B.D.'s Mother

On November 4, 2017, T.B.D.'s mother, Vanessa Drake ("Mrs. Drake"), executed an affidavit. Pet. Ex. 18 at 5. T.B.D. was born through a normal vaginal delivery at 36.3 weeks on October 22, 2015, weighing 6.6 pounds. Id. at ¶¶ at 3-4. She recalled the first seven weeks of his life, T.B.D. was "in good health and appeared to be a normal, healthy, alert baby." Id. at ¶ 5. She nursed him frequently and T.B.D. enjoyed physical contact. Id. Whenever Mrs. Drake would put T.B.D. down, "he would wake within minutes and cry to be held but he was immediately and easily soothed whenever [her] husband [] or [she] picked him up." Id.

Petitioners took T.B.D. for his two-month well visit on December 15, 2015. Pet. Ex. 18 at ¶ 6. Mrs. Drake was told that T.B.D. "was a perfectly healthy baby and his growth was on track." Id. The nurse administered the round of vaccinations and T.B.D. "began to scream and cry immediately. [She] had never heard him scream and cry so forcefully in his short life." Id. They left the doctor's office and returned home but T.B.D.'s "cries and screams intensified" for the three hours following the December 15 vaccinations. Id. at ¶ 7. Mrs. Drake recalled T.B.D. was inconsolable and nothing would comfit him, not even nursing. Id. She "reluctantly gave him a dose of infant Tylenol because his cries and screams seemed so unusual and painful and within 30 minutes he fell asleep." Id.

The next morning, on December 16, 2015, Mrs. Drake noticed T.B.D. was lethargic; he "did not fuss to be held, cuddled, or nursed as he normally did." Pet. Ex. 18 at ¶ 8. "The few times he did open his eyes, he did not focus on [Mrs. Drake] or respond to [her] voice as he normally did." Id. Mrs. Drake just assumed he was "extra sleepy from his vaccinations." Id. Mrs. Drake's mother (T.B.D.'s grandmother), who had been staying with Petitioners on and off since T.B.D. was born, was then watching T.B.D. while Mrs. Drake showered and dressed for the day. Id. at ¶ 9. She told Mrs. Drake that she "noticed something different about [T.B.D.]—that he would not look her in the eyes and that his eyes appeared vacant." Id. Mrs. Drake's mother left at approximately 9:30 a.m. Id.

Mrs. Drake was normally "accustomed to holding [T.B.D.] all day while he slept." Pet. Ex. 18 at ¶ 10. But on the morning of December 16, 2015, T.B.D. "did not protest when [she] laid him down in his rock-n-play. Surprised, [Mrs. Drake] took [T.B.D.] into his room and laid him in his crib, and for the first time ever he just lay there quietly, and closed his eyes." Id. Her and her husband took a picture of him because they "could not believe he let [them] lay him down in his crib." Id. Mrs. Drake did laundry, walking back and forth T.B.D.'s room "admiring him taking his first nap on his own in his crib." Id.

At approximately 1:45 p.m. on December 16, Mrs. Drake moved T.B.D. to the living room where she nursed him, and he fell asleep. Pet. Ex. 18 at ¶ 11. Mrs. Drake recalled that within 10 minutes, T.B.D. awoke crying with "that same piercing, painful cry as the day before." Id. She tried to nurse him again "but he refused and just screamed louder. As [she] held him up, he grabbed [her] hair, his eyes rolled back, his skin and lips went pale, and he went silent and

lifeless." Id. She though he had stopped breathing and wondered whether he was choking. Id. Mrs. Drake called 911 but could not reach a live operator. Id. at ¶ 12. She "ran out into the street screaming for help and started CPR on [T.B.D.]." Id. Her neighbor was able to reach 911 and Mrs. Drake shouted that her "baby stopped breathing" and she still thought he choked. Id.

The paramedics arrived and transported T.B.D. to the hospital where he was stabilized. Pet. Ex. 18 at ¶ 13. Mrs. Drake explained that the doctors told her T.B.D. "suffered a cardiac arrest in the ambulance and his heart was continuing to have arrhythmias." Id. at ¶ 14.

Mrs. Drake recalled that T.B.D. spent 34 nights at the hospital during which imaging revealed "he had suffered brain damage and a slight thickening of his heart wall." Pet. Ex. 18 at ¶ 15. During his hospitalization, T.B.D. "endured life-threatening conditions as a result of the tests and treatment he was undergoing. He developed a blood clot and had to be placed on blood thinners. [T.B.D.'s] heart rhythms fluctuated wildly while the hospital staff tried to find the correct heart medications for his condition." Id. at ¶ 16.

T.B.D. was released from the hospital on January 19, 2016. Pet. Ex. 18 at ¶ 17. Per Mrs. Drake, Petitioners had to give T.B.D. "two potent and dangerous heart medications" every 12 hours for over a year. Id. In January 2017, his heart medication changed and as of the date of the affidavit, Petitioner still give it to him twice a day. Id. Mrs. Drake averred T.B.D.'s gross motor function and development is below average for his age. Id. at ¶ 18. "He could not sit up until [eight] months old, and [he] did not crawl until [one] week before his [first] birthday. He took his first steps at 17 months old." Id.

2. T.B.D.'s Father

On November 4, 2017, T.B.D.'s father, Lance Drake ("Mr. Drake"), executed an affidavit. Pet. Ex. 19 at 2. He recalled that on December 16, 2015, the morning after T.B.D.'s vaccinations, T.B.D. was "uncharacteristically quiet and lethargic" compared to the first seven weeks of his life. Id. at ¶ 3. Normally Petitioners would have to be quiet when T.B.D. napped or "he would wake up and want to be held." Id. But that morning, Petitioners were able to get things done around the house that would have awakened T.B.D. in the past. Id. at ¶¶ 3-4. T.B.D. was "so lethargic, [Petitioners] were able to move him to his crib to nap for the first time." Id. at ¶ 4. Shortly after, Mr. Drake left the house for the day and the next time he saw T.B.D. was in the emergency room "fighting for his life." Id. at ¶ 5.

3. T.B.D.'s Grandmother

On October 30, 2017, T.B.D.'s grandmother and Mrs. Drake's mother, Karen Kisvarday ("Ms. Kisvarday"), executed an affidavit. Pet. Ex. 20 at 2. After the birth of T.B.D., she spent many nights at the Drake family home to spend time with her grandson, including the night of December 15, 2015. Id. at ¶ 3. The next morning, at approximately 8:30 a.m., Ms. Kisvarday was holding T.B.D. and talking to him but recalled T.B.D. "seemed strangely detached." Id. at ¶ 4. "He didn't look at [her]. In fact, he was not moving in [her] arms or looking at anything. He just gazed to the side of [her], unfocused for an hour." Id. She was "disturbed by how unreactive, unfocused, and completely distant [T.B.D.] was" so she sat on the couch and held him. Id. at ¶ 5.

During that time, T.B.D. did not fall asleep. Id. "He had his eyes open but he never moved. He looked very lethargic and dazed." Id. Ms. Kisvarday had to leave the house at approximately 9:20 a.m., but because she was uneasy leaving with how strange T.B.D. was acting, she mentioned his behavior to Mrs. Drake. Id. at ¶ 6.

E. Expert Opinions

1. Petitioner's Expert, Dr. Anthony Chang[27]

a. Background and Qualifications

Dr. Chang is a board-certified pediatrician with a sub-board certification in cardiology. Pet. Ex. 23 at 6. He received his M.D. from Georgetown University Medical School and subsequently competed a pediatrics residency at the Children's Hospital National Medical Center in Washington, DC, and a cardiology fellowship at the Children's Hospital of Philadelphia. Id. He has authored or co-authored numerous publications primarily in cardiology. Id. at 20-33. Dr. Chang has been "actively involved in the care of over 100,000 pediatric patients with [congenital heart disease] as an attending in the cardiac intensive care unit and as an attending pediatric cardiologist with clinical interest in heart failure and sudden cardiac death for close to 30 years, including diagnosis such as cardiomyopathy." Id. at 5.

b. Opinions

In his first expert report, Dr. Chang opined that "[t]o a reasonable degree of medical probability" T.B.D. suffered a sudden cardiac arrest "as a direct result of cardiac sequelae from his immunization[s] on [December 15, 2015]." Pet. Ex. 23 at 4. He deemed this an "adverse reaction to [] vaccination." Id.

Regarding diagnosis, Dr. Chang opined that T.B.D. "acute hypersensitivity myocarditis,[28] an inflammatory condition of the heart muscle, following [] vaccination." Pet. Ex. 23 at 4. The causal mechanism of T.B.D.'s acute hypersensitivity myocarditis was inflammation, "an inflammatory response that connected vaccination and this injury." Id.

Dr. Chang opined there was a logical sequence of cause and effect between vaccination and "the sequelae of cardiac arrest and hypoxic ischemic brain damage." Pet. Ex. 23 at 4. He explained that T.B.D. was healthy prior to vaccination, with no respiratory symptoms. Id. Further, Dr. Chang noted that T.B.D. underwent an extensive diagnostic workup and genetic testing that was ultimately negative for a "pre-existing cardiac risk factor or disease." Id. Dr. Chang opined that "the most plausible explanation for [T.B.D.'s] relatively sudden deterioration"

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[27] Dr. Chang submitted three expert reports. Pet. Exs. 23, 27, 33.

[28] Hypersensitivity myocarditis is "myocarditis due to allergic reactions caused by hypersensitivity to various agents, particularly sulfonamides, penicillins, and methyldopa." Hypersensitivity Myocarditis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=91286 (last visited Aug. 20, 2025).

was a "lethal ventricular tachydysrhythmia such as ventricular tachycardia or ventricular fibrillation." Id. Since there was no other alternative cause and no family history that would explain sudden cardiac arrest, Dr. Chang opined "there is preponderant evidence that [] vaccination" was the cause of T.B.D.'s injury. Id.

Lastly, Dr. Chang noted there was a "definite proximate temporal relationship" between vaccination and the injury. Pet. Ex. 23 at 4.

In his second report, Dr. Chang added the injury of "acute encephalopathy" to his diagnosis opinion. Pet. Ex. 27 at 6. He stated that T.B.D. "suffered acute encephalopathy and his sudden cardiac arrest as a direct result of his cardiac sequelae" from vaccination. Id.

Dr. Chang disagreed with Respondent's expert, Dr. Ringel, that there was "no cardiac manifestation of [] myocarditis." Pet. Ex. 27 at 6. Dr. Chang opined that "heart dysrhythmias (especially both atrial and ventricular tachydysrhythmias) [] could be a manifestation of myocarditis." Id. Further, Dr. Chang noted that Dr. Ringel failed to recognize that left ventricular dysfunction seen on T.B.D.'s second echocardiogram could also be a manifestation of myocarditis. Id. at 6-7. Moreover, Dr. Chang opined that T.B.D.'s "sudden collapse" without "obvious seizure activity" was consistent with a "sudden cardiac arrest." Id. at 7.

Although Dr. Chang thought it was plausible that "encephalopathy alone" could lead to cardiac arrest, he thought it was "much more likely" that T.B.D. had "both myocarditis and encephalopathy." Pet. Ex. 27 at 7. He explained that these "two manifestations of inflammation" caused by vaccination are "not mutually exclusive," and instead, they were "in fact coupled."[29] Id.

Dr. Chang's third report also identifies T.B.D.'s vaccine related condition as "acute hypersensitivity myocarditis." Pet. Ex. 33 at 2. He cited a case report by Thanjan et al.,[30] in support of his causal opinions. Id. (citing Pet. Ex. 35). The report describes a 17-year-old who developed acute myocarditis two days after receiving DTaP, meningococcal conjugate, and hepatitis A vaccinations. Pet. Ex. 35 at 3. The day after his vaccinations, the patient had diffuse arthralgia, chest pain, and low-grade fever. Id. Physical examination was normal, but EKG was abnormal (ST-segment elevation), and serum cardiac enzymes were elevated (troponin, creatine kinase, and creatine kinase MB). Id. Testing was negative for adenovirus, enterovirus, Lyme disease, cytomegalovirus, Epstein-Barr virus, antinuclear antibody, and anti-double stranded DNA. Id. at 4. Cardiac MRI showed enhancement in areas of the left ventricular wall. Id. Over the next several days, cardiac enzymes decreased, and the patient improved and was discharged.

[29] Dr. Chang did not develop the opinion that T.B.D. had two distinct injuries, myocarditis and encephalopathy, independent of each other. Since this idea was not developed, and seemed contrary to the weight of the evidence, the undersigned did not find it persuasive, and did not factor it into the analysis.

[30] Maria T. Thanjan et al., Acute Myopericarditis After Multiple Vaccinations in an Adolescent: Case Report and Review of the Literature, 119 Pediatrics e1400 (2007). This study was also filed as Respondent's Exhibit C.

Id. One week after onset of symptoms, his cardiac enzymes, and abnormal ST-segment elevation had normalized. Id. Ten weeks after onset, EKG was normal and MRI showed decreased enhancement of the left ventricle. Id. He remained asymptomatic thereafter. Id. at 3-4.

Thanjan et al. identified hypersensitivity reaction as the causal mechanism of acute myocarditis following vaccination, primarily based on the temporal association and the lack of alternative causes by infectious agents. Pet. Ex. 35 at 4. The authors described the pathogenesis as a "maladaptive immune response that leads to myocardial injury" based on biopsies which showed "CD3+ T-cell infiltrate[31] with prominent degranulating eosinophils" that have been observed after smallpox vaccination. Id. at 4-5. Troponin elevations have a "high specificity in supporting the diagnosis, especially in conjunction with strong clinical suspicion." Id. at 5. Cardiac MRI is "highly sensitive and specific for myocarditis detection" and may show "enhancement of the myocardium" which is "usually subepicardial," distinct from that the "subendocardial involvement [] seen with ischemia and infarction." Id.

Lastly, Thanjan et al. explained that most cases of "postvaccination myopericarditis are self-limited." Pet. Ex. 35 at 5. Treatment included either no medications, or nonsteroidal anti-inflammatory medications, or corticosteroids. Id.

Regarding the timing of events, Dr. Chang opined in his third report that T.B.D.'s sudden cardiac arrest the day following vaccination was "preceded by clinical features suggestive of acute encephalopathy." Pet. Ex. 33 at 3. He further opined that T.B.D.'s acute encephalopathy supported the "retrospective circumstantial diagnosis of acute hypersensitivity myocarditis." Id.

In addition to Thanjan et al., Dr. Chang referenced three other case reports. Dilber et al.[32] described a 14-year-old with fever and intermittent chest pain three days after tetanus vaccination. Pet. Ex. 36 at 2-3. The patient had elevated troponin, myoglobulin, and creatinine kinase levels, as well as elevated creatine kinase-MB fraction. Id. at 3. Echocardiography was normal. Id. The initial EKG showed "mild ST-segment elevation" which progressed in several days to "inverted T waves." Id. Three days later the patient was asymptomatic, EKG was normal, and cardiac enzyme levels had decreased. Id. Diagnosis was hypersensitivity myocarditis. Id. at 2-3. The authors noted that heart involvement can occur within hours of drug exposure, resulting in tachycardia, "mild cardiomegaly, conduction delays, [] nonspecific ST-T changes," and elevated cardiac enzymes. Id. at 3. The mechanism of action was thought to be a "delayed hypersensitivity reaction." Id. The authors concluded that "[h]ypersensitivity

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[31] CD3+ T-cells are a type of T lymphocytes, "the cells primarily responsible for cell-mediated immunity." T Lymphocytes, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=87562 (last visited Aug. 25, 2025). Infiltrate is "the pathologic accumulation in tissue or cells of substances not normal to it or in amounts in excess of the normal." Infiltration, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=25196 (last visited Aug. 25, 2025).

[32] Embiya Dilber et al., Acute Myocarditis Associated with Tetanus Vaccination, 78 Mayo Clinic Procs. 1431 (2003).

myocarditis should be considered when new [EKG] changes occur in association with acute-onset chest pain, mildly elevated cardiac enzyme levels, and eosinophilia due to drugs and vaccination." Id. at 4.

Boccara et al.[33] reported a case of myopericarditis in a 31-year-old patient four days after a diphtheria, tetanus, and polio vaccination. Pet. Ex. 37 at 2. The patient experienced chest pain, EKG changes, and elevated cardiac enzymes. Id. Prior to hospital admission, he had fever, chills, chest pain, and elevated heart rate. Id. Serum blood count revealed elevated eosinophilia (2.9%). Id. Echocardiography was normal. Id. Biopsy showed interstitial edema of cardiac muscle. Id. at 2-3, 2 fig.1. Diagnostic testing for viruses and bacteria was negative. Id. at 3. The patient's subsequent clinical course was uneventful and he was discharged five days after admission. Id. The mechanism of hypersensitivity myopericarditis was suggested, which they described as "an immune complex-mediated[34] pathogenic mechanism" supported by fever, arthralgias, chest pain, laboratory, and histology findings, although the authors acknowledged the lack of direct evidence of causation. Id.

Lastly, Kumar et al.[35] reported myocarditis in a six-week-old infant with a congenital heart defect "large ventricular septal defect" ("VSD")[36] who had onset of illness two days after diphtheria, whole cell pertussis, tetanus ("DPT") vaccine. Pet. Ex. 38 at 2.[37] On presentation, the infant had a severely increased heart rate, low blood pressure, increased respiratory rate, hypoxia, and metabolic acidosis. Id. EKG showed ST segment depression. Id. Echocardiogram revealed severe ventricular dysfunction. Id. Cardiac enzymes were elevated. Id. The infant was treated with methylprednisolone, gradually improved, and was discharged after four days. Id. at 2-3. Later she was given the acellular form of the vaccine without adverse reaction. Id. at 3. The authors suggested that the proposed mechanism of the infant's myocarditis was a

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[33] Franck Boccara et al., Acute Myopericarditis After Diphtheria, Tetanus, and Polio Vaccination, 120 Chest 671 (2001).

[34] Immune complex–mediated hypersensitivity reaction is a "local or general inflammatory response due to formation of circulating antigen-antibody complexes and their deposition in tissues; the complexes activate complement and other inflammatory mediators, initiating processes . . . resulting in tissue damage." Type III Hypersensitivity Reaction, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=119516 (last visited Aug. 22, 2025).

[35] Vivek Kumar et al., Myocarditis Following Diphtheria, Whole-Cell Pertussis, and Tetanus Toxoid Vaccination in a Young Infant, 11 Annals Pediatric Cardiology 224 (2018).

[36] VSD is "a congenital cardiac anomaly in which there is persistent patency [i.e. opening . . .] of the ventricular septum." Ventricular Septal Defect, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=68728 (last visited Aug. 20, 2025).

[37] The Kumar et al. article is identified as Petitioner's Exhibit 38 in the Notice of Filing. ECF No. 93. However, the article is incorrectly labeled as Petitioner's Exhibit 39. The undersigned will refer to the article as Petitioner's Exhibit 38.

"maladaptive immune dysfunction." Id. They summarized three other reported cases in infants, two who received DPT (whole cell pertussis) vaccines and one who received diphtheria, polio, and tetanus vaccines. Id. at 3 tbl.2.

2. Petitioner's Expert, Dr. Perry Lubens[38]

a. Background and Qualifications

Dr. Lubens is a board-certified pediatric neurologist. Pet. Ex. 30 at 1. He received his M.D. from the University of Michigan. Id. at 3. Thereafter, he competed a pediatric residency at the Children's Hospital Medical Center in Cincinnati, Ohio, a neurology residency at the University of California, Los Angeles ("UCLA") Department of Neurology, and a pediatric neurology fellowship at UCLA Division of Pediatric Neurology. Id. Dr. Lubens currently has a private neurology practice, is an Attending Pediatric Neurologist at Miller Children's Hospital of Long Beach Memorial Medical Center, is the Medical Director of the Neurodiagnostic Department of Long Beach Memorial Medical Center, and is an Associate Clinical Professor in the Department of Pediatric Neurology at the University of California, Irvine School of Medicine. Id. at 4.

b. Opinions

Dr. Lubens offered opinions about the cause and nature of T.B.D.'s acute encephalopathy in support of Petitioners' Table claim of acute encephalopathy following the DTaP vaccination. Pet. Ex. 30 at 1-2; Pet. Ex. 32 at 1-2. He opined that the DTaP vaccination was the cause of T.B.D.'s acute encephalopathy, stating that "DTaP induced encephalopathy is . . . a well-recognized condition" experienced by a "very small percentage of vaccinees." Pet. Ex. 32 at 2.

Based on the affidavits submitted by T.B.D.'s mother and grandmother, Dr. Lubens opined T.B.D. had a "significant decrease in alertness . . . within approximately 24 hours of his vaccination [] indicative of acute clinical encephalopathy." Pet. Ex. 30 at 1; see also Pet. Ex. 39 at 5. His "symptoms of acute encephalopathy preceding his cardiac arrest . . . clearly worsened after the arrest." Pet .Ex. 30 at 1. Dr. Lubens emphasized that T.B.D.'s "significant decrease in level of consciousness and alertness" occurred "prior to any medication or sedatives being administered to T.B.D." Id. at 1-2. He further opined that T.B.D. did not have any underlying medical condition or disease that "caused or contributed to [his] encephalopathy." Id. at 2.

According to Dr. Lubens, T.B.D.'s parents and grandmother described him as "lethargic, strangely detached, not moving" as well as "unreactive, unfocused, and completely distant." Pet. Ex. 32 at 1. T.B.D. "screamed and cried" and was "inconsolable," and when nursed, "he refused and just screamed louder." Id. Based on these behaviors, Dr. Lubens opined T.B.D. had acute vaccine-induced encephalopathy. Id.

[38] Dr. Lubens submitted three expert reports. Pet. Exs. 30, 32, 39. Dr. Lubens also conducted an independent medical examination of T.B.D. on July 29, 2021. Pet. Ex. 34. Dr. Lubens' examination is not relevant to causation, and thus, only his conclusion is discussed herein.

Regarding a temporal association, Dr. Lubens noted that T.B.D.'s acute encephalopathy "occurred within 24 hours of vaccination." Pet. Ex. 32 at 2.

Dr. Luben disagreed with Respondent's expert Dr. Bingham's opinion that cardiac arrest caused T.B.D.'s acute encephalopathy. Pet. Ex. 32 at 2. Instead, Dr. Lubens asserted that T.B.D.'s acute encephalopathy occurred before his cardiac arrest. Id. Further, he opined that T.B.D.'s "cardiac arrest played no role in causing his acute encephalopathy." Id. He explained that the MRI showed evidence of a "brain inflammatory process," distinct and "independent of his myocarditis." Id.

In addition, Dr. Lubens offered opinions related to the two separate abnormalities/injuries to T.B.D.'s brain as reflected in the medical records and MRI studies performed in 2015. Pet. Ex. 39 at 1. He opined that the MRIs show an injury to the "insular region . . . likely [as] a result of the sudden cardiac arrest." Id. at 2. The MRIs also show "changes in the periventricular white matter." Id. Dr. Luben disagreed with the treating physicians and experts who opined that the periventricular white matter injury reflects a remote injury that occurred during the prenatal period or were due to prematurity. Id. at 2-6. Instead, Dr. Luben opined that the periventricular white matter injury was caused by T.B.D.'s vaccination on December 15, 2015. Id. at 3-4.

He offered four reasons for this opinion. Pet. Ex. 39 at 3. First, Dr. Lubens opined that T.B.D. was born at 36 weeks and four days, which is "late preterm," and at that point, T.B.D.'s brain volume was "very close to term," "making it extremely unlikely that the periventricular white matter changes occurred during the prenatal period." Id. Second, Dr. Lubens opined that if the periventricular white matter changes were due to prematurity, "there would have been a loss of white matter volume seen on MRI," which was not seen. Id. Third, there were no other findings (accompanying cysts, ventriculomegaly, or "thinning of the periventricular white matter") typically seen if this abnormality was due to prematurity. Id. And fourth, from the time of birth until the date of the cardiac arrest there was "no clinical evidence . . . of neurological deficient." Id.

Lastly, Dr. Lubens opined, based on his review of the records and examination in July 2021,[39] T.B.D. has made "a very significant recovery" but has "behavior and cognitive problems" consistent with the brain injury caused by his cardiac arrest and "vaccine induced acute encephalopathy." Pet. Ex. 39 at 5-6.

3. Respondent's Expert, Dr. Richard E. Ringel[40]

a. Background and Qualifications

Dr. Ringel is a board-certified pediatrician with a sub-board certification in pediatric cardiology. Resp. Ex. B at 7. He received his M.D. from the Albert Einstein College of Medicine in New York and subsequently completed a pediatric residency and a pediatric

[39] See Pet. Ex. 34 (independent medical examination performed July 29, 2021).

[40] Dr. Ringel submitted two expert reports. Resp. Exs. A, F.

fellowship in pediatric cardiology at the University of Maryland Hospital. Id. at 1. Dr. Ringel has been a "pediatric cardiologist for 35 years and [] Director of Pediatric and Congenital Heart Catheterization at Johns Hopkins Hospital for . . . 18 years." Resp. Ex. A at 5. He is currently a Professor of Pediatrics in the Division of Pediatrics Cardiology at the Johns Hopkins School of Medicine. Resp. Ex. B at 1. He has authored or co-authored numerous publications. Id. at 1-4, 6.

b. Opinions

In his first report, Dr. Ringel offered opinions related to Petitioners' causation-in-fact claim asserting that T.B.D. suffered acute vaccine-induced hypersensitivity myocarditis which caused his arrhythmia and cardiac arrest. Resp. Ex. A at 4-5.

Dr. Ringel disagreed with the diagnosis of hypersensitivity myocarditis and theory of causation for two reasons. Resp. Ex. A at 4-5. First, he asserted that T.B.D.'s history was "very dissimilar" from the case reports by Thanjan et al. and others cited by Petitioners. Id. at 4. He opined that the cited cases involved adolescents, with no arrhythmias reported, and MRI findings consistent with myocarditis. Id. Further, Dr. Ringel noted that the case reports in 2008 showed echocardiograms with ventricular dysfunction and "very abnormal left ventricular ejection fractions,"[41] whereas T.B.D. had "normal left ventricular function" based on echocardiograms done the day of his cardiac arrest and two days later. Id. Further, Dr. Ringel noted that in two of the other case reports of vaccine-related myocarditis, myocardial biopsy showed eosinophilic myocarditis. Id. (citing Resp. Ex. D).[42] Dr. Ringel noted that T.B.D. did not have a myocardial biopsy. Id.

Next, Dr. Ringel stated that although T.B.D.'s genetic testing did not show any genetic cause for his cardiomyopathy, such testing does identify all inherited cases of cardiac conditions. Resp. Ex. A at 4-5. He cited Pua et al.,[43] who noted that inherited cardiac disorders are not found in "up to 12% of samples." Id. at 5 (citing Resp. Ex. E).

In his second report, Dr. Ringel offered opinions related to Petitioners' Table claim based on acute encephalopathy. Resp. Ex. F at 1-2. He opined that based on his review of Petitioners' affidavits and the medical records, T.B.D. did not meet the Vaccine Injury Table criteria for acute encephalopathy. Id. at 2. Moreover, Dr. Ringel asserted that even if present,

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[41] T.B.D.'s ejection fraction appears in the records; however, the results were not interpreted by the treating physicians, or if they were, it is not apparent whether they were normal or abnormal. Due to the lack of evidence about T.B.D.'s ejection fraction, and the significance of it, the undersigned does discuss it or factor the results into her findings or Ruling.

[42] Michelle Barton et al., Eosinophilic Myocarditis Temporally Associated with Conjugate Meningococcal C and Hepatitis B Vaccines in Children, 27 Pediatric Infectious Disease J. 831 (2008).

[43] Chee Jian Pua et al., Development of a Comprehensive Sequencing Assay for Inherited Cardiac Condition Genes, 9 J. Cardiovascular Translational Rsch. 3 (2016).

encephalopathy "would not help produce a connection to his claim of vaccine-induced hypersensitivity myocarditis" and that the patients in the case reports did not have encephalopathy. Id.

Dr. Ringel offered two alternative causes for T.B.D.'s cardiac arrest in his second report, which he characterized as "rather common explanations" for T.B.D.'s presentation: (1) "vomiting and aspiration producing respiratory arrest and subsequent cardiac arrest" and (2) "viral myocarditis resulting in arrhythmia followed by cardiac arrest." Resp. Ex. F at 3-4. He cited portions of the medical records in support of these opinions. See id. at 3 (noting an examination found "vomitus in the airway"). As for his opinion that a virus caused T.B.D.'s myocarditis and arrythmia, Dr. Ringel stated that acute viral myocarditis can present "with lethal arrythmia and little else in the way of symptoms." Id. at 4. Dr. Ringel explained that although T.B.D. had a peripheral "viral panel" two weeks after his cardiac arrest, and it was negative, this does not rule out a viral case, since this test is known to have a "low yield in infants and children with . . . myocarditis."[44] Id.

In conclusion, Dr. Ringel disagreed that Dr. Chang offered information to support the diagnosis of encephalopathy under the Vaccine Injury Table. Resp. Ex. F at 4. He also disagreed that the medical literature supported a finding of "vaccine-induced hypersensitivity myocarditis" and instead offered an alternative cause of viral myocarditis. Id. Dr. Ringel did not offer opinions distinguishing the clinical presentation of viral myocarditis from that of hypersensitivity myocarditis as it relates to lethal arrythmias.

Dr. Ringel cited two papers related to myocarditis/pericarditis in children. Resp. Ex. F at 3-4. Barton et al. (2008) described two cases and provided a summary of a literature review identifying 37 papers that reported 269 cases of post-vaccination myocarditis/pericarditis (from 1966 to 2007). Resp. Ex. D at 1. Starting with the case reports, the first involved a 12-year-old girl who developed fever, chills, and weakness one day after the second dose and 30 days after the first dose of the Hep B vaccine. Id. at 2. The child presented with tachycardia, low blood pressure (84/50), liver enlargement, and rash. Id. Echocardiography showed "moderately diminished biventricular function" with decreased "ejection fraction of 30%." Id. Myocardial muscle biopsy revealed "eosinophilic infiltration in the presence of myocyte necrosis." Id. There was no peripheral eosinophilia. Id. Skin biopsy showed "perivascular lymphocytic infiltrates." Id. Diagnosis was hypersensitivity vasculitis. Id. Diagnostic workup did not reveal an infectious etiology. Id. Treatment with steroids was instituted with improvement noted within one week and normalization of heart function by eight weeks. Id.

The second case involved a 14-year-old boy who received the meningococcal conjugate vaccine. Resp. Ex. D at 2. Prior to vaccination, the patient had group A streptococcal pharyngitis treated with a 10-day course of penicillin.[45] Id. The day of vaccination, the patient

[44] The "viral panel" referred to by Dr. Ringel appears to be the "the myocarditis panel" performed on T.B.D. referred to in the medical records that returned negative results.

[45] The authors noted that a delayed hypersensitivity reaction to penicillin could not be excluded. Resp. Ex. D at 4.

developed swelling and stiffness in the fingers which resolved, followed by headache, fever, and myalgias. Id. Ten days after vaccination, he had difficulty breathing, chest pain, and heart palpitations. Id. He presented to the emergency department 13 days after vaccination where echocardiography showed "decreased biventricular function" and "small pericardial effusion." Id. at 2-3. Myocardial biopsy revealed "eosinophilic infiltration with areas of eosinophilic degranulation." Id. at 3. Cardiac enzyme levels were elevated. Id. There was mild peripheral eosinophilia at presentation. Id. Workup for other causes was negative. Id. Steroids were administered with improvement in cardiac function. Id. The child completely recovered within three months. Id.

The literature review by Barton et al. identified 269 cases of myocarditis/pericarditis after vaccinations, dominated by smallpox cases (251 cases). Resp. Ex. D at 3. There were 18 cases of myocarditis associated with other vaccines. Id. Nine were associated with the influenza vaccine, two occurred after the Hep B vaccine, two after DPT vaccination, one after multiple vaccines (DTaP, Hep A, and meningococcal conjugate vaccine), with the remainder following less common vaccines such as yellow fever, cholera, and anticatarrh. Id. Onset ranged from one to 30 days post-vaccination. Id. at 3, 3 tbl.2. Biopsy proven eosinophilic myocarditis was documented in "some cases." Id. at 3. The authors noted that these "reports of cardiac injury associated with common vaccines, such as diphtheria, tetanus," and others, "support an immune-mediated hypersensitivity mechanism." Id. at 4. They noted, however, that "patients with eosinophilic myocarditis have mildly elevated or normal eosinophilic counts" (referencing peripheral eosinophilia levels). Id. Cardiac manifestations may "occur as early as [one to four] days postvaccination." Id.

Barton et al. noted that potential mechanisms of "myocardial injury include autoimmune myocardial damage and type III hypersensitivity immune complex-mediation reaction." Resp. Ex. D at 4. The authors explained that while biopsies are invaluable in reaching a diagnosis, obtaining such specimens is challenging, "especially in children," likely resulting in "under-diagnosis." Id.

Another paper by Dettmeyer et al.,[46] described four children with sudden infant death syndrome ("SIDS") who had post-mortem evidence of coxsackie viral associated myocarditis. Resp. Ex. J at 1. Dr. Ringel cited this paper in support of his opinion that there may have been a viral cause of T.B.D.'s myocarditis. Resp. Ex. F at 4. The authors noted that enteroviruses, like the coxsackie virus, are common causes of myocarditis. Resp. Ex. J at 1. The autopsies of four children (eight to 40 weeks of age) who died of SIDS showed immunohistological evidence of inflammation suggestive of enteroviral myocarditis. Id. at 6. Of importance here, the authors identified myocarditis/inflammation as a cause of lethal cardiac arrhythmias. Id. at 6-7.

Hypersensitivity myocarditis has also been reported after the COVID-19 mRNA vaccination, as evidenced by medical literature filed by Respondent. One paper described three

[46] Reinhard Dettmeyer et al., Coxsackie B3 Myocarditis in 4 Cases of Suspected Sudden Infant Death Syndrome: Diagnosis by Immunohistochemical and Molecular-Pathologic Investigations, 198 Pathology Rsch. & Practice 689 (2002).

patients with severe post-vaccination myocarditis with biopsy evidence of myocardial inflammatory infiltrates (degranulated eosinophils). Resp. Ex. L at 1, 6.[47] All three patients had chest pain and troponin I elevation within two weeks of vaccination. Id. at 3. One patient had syncopal events due to an abnormal junctional rhythm with an absence of atrial electrical activity. Id. at 4. The other two patients had severely decreased myocardial contractility with decreased ejection fractions (equal to or less than 35%). Id. at 2. Cardiac magnetic resonance ("CMR") showed diffuse inflammation, myocardial edema, and abnormal hyperintense signals in the septum, inferior interventricular junction, and anterolateral wall. Id. at 5 fig.2, 6 fig.3. Cardiac muscle biopsies showed inflammatory infiltrates, primarily degranulated eosinophils. Id. at 6. Myocardial biopsy in a patient with a junctional rhythm and no atrial activity showed electrical conduction tissue (Purkinje fibers) areas that were "infiltrated and damaged by eosinophils." Id. at 7. All three patients responded well to steroid therapy. Id.

Two additional relevant papers filed by Respondent include a letter to the editor by Kounis and Mplani,[48] citing a recent study reporting histology findings in biopsies of adults and children with myocarditis.[49] Resp. Ex. N at 1. Biopsies revealed that the most common histology of myocarditis was lymphocytic, which constituted most cases (82.6%), with eosinophilic cases considerably rarer (3.5%). Id. The authors explained that hypersensitivity myocarditis is caused by an allergic or hypersensitivity reaction and eosinophilic infiltration of heart muscle. Id. Of note, one-third of patients with eosinophilic hypersensitive myocarditis did not have peripheral eosinophilia. Id.

The second article is by Frustaci et al.[50] and described immunomodulating /immunosuppressive therapy for immune-mediated forms of myocarditis. Resp. Ex. O at 1-2. The authors noted that the clinical manifestations of the illness ranges from mild to severe, and include "life-threatening arrhythmias and sudden death." Id. at 1. "[T]oxins (drugs, vaccines, toxic agents)" comprise a known cause of myocarditis. Id. The authors explain that diagnosis may be "challenging" due to the "wide variability in clinical presentation and unpredictable course." Id.

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[47] Andrea Frustaci et al., Hypersensitivity Myocarditis After COVID-19 mRNA Vaccination, 11 J. Clinical Med. 1660 (2022).

[48] Nicholas G. Kounis & Virginia Mplani, The Uncertain Nature of Myocarditis Classification and the Challenging Case of Eosinophilic Myocarditis Leading to Heart Failure and Transplantation, 73 Cardiovascular Pathology 107682 (2024).

[49] The cited study was not filed. See ALP Caforio et al, Endomyocardial Biopsy: Safety and Prognostic Utility in Paediatric and Adult Myocarditis in the Euopena Society of Cardiology EURObservational Research Programme Cardiomyopathy and Myocarditis Long-Term Registry, 45 Eur. Heart J. 2548 (2024).

[50] Andrea Frustaci et al., Immunomodulating and Immunosuppressive Therapy for Virus-Negative Immune-Mediated Myocarditis, 12 Biomeds. 1565 (2024).

4. Respondent's Expert, Dr. Peter M. Bingham[51]

a. Background and Qualifications

Dr. Bingham is a board-certified pediatric neurologist and clinical researcher. Resp. Ex. G at 1. He received his M.D. from Columbia College of Physicians & Surgeons in New York. Resp. Ex. H at 1. He completed a pediatrics residency, a neurology residency, and a fellowship in neuromuscular diseases at the Children's Hospital of Philadelphia. Id. Dr. Bingham is currently a Professor of Neurology & Pediatrics at the University of Vermont. Id. He has authored or co-authored numerous publications primarily in neurology. Id. at 3-7. Dr. Bingham has 29 years of "postresidency experience in general child neurology" and has "diagnosed and managed approximately 500 cases of encephalopathy in infants and newborns." Resp. Ex. G at 1.

b. Opinions

In his initial expert report, Dr. Bingham addressed Petitioners' Table claim of encephalopathy following the DTaP vaccination. Resp. Ex. G at 4-7.

Regarding the question of whether T.B.D. had encephalopathy, defined as "a significantly decreased level of consciousness" lasting at least 24 hours, not attributable to a postictal state from seizure or caused by medication, Dr. Bingham was not able to determine what degree of encephalopathy was caused by cardiac arrest versus what was caused by the sedatives/medications T.B.D. was given post-arrest. Resp. Ex. G at 4. He explained that after cardiac arrest, an infant must be sedated while they are mechanically ventilated, and that this sedation "obscures the neurologic examination." Id. at 4-5. Thus, he opined there is no way to discern the duration of encephalopathy post-vaccination because T.B.D. was sedated and ventilated during the first few days of his hospitalization. Id. at 5.

Next, Dr. Bingham addressed the question of whether the family members pre-hospital observations are consistent with encephalopathy after vaccination but before cardiac arrest. Resp. Ex. G at 6. He did not agree that these observations constituted evidence of encephalopathy. Id. He specifically disagreed that periods of "increased sleep interspersed with periods of being irritable and fussy" were consistent with encephalopathy. Resp. Ex. I at 1. He cited portions of the medical records which described T.B.D.'s behavior prior to his arrest as reported by the family on arrival to the hospital, noting that T.B.D. was able to engage in breastfeeding grab his mother's shirt before he became unresponsive. Id. at 2 (citing Pet. Ex. 8 at 306; Pet. Ex. 7 at 11). Dr. Bingham opined that these behaviors are "inconsistent with acute encephalopathy." Id. (internal quotations omitted).

Instead, Dr. Bingham believed that the onset of decreased alertness began "at the moment of cardiac arrest." Resp. Ex. I at 2; Resp. Ex. G at 6. The endpoint of decreased alertness is difficult to discern due to sedation, as described above, but he opined "it ended at some point

[51] Dr. Bingham submitted three expert reports. Resp. Exs. G, I, Q.

before [T.B.D.] was extubated [two to three] days later." Resp. Ex. G at 6. He opined that T.B.D.'s "acute encephalopathy was due to cardiac arrest" and its "aftermath—a hypoxic ischemic stress to the brain." Id. Dr. Bingham attributed the acute encephalopathy to cardiac arrest, opining this was inconsistent with the Vaccine Injury Table definition. Id.

Dr. Bingham did not offer an opinion about whether T.B.D.'s cardiac arrest was caused by vaccination. See Resp. Ex. G at 5, 7. But he did agree that T.B.D.s cardiac arrest was "the cause of any degree of encephalopathy" during the first week of hospitalization in December 2015. Id. at 7.

Further, although Dr. Bingham agreed T.B.D. experienced acute encephalopathy due to cardiac arrest, he disagreed that T.B.D. had chronic encephalopathy. Resp. Ex. G at 7. After discharge from the hospital, moving forward to February 2016, T.B.D. had decreased tone and "mild motor gross delay noted at 15 months of age. Id. Dr. Bingham opined that these abnormalities were "not clearly related to his encephalopathy" that began on December 15, 2015. Id. Instead, based on the EEGs and MRIs, Dr. Bingham opined that these problems were "probably more related to brain pathology that preceded" his cardiac arrest on December 15. Id.

Dr. Bingham disagreed with Dr. Chang's opinion that the cardiac arrest and encephalopathy were independent and separate processes. Resp. Ex. G at 7.

Dr. Bingham also disagreed with Dr. Lubens' dismissal of the cardiac arrest as the cause of encephalopathy, noting that the arrest was "associated with significant changes in blood chemistry (acidemia) and changes on [MRI] that certainly resulted from ischemia/circulatory failure." Resp. Ex. I at 2. MRI showed "hyperintensities within [T.B.D.'s] right insular region" of the brain that the consulting neurologist attributed to cardiac arrest. Id. He further explained that the MRI done after cardiac arrest demonstrated two independent abnormalities. Resp. Ex. Q at 2. The first was an abnormality of the temporal lobe, caused by the cardiac arrest. Id. This abnormality is "also reflected in EEG changes." Id. These acute MRI findings affecting the right and left temporal lobe, attributable to the cardiac arrest, are "plausible markers of brain injury" and account for T.B.D.'s "problems staying on task and [] behavioral issues." Id. at 3.

The second abnormality seen on MRI was caused by an event that Dr. Bingham opined was more remote and occurred over one month before the cardiac arrest, which he described as "periventricular white matter signal change." Resp. Ex. Q at 2. This second abnormality was caused by "pre- or peri-natal stress to the brain." Id. Dr. Bingham suggested that T.B.D.'s gross motor delay may have been associated with this pre-existing brain abnormality, and it was likely temporary, and improved with physical therapy. Id. at 3.

To the extent that Dr. Luben opined that the periventricular while matter changes seen on MRI represent inflammation attributable to vaccination, Dr. Bingham disagreed. Resp. Ex. Q at 3-4. If this were the case, Dr. Bingham stated there would have been evidence of inflammation in the spinal fluid. Id. at 4. However, he opined that T.B.D.'s doctors did not test his spinal fluid, since they did not suspect encephalitis. Id. at 4-5. Further, Dr. Bingham opined T.B.D. did not have characteristic MRI findings of encephalitis, fever, or EEG changes consistent with encephalitis. Id. at 5.

Lastly, Dr. Bingham agreed with T.B.D.'s treating physician Dr. Ramos-Platt's interpretations of T.B.D.'s diagnostic studies (MRI and EEG) and opinions.[52] Resp. Ex. Q at 5. Specifically, Dr. Bingham agreed that T.B.D. had two insults to the brain, as described above, one caused by the cardiac arrest and the other more remote that probably occurred during the perinatal period. Id. at 6.

In conclusion, Dr. Bingham summarized his opinions as follows:

> (1) The deduction of a state of encephalopathy independent of the medicines [T.B.D.] received is tentative in that [he] was sedated within minutes of his initial arrest.
> (2) The cause of [T.B.D.'s] alleged encephalopathy, if it did last more than 24 hours, was his cardiac arrest; there is no clear way to separate arrest-induced encephalopathy from the sedatives he received . . . to facilitate mechanical ventilation.
> (3) Dr. Chang does not explain why the alleged encephalopathy was a phenomenon independent of his cardiac arrest.
> (4) There is no evidence of chronic or ongoing encephalopathy.

Resp. Ex. G at 7-8.

IV. LEGAL STANDARDS

A. Standards for Adjudication

The Vaccine Act was established to compensate vaccine-related injuries and deaths. § 10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as

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[52] Dr. Bingham quoted Dr. Ramos-Platt's opinions:

> Neurologic exam[ination] significant for diffuse hypertonia with cortical fisting and [bilateral lower extremity] hyper-reflexia. MRI brain with restricted diffusion in the insular region of temporal lobe, as well as periventricular white matter changes. He most likely had two separate insults to the brain resulting in his MRI findings. The restricted diffusion of the insular region is most likely acute/subacute, likely to have happened within the last 14 days. This is most likely a result of the cardiac arrest he suffered on admission. Given the location of the restricted diffusion, he is at increased risk of developing temporal lobe seizures. The periventricular white matter changes are most likely remote and occurred during the perinatal period. These findings could be due to his premature status, or due to periods of hypoxia secondary to his underlying cardiac condition. The periventricular white matter involvement is likely responsible for his increased tone.

Resp. Ex. Q at 6 (quoting Pet. Ex. 8 at 288).

a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" Rooks v. Sec'y of Health & Hum. Servs., 35 Fed. Cl. 1, 7 (1996) (quoting H.R. Rep. No. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

Petitioners' burden of proof is by a preponderance of the evidence. § 13(a)(1); see also de Bazan v. Sec'y of Health & Hum. Servs., 539 F.3d 1347, 1351 (Fed. Cir. 2008) (noting § 13(a)(1) applies to both Table and causation-in-fact claims).

Testimony that merely expresses the possibility—not the probability—is insufficient, by itself, to substantiate a claim that such an injury occurred. See Waterman v. Sec'y of Health & Hum. Servs., 123 Fed. Cl. 564, 573-74 (2015) (denying Petitioner's motion for review and noting that a possible causal link was not sufficient to meet the preponderance standard). The Federal Circuit has made clear that the mere possibility of a link between a vaccination and a petitioner's injury is not sufficient to satisfy the preponderance standard. Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1322 (Fed. Cir. 2010) (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and the injury" does not equate to proof of causation by a preponderance of the evidence); Boatmon v. Sec'y of Health & Hum. Servs., 941 F.3d 1351, 1359-60 (Fed. Cir. 2019). While certainty is by no means required, a possible mechanism does not rise to the level of preponderance. Moberly, 592 F.3d at 1322; see also de Bazan, 539 F.3d at 1351.

B. Factual Issues

Petitioners must prove, by a preponderance of the evidence, the factual circumstances surrounding their claim. § 13(a)(1)(A). To resolve factual issues, the special master must weigh the evidence presented, which may include contemporaneous medical records and testimony. See Burns v. Sec'y of Health & Hum. Servs., 3 F.3d 415, 417 (Fed. Cir. 1993) (explaining that a special master must decide what weight to give evidence including oral testimony and contemporaneous medical records).

Medical records, specifically contemporaneous medical records, are presumed to be accurate and generally "warrant consideration as trustworthy evidence." Cucuras v. Sec'y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). But see Kirby v. Sec'y of Health & Hum. Servs., 997 F.3d 1378, 1382 (Fed. Cir. 2021) (rejecting the presumption that "medical records are accurate and complete as to all the patient's physical conditions"); Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 538 (2011) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance." (quoting Murphy v. Sec'y of Health & Hum. Servs., 23 Cl. Ct. 726, 733 (1991), aff'd per curiam, 968 F.2d 1226 (Fed. Cir. 1992))), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 503 F. App'x 952 (Fed. Cir. 2013). The weight afforded to contemporaneous records is due to the fact that they "contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium." Id. To overcome the presumptive accuracy of medical records, a petitioner may present testimony which is "consistent, clear, cogent, and compelling." Sanchez v. Sec'y of Health & Hum. Servs., No. 11-

685V, 2013 WL 1880825, at *3 (Fed. Cl. Spec. Mstr. Apr. 10, 2013) (citing Blutstein v. Sec'y of Health & Hum. Servs., No. 90-2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)), mot. for rev. denied, 142 Fed. Cl. 247 (2019), vacated on other grounds & remanded, 809 F. App'x 843 (Fed Cir. 2020).

There are situations in which compelling testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. Campbell v. Sec'y of Health & Hum. Servs., 69 Fed. Cl. 775, 779 (2006) ("[L]ike any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking."); Lowrie v. Sec'y of Health & Hum. Servs., No. 03-1585V, 2005 WL 6117475, at *19 (Fed. Cl. Spec. Mstr. Dec. 12, 2005) ("[W]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent." (quoting Murphy, 23 Cl. Ct. at 733). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. Andreu v. Sec'y of Health & Hum. Servs., 569 F.3d 1367, 1379 (Fed. Cir. 2009); Bradley v. Sec'y of Health & Hum. Servs., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

Despite the weight afforded medical records, special masters are not bound rigidly by those records in determining onset of a vaccinee's symptoms. Valenzuela v. Sec'y of Health & Hum. Servs., No. 90-1002V, 1991 WL 182241, at *3 (Fed. Cl. Spec. Mstr. Aug. 30, 1991); see also Eng v. Sec'y of Health & Hum. Servs., No. 90-1754V, 1994 WL 67704, at *3 (Fed. Cl. Spec. Mstr. Feb. 18, 1994) (Section 13(b)(2) "must be construed so as to give effect also to § 13(b)(1) which directs the special master or court to consider the medical records (reports, diagnosis, conclusions, medical judgment, test reports, etc.), but does not require the special master or court to be bound by them").

C. Table Claim

To receive compensation through the Program, Petitioners must prove either (1) that T.B.D. suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that he received, or (2) that T.B.D. suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); Capizzano v. Sec'y of Health & Hum. Servs., 440 F.3d 1317, 1319-20 (Fed. Cir. 2006). Here, Petitioners allege both a Table injury and a causation-in-fact injury. Pet. Br. at 1. As acknowledged by Respondent, the alleged Table injury is based on the question of whether T.B.D. suffered an "encephalopathy." See Resp. Rept. at 15; see also 42 C.F.R § 100.3(a)(II)(B).

The Vaccine Injury Table provides that encephalopathy is a recognized injury for the DTaP vaccination if the first symptom or manifestation of onset occurs within 72 hours of vaccine administration. 42 C.F.R. § 100.3(a)(II)(B). A "vaccine recipient shall be considered to have suffered an encephalopathy if an injury meeting the [] description of acute encephalopathy occurs within the applicable time period and results in a chronic encephalopathy." Id. at § 100.3(c)(2).

Acute encephalopathy, for children less than 18 months of age, that presents without a seizure "is indicated by a significantly decreased level of consciousness that lasts at least 24 hours." 42 C.F.R. § 100.3(c)(2)(i)(A)(1). Acute encephalopathy following a seizure "is demonstrated by a significantly decreased level of consciousness that lasts at least 24 hours and cannot be attributed to a postictal state—from a seizure or a medication." Id. at § 100.3(c)(2)(i)(A)(2). "Clinical features" that do not in themselves "demonstrate an acute encephalopathy or a significant change in either mental status or level of consciousness" include "sleepiness, irritability (fussiness), high-pitched and unusual screaming, poor feeding, persistent inconsolable crying, bulging fontanelle, or symptoms of dementia." Id. at § 100.3(c)(2)(i)(C). Moreover, "[s]eizures in themselves are not sufficient to constitute a diagnosis of encephalopathy and in the absence of other evidence of an acute encephalopathy, seizures shall not be viewed as the first symptom or manifestation of an acute encephalopathy." Id. at § 100.3(c)(2)(i)(D).

In addition to proving an acute encephalopathy, Petitioners must also prove that the encephalopathy "persist[ed] for at least [six] months from the first symptom or manifestation of onset . . . of an acute encephalopathy." 42 C.F.R. § 100.3(d)(1)(i). Thus, Petitioner must prove a "chronic encephalopathy" which "occurs when a change in mental or neurological status" lasts at least six months. Id.

The last issue to resolve to determine if this is a Table Claim is whether there are applicable exclusionary criteria that explain T.B.D.'s encephalopathy. Exclusionary criteria for encephalopathy indicate that

> [r]egardless of whether or not the specific cause of the underlying event, systemic disease or acute event (including an infectious organism) is known, an encephalopathy shall not be considered to be a condition set forth in the Table if it is shown that the encephalopathy was caused by:
>
> (A) An underlying condition or systemic disease shown to be unrelated to the vaccine (such as malignancy, structural lesion, psychiatric illness, dementia, genetic disorder, prenatal or perinatal central nervous system (CNS) injury); or
>
> (B) An acute event shown to be unrelated to the vaccine such as head trauma, stroke, transient ischemic attack, complicated migraine, drug use (illicit or prescribed) or an infectious disease.

42 C.F.R. § 100.3(c)(2)(ii).

D. Causation-in-Fact Claim

Petitioners also allege a causation-in-fact claim. To prevail on this claim, Petitioners must prove that a vaccine T.B.D. received caused his injury. To do so, Petitioners must establish, by preponderant evidence: (1) a medical theory causally connecting the vaccine and T.B.D.'s injury ("Althen Prong One"); (2) a logical sequence of cause and effect showing that the vaccine was the reason for T.B.D.'s injury ("Althen Prong Two"); and (3) a showing of a proximate temporal relationship between the vaccine and T.B.D.'s injury ("Althen Prong

Three"). Althen, 418 F.3d at 1278; § 13(a)(1).

The causation theory must relate to the injury alleged. Petitioners must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the explanation need only be "legally probable, not medically or scientifically certain." Knudsen v. Sec'y of Health & Hum. Servs., 35 F.3d 543, 548-49 (Fed. Cir. 1994). Petitioners cannot establish entitlement to compensation based solely on their assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). In determining whether Petitioners are entitled to compensation, the special master shall consider all material in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 13(b)(1)(A). The undersigned must weigh the submitted evidence and the testimony of the parties' proffered experts and rule in Petitioner's favor when the evidence weighs in her favor. See Moberly, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence."); Althen, 418 F.3d at 1280 (noting that "close calls" are resolved in Petitioner's favor).

V. ANALYSIS OF VACCINE INJURY TABLE CLAIM

The undersigned finds that Petitioners have shown by preponderant evidence that T.B.D. suffered a Table encephalopathy injury after DTaP vaccination. Further, the undersigned finds that T.B.D.'s cardiac arrest does not constitute an exclusion to a Table encephalopathy based on the specific facts and circumstances presented here.

A. Table Claim

Relevant to the Table claim, the undersigned finds that T.B.D. meets the Table definitions for both acute and chronic encephalopathy, as described below.

1. T.B.D. Suffered Acute Encephalopathy Within 72 Hours of His DTaP Vaccination That Lasted at Least 24 Hours

Pursuant to the Vaccine Injury Table, an acute encephalopathy in a two-month-old child who presents without evidence of seizure[53] is a "significantly decreased level of consciousness that lasts at least 24 hours." 42 C.F.R. § 100.3(c)(2)(i)(A)(1). T.B.D.'s clinical course, as demonstrated by the medical records and expert testimony of Dr. Bingham, show that this definition is met.

On December 15, 2015, T.B.D. received his DTaP vaccination. The next day, December 16 at approximately 1:55 pm, he had a cardiac arrest. When emergency responders arrived at the

[53] Dr. Bingham opined that T.B.D. had "no overt seizures" during his initial hospitalization post-cardiac arrest. Resp. Ex. G at 2. There is no indication in the record that T.B.D.'s acute encephalopathy was due to a seizure or seizures.

scene, T.B.D. had agonal respirations and a slow pulse. CPR was initiated with chest compressions and bag-valve mask. On arrival to the hospital, T.B.D. was "unresponsive" and had "no cardiac activity." Pet. Ex. 7 at 11-12. T.B.D. was intubated and noted to have "minimal spontaneous movements" and "pinpoint pupils" that were "minimally responsive." Pet. Ex. 8 at 8. T.B.D. remained sedated and intubated for two days until he was extubated on December 18.[54]

In his expert reports, Dr. Bingham explained that T.B.D.'s initial blood tests revealed "significant metabolic acidosis consistent with hypoxia and or ischemia as the cause of encephalopathy." Resp. Ex. G at 2. Dr. Bingham opined that T.B.D.'s encephalopathy was caused by his cardiac arrest but also influenced by the sedatives given while he was "being mechanically ventilated for several days." Id. at 4. He further opined that it was impossible to determine how much of T.B.D. decreased level of consciousness was caused by the cardiac arrest as compared to the sedatives he was given because the sedation required for mechanical ventilation "obscures the neurological examination." Id. at 5. Dr. Bingham concluded by stating that "T.B.D.'s acute encephalopathy [] began at the moment of his cardiac arrest[] and persisted until after he was extubated [two to three] days later." Resp. Ex. I at 2.

Although it was difficult to obtain a complete neurological examination on T.B.D. due to the sedatives required for his mechanical ventilation for two to three days, this does not defeat Petitioners' Table claim. When T.B.D. was first assessed by the emergency responders, he had agonal respirations and there were brief periods of time he was without pulse or respirations. On arrival to the hospital, T.B.D. was unresponsive. There is no question that prior to being sedated, T.B.D. was unresponsive, therefore meeting the definition of "significantly decreased level of consciousness." 42 C.F.R. § 100.3(c)(2)(i)(A)(1). After sedation and intubation and once he was transferred to CHLA, physical examination revealed "minimal spontaneous movements" and "pinpoint pupils." Pet. Ex. 8 at 8. These observations also meet the definition of "significantly decreased level of consciousness." Dr. Bingham explains it is not possible to discern what degree T.B.D.'s decreased level of consciousness was caused by sedation versus encephalopathy; however, Dr. Bingham does not assert that sedation was the sole reason for T.B.D.'s clinical features of acute encephalopathy.

Dr. Lubens dates the onset of T.B.D.'s encephalopathy to before cardiac arrest based on statements by family members who described T.B.D. as lethargic, detached, not moving, unreactive, and crying inconsolably after vaccination and before his cardiac arrest. The undersigned disagrees and finds Dr. Bingham more persuasive. T.B.D.'s described post-vaccination and pre-cardiac arrest behaviors do not rise to the level required by the Vaccine Injury Table to demonstrate an acute encephalopathy. See 42 C.F.R. § 100.3(c)(2)(i)(C) (stating sleepiness, irritability or fussiness, or persistent inconsolable crying do not themselves "demonstrate an acute encephalopathy"). Thus, the undersigned finds that T.B.D. did not exhibit clinical features of acute encephalopathy until after cardiac arrest.

[54] T.B.D. was later sedated and re-intubated on December 21 for diagnostic tests. Pet. Ex. 8 at 43.

Moreover, Respondent appears to concede that T.B.D. had an acute encephalopathy. Respondent's brief states

> T.B.D.'s cardiac arrest on December 16, 2015[] is well documented in the medical records. The neurological effects of T.B.D.'s arrest are also well documented. On December 21, 2016, six days after vaccination, an MRI of T.B.D.'s brain revealed "restricted diffusion along the posterior aspects of the insular cortex and posterior temporal lobe on right," which was "suggestive of infarction/hypoperfusion injury." Pet. Ex. 5 at 12-13. A subsequent neurology consult noted:
>
> > [T.B.D.] most likely had two separate insults to the brain resulting in his MRI findings. The restricted diffusion of the insular region is most likely acute/subacute, likely to have happened within the last 14 days. ***This is most likely a result of the cardiac arrest he suffered on admission.*** The peri-ventricular white matter changes are most likely remote and occurred during the perinatal period. These findings could be due to his premature status, or due to periods of hypoxia secondary to his underlying cardiac condition. The peri-ventricular white matter involvement is likely responsible for his increased tone.
>
> Pet. Ex. 8 at 135 (emphasis added [by Respondent]). To borrow the language used by Dr. Bingham, "the cause of [T.B.D.'s] alleged encephalopathy was, if it did last more than 24 hours, due to his cardiac arrest." Resp. Ex. G at 7. Dr. Bingham also noted T.B.D.'s cardiac arrest was associated with a significant change in T.B.D.'s blood chemistry (acidemia) and changes in his neuroimaging (described above), which "certainly resulted from ischemia/circulatory failure." Resp. Ex. I at 2.
>
> Nothing in T.B.D.'s records or expert reports disputes the findings of Dr. Bingham or T.B.D.'s treating neurologist. As such, T.B.D.'s alleged encephalopathy was due to cardiac arrest and its immediate aftermath—hypoxic ischemic stress to the brain. Resp. Ex. I at 2.

Resp. Response at 20.

In summary, T.B.D. had the onset of significantly decreased level of consciousness within 72 hours of vaccination. T.B.D.'s decreased level of consciousness lasted for at least 24 hours. Therefore, the undersigned finds that Petitioners have shown by preponderant evidence that T.B.D. suffered an acute encephalopathy within 72 hours of his DTaP vaccination as defined by the Vaccine Injury Table.

2. T.B.D.'s Acute Encephalopathy Resulted in a Chronic Encephalopathy That Persisted for At Least Six Months

To meet the Table severity requirement, the evidence must show that T.B.D.'s acute encephalopathy resulted in chronic encephalopathy that lasted at least six months. 42 C.F.R. § 100.3(d)(1)(i) ("A chronic encephalopathy occurs when a change in mental or neurologic status, first manifested during the applicable Table time period as an acute encephalopathy [], persists for at least [six] months from . . . the manifestation of onset.").

T.B.D.'s cardiac arrest occurred on December 16, 2015. He had clinical features consistent with acute encephalopathy after cardiac arrest and during his hospitalization. His diagnostic testing was also consistent with brain injury, with MRI showing areas of "infarction/hypoperfusion injury in the setting of recent cardiac arrest."[55] Pet. Ex. 8 at 134. T.B.D.'s neurologist Dr. Ramos-Platt opined that T.B.D. had an insult to his brain, the abnormality in the temporal lobe and insula, that was "most likely a result of his cardiac arrest." Id. at 286-87.[56] His EEG was also abnormal.

After discharge from the hospital, T.B.D. had follow-up appointments with his health care providers. Relevant to chronic encephalopathy, T.B.D. had a developmental evaluation on February 1, 2016, which documented poor head and trunk control. The evaluation concluded that T.B.D. had "delays in gross motor skills and adaptive behavior . . . [with] limited movement repertoire and work against gravity." Pet. Ex. 11 at 5. T.B.D.'s neurological follow-up on April 4, 2016 noted decreased abnormal tone in the left upper extremity and that T.B.D. was unable to get into a sitting position. Pet. Ex. 5 at 11. Examination by Dr. Hartstein on April 25, 2016 again showed decreased truncal tone. Id. at 18. Dr. Hartstein assessed T.B.D. with mild gross motor delay. Id. On April 25, T.B.D.'s active problem list identified a chronic hypoxic-ischemic brain injury with an onset date of December 16, 2015. Id. at 16.

In the summer and fall of 2016, T.B.D. continued to have physical therapy. Physical therapy evaluation on July 15, 2016 documented that T.B.D. had "decreased truck and lower extremity strength required for independent mobility." Pet. Ex. 13 at 2. Diagnosis was "[d]evelopmental delay" and the evaluation noted T.B.D. "experienced mild hypoxia on the left temporal lobe due to cardiac arrest." Id. at 1. This note evidences that T.B.D. had an abnormal neurological status due to decreased trunk and lower extremity strength, evidence of a persistent change in his neurological condition for over six months.

[55] Periventricular while matter changes were also seen, but were not attributed to the cardiac arrest, but "suggestive of prior white matter injury." Pet. Ex. 8 at 134-35.

[56] Again, the periventricular white matter changes were discussed, and attributed to the perinatal period. Pet. Ex. 8 at 286. Dr. Ramos-Platt opined the periventricular white matter changes were "likely responsible for [T.B.D.'s] increased tone [] evidence even prior to the arrest as [Petitioners] noted the persistent fisting prior to the arrest." Id.

Further, chronic encephalopathy can be present even when there is improvement over time. In S.D., the undersigned found preponderant evidence of chronic encephalopathy when the vaccinee had normal gross motor skills, balance, and gait within four months but continued to demonstrate neurological abnormalities on MRI and EEG. S.D. ex rel. Deters v. Sec'y of Health & Hum. Servs., No. 19-459V, 2024 WL 3950726, at *19 (Fed. Cl. Spec. Mstr. July 30, 2024); see also Ramsey v. Sec'y of Health & Hum. Servs., No. 21-1486V, 2023 WL 2823403, at *9 (Fed. Cl. Spec. Mstr. Apr. 7, 2023) (finding no chronic encephalopathy when a vaccinee returned to his "neurological baseline" within six months). Here, T.B.D. continued to have neurological abnormalities in the form of his mild gross developmental delay in the six months following his acute encephalopathy.

Thus, the undersigned finds T.B.D. had an acute encephalopathy that was followed by a chronic change in his neurologic status (developmental delay) that persisted for more than six months.

As discussed above, Respondent concedes in his brief that T.B.D.'s cardiac arrest caused a hypoxic brain injury.[57] Resp. Response at 20. However, Respondent disagrees that T.B.D. suffered a chronic encephalopathy based on the opinions of their expert Dr. Bingham. Id. at 24.

Dr. Bingham contends that T.B.D.'s mild gross motor delay identified at 15 months of age was "not clearly related to his encephalopathy that began on December 15, 2015." Resp. Response at 25 (citing Pet. Ex. 5 at 18; Resp. Ex. G at 7). This position ignores the opinions of the treating physicians, specifically the notes explaining that T.B.D. had two brain abnormalities. One occurred before cardiac arrest and was consistent with periventricular white matter abnormalities seen on MRI. This abnormality was associated with increased tone and cortical fisting. It was not associated with T.B.D.'s other abnormal mental and neurological abnormalities which were caused by his cardiac arrest and hypoxic/ischemic injury. T.B.D.'s physicians did not attribute his decreased tone, decreased strength, delayed motor skills, or his other neurological injuries to periventricular white matter abnormalities. The undersigned finds the notes by the treating physicians to be more persuasive, especially since they were not prepared for purposes of litigation.[58]

> The opinions and views of the vaccinee's treating physicians are entitled to some weight. Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326 ("[M]edical records and medical opinion testimony are favored in vaccine cases."); see also L.M. ex rel. McClellan v. Sec'y of Health & Hum. Servs., No. 14-714V, 2019 WL 4072130, at *32 (Fed. Cl. Spec. Mstr. July 23, 2019) ("[I]t is reasonable to give a treater view reflected in the contemporaneous medical record (before thought of litigation has occurred) greater weight than a subsequent statement prepared specifically to support a Vaccine Act case.").

[57] Respondent concedes that T.B.D. had encephalopathy due to cardiac arrest but argues that the cardiac arrest was unrelated to vaccination.

[58] The undersigned also rejects the opinions of Dr. Lubens asserting that the periventricular white matter changes were caused by vaccination, as these opinions do not reflect the greater weight of the evidence established by the medical records and treating physician opinions.

Therefore, the undersigned finds that T.B.D. suffered an acute encephalopathy which resulted in chronic encephalopathy that persisted for at least six months, characterized by decreased tone, limitations in independent mobility, and his other neurological abnormalities consistent with his hypoxic/ischemic brain injury caused by cardiac arrest.

3. Applicable Exclusions

For the following reasons, the undersigned finds that there are no applicable exclusions to T.B.D.'s Table claim.

a. The Sedatives Administered to T.B.D. After His Cardiac Arrest Do Not Constitute an Acute Event (Drug Use) Under the Exclusionary Criteria for Encephalopathy

Drug use, including prescribed medication, only constitutes an exclusion if the encephalopathy is shown to be caused by the drug use. The plain language of the Vaccine Injury Table provides that "an encephalopathy shall not be considered to be a condition set forth in the Table if it is shown the encephalopathy was caused by . . . [a]n acute event shown to be unrelated to the vaccine such as . . . drug use (illicit or prescribed)." 42 C.F.R. § 100.3(c)(2)(ii)(B).

Here, there is no evidence that the sedatives given to T.B.D. after his cardiac arrest "caused" his encephalopathy. The sedatives may have made it difficult to ascertain T.B.D.'s neurological status or perform a neurological examination but there is no suggestion in the medical records, or by the treating physicians or experts, that the sedatives caused T.B.D. to suffer encephalopathy. This conclusion is supported by Respondent's expert, Dr. Bingham, when he states, "the need to sedate [infants] while they are mechanically ventilated obscures the neurologic examination." Resp. Ex. G at 5. Dr. Bingham did not opine that the sedative caused encephalopathy.

Respondent argues that the sedatives administered to T.B.D. after his cardiac arrest constituted "an acute event shown to be unrelated to the vaccine." Resp. Response at 19. Respondent asserts the sedatives "altered his consciousness" such that Petitioners cannot show that T.B.D.'s decreased level of consciousness lasted 24 hours. Id. But in advancing this argument, Respondent omits the critical language required to show that an exclusion is applicable. It must be "shown that the encephalopathy was caused by" the "acute event." 42 C.F.R. § 100.3(c)(2)(ii)(B). T.B.D.'s encephalopathy was not caused by an acute event of drug use (sedatives). The sedatives were given so that T.B.D. could be intubated and ventilated.

As such, Respondent asserts that the sedatives were a "confounding factor," preventing Petitioners from showing that T.B.D.'s decreased level of consciousness lasted 24 hours. Resp. Response at 19. This argument is also misguided. The provisions in the Table relating to exclusionary criteria do not reference "confounding factors." While it may have been difficult to assess T.B.D.'s level of consciousness due to the sedatives administered to him, that difficulty does not constitute an exclusionary criterion.

Further, accepting the Respondent's arguments would create an untenable result, where medications given to treat encephalopathy or its accompanying sequela could be argued to constitute an exclusion. For example, if encephalopathy is accompanied by seizures, and anti-seizure medication is given which has the side effect of sedating the infant, making it difficult to assess the child's level of consciousness, Respondent could assert that the anti-seizure drug use constituted an exclusion. Denying compensation for Table claims due to medications used to treat encephalopathic conditions was not intended by the wording of the applicable provisions. Instead, the exclusionary criteria contemplate underlying conditions or acute events that are shown to cause the encephalopathy at issue.

b. The Cardiac Arrest Suffered by T.B.D. Does Not Constitute an Acute Event Shown to Be Unrelated to the Vaccine Under the Exclusionary Criteria for Encephalopathy

The Vaccine Injury Table provides that encephalopathy "shall not be considered to be a condition set forth in the Table if it is shown that the encephalopathy was caused by . . . [a]n acute event shown to be unrelated to the vaccine such as head trauma, stroke, transient ischemic attack, complicated migraine, drug use (illicit or prescribed)[,] or an infectious disease." 42 C.F.R. § 100.3(c)(2)(ii)(B).

"Cardiac arrest" is not identified as an acute event that constitutes an exclusion. Therefore, it can be argued that because it is not specifically referenced, it is not an exclusionary event. However, the context of the provision suggests that acute events that are not specifically referenced can also act as exclusions if they are shown to be unrelated to the vaccine "such as" the events that are listed.

Head trauma is the first acute event listed. Head trauma "refers to any damage to the scalp, skull[,] or brain caused by injury." Sally Robertson, What is Head Trauma?, News Med. Life Scis., https://www.news-medical.net/health/What-is-Head-Trauma.aspx (last updated Dec. 21, 2022). Two examples illustrate the point. Head trauma caused by a fall on a playground, leading to loss of consciousness and encephalopathy would be "unrelated to the vaccine." However, head trauma caused after a child received a vaccine and then fainted, hitting his skull, and causing brain injury resulting in loss of consciousness and a change in mental status that lasted for at least 24 hours (acute encephalopathy), would be related to the vaccine.

Respondent concedes that T.B.D.'s "alleged encephalopathy was due to cardiac arrest and its immediate aftermath—hypoxic ischemic stress to the brain." Resp. Response at 20 (citing Resp. Ex. I at 2). This concession is based on the medical records. Id. Respondent's expert, Dr. Bingham, opined "the cause of [T.B.D.'s] alleged encephalopathy was, if it did last more than 24 hours, due to his cardiac arrest." Resp. Ex. G at 7. Dr. Bingham also noted T.B.D.'s cardiac arrest was associated with a significant change in T.B.D.'s blood chemistry and changes in his neuroimaging which "certainly resulted from ischemia/circulatory failure." Resp. Ex. I at 2.

Dr. Bingham's opinions are consistent with the medical records that note T.B.D. had a "hypoperfusion injury of the temporal lobe from cardiac arrest." Pet. Ex. 5 at 85. Thus, the undersigned finds that the cardiac arrest caused T.B.D.'s encephalopathy.

The next question is whether T.B.D.'s cardiac arrest was "shown to be unrelated to the vaccine." The undersigned finds that Respondent has not shown by preponderant evidence that T.B.D.'s cardiac arrest was unrelated to the vaccine.

T.B.D. underwent a thorough evaluation to establish the cause of his cardiac arrest, including genetic, cardiology, and neurological workup. T.B.D.'s cardiologist, Dr. Silka, documented that cardiac genetic screening did not reveal any mutation. Pet. Ex. 5 at 8 (noting Dr. Silka had "a long discussion with the family as testing continues with no apparent cause/basis for [T.B.D.'s] initial arrest"). By June 27, 2016, whole exome genome testing was complete and no abnormality was found. Pet. Ex. 10 at 19. On January 18, 2017, Dr. Silka documented that an "extensive work up [was] NON-diagnostic." Pet. Ex. 15 at 16 (emphasis in original). In May 2017, dilated cardiomyopathy secondary to MPS was ruled out. Pet. Ex. 5 at 85. At a follow-up visit with Dr. Silka in November 2020, five years after T.B.D.'s cardiac arrest, Dr. Silka reiterated that the event was "unexplained." Pet. Ex. 26 at 3. Therefore, although T.B.D. underwent diagnostic evaluations by all relevant specialties, no underlying condition was found to be the cause of his cardiac arrest. Further, there was no evidence of a "malignancy, structural lesion, psychiatric illness, [] prenatal or perinatal central nervous system injury" that caused his cardiac arrest. 42 C.F.R. § 100.3(c)(2)(ii)(A).

Moreover, no other event has been shown to be the cause of T.B.D.'s cardiac event. There is no evidence that T.B.D. had "head trauma, stroke, transient ischemic attack, complicated migraine, drug use[,] [] or infectious disease." 42 C.F.R. § 100.3(c)(2)(ii)(B).

The vaccination T.B.D. received on December 15, 2015 was suggested to be a cause of his cardiac arrest. On April 25, 2016, T.B.D.'s primary care physician, Dr. Hartstein, documented her conversation with T.B.D.'s parents about this issue, stating, "there's no way that I can say whether or not his arrest was associated with his vaccinations." Pet. Ex. 5 at 18. Dr. Hartstein also completed a VAERS report the following day, stating T.B.D.'s cardiac arrest followed vaccination. Pet. Ex. 9 at 1-3. Later that year, in October 2016, Dr. Hartstein wrote she was "uncertain of the etiology of [T.B.D.'s] cardiac arrest, but because it happened 24 hours after receiving his first vaccines, [she] cannot guarantee safety with subsequent vaccinations." Pet. Ex. 5 at 105. At T.B.D.'s 15-month visit, Dr. Hartstein concluded that certain vaccines, including DTaP were "contraindicated" for T.B.D. Id. at 98.

In June 2016, T.B.D.'s cardiologist, Dr. Silka, discussed "the possibility" that T.B.D.'s cardiac arrest was due to "an untoward/unanticipated reaction to the vaccine." Pet. Ex. 10 at 22. In 2017, Dr. Silka's records stated that Dr. Hartstein had discussed T.B.D.'s vaccinations with "CHLA immunology and the decision has been made to not proceed" with further vaccinations. Pet. Ex. 15 at 9.

Dr. Chang, Petitioners' expert cardiologist, opined that T.B.D.'s "suffered [] sudden cardiac arrest as a direct result of cardiac sequelae from his immunization." Pet. Ex. 23 at 4.

The "vaccination induced an inflammatory response," specifically "acute hypersensitivity myocarditis, an inflammatory condition of heart muscle." Id. He noted the lack of any other explanation, "especially given the negative findings of an extensive array of cardiac and genetic testing (with whole exome analysis) and other clinical tests (including a negative cardiomyopathy panel), and an essentially normal metabolic panel which ruled out other potential causes for [T.B.D.'s] cardiac arrest." Pet. Ex. 33 at 3.

Petitioner's pediatric neurology expert, Dr. Lubens, did not find evidence of "any underlying medical condition or disease that caused or contributed to T.B.D.'s encephalopathy."[59] Pet. Ex. 30 at 2. Respondent's expert pediatric neurologist, Dr. Bingham, opined that T.B.D.'s cardiac arrest caused his encephalopathy "experienced in the first week of hospitalization in December 2015." Resp. Ex. G at 7.

Respondent's cardiology expert, Dr. Ringel agrees that T.B.D.'s workup for "his sudden cardiac arrest was unrevealing." Resp. Ex. A at 4. He suggests two other explanations for T.B.D.'s cardiac arrest (aspiration or viral myocarditis), but he does not state that he holds these opinions to a standard of more likely than not.

In conclusion, a thorough review of the medical records show that no "underlying condition" or "acute event" was established to be the cause of T.B.D.'s cardiac arrest. Although Respondent argues that the "cardiac arrest" was an "acute event" shown to be unrelated to the vaccine, that argument fails when there is no preponderant evidence shown to be the cause of T.B.D.'s cardiac arrest other than vaccination.

Further, in Table claims, petitioners who show that their minor child received a vaccination listed on the Table (DTaP) and suffered an injury listed on the Table (encephalopathy) within the prescribed time period (72 hours) are afforded a presumption of causation. § 11(c)(1)(c)(i); see Pafford v. Sec'y of Health & Hum. Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). Here, Petitioners have shown that T.B.D. received a DTaP vaccine, suffered an acute encephalopathy that resulted in a chronic encephalopathy with sequalae that lasted for at least six months, and there has been no evidence of any exclusionary condition or acute event unrelated to vaccination.

For all these reasons, the undersigned finds that Petitioners have proven by preponderant evidence that they have satisfied the requirements for a Vaccine Injury Table claim for encephalopathy post-DTaP vaccination.

VI. ANALYSIS OF CAUSATION-IN-FACT CLAIM

In addition to their Table claim, and in the alternative, Petitioners assert a causation-in-fact claim, specifically that T.B.D.'s injuries were caused-in-fact by the vaccinations he received

[59] Dr. Lubens opined that T.B.D.'s encephalopathy began after vaccination when he exhibited "a decrease in alertness," described by family members as "lethargy," acting "strangely detached," and "not looking at anything." Pet. Ex. 32 at 1. He further opined that T.B.D.'s encephalopathy worsened after his arrest. Pet. Ex. 30 at 1.

on December 15, 2015. Pet. Br. at 23. The parties disagree about whether Petitioners have provided preponderant evidence of causation for all three Althen prongs. See Joint Submission at 2; Pet. Br. at 24-29; Resp. Response at 25-28, 31-38; Althen, 418 F.3d at 1280.

For the reasons explained below, the undersigned finds that the Petitioners have proved by preponderant evidence that the vaccinations at issue caused hypersensitivity myocarditis and cardiac arrythmia which resulted in T.B.D.'s cardiac arrest and hypoxic-ischemic brain injury.

A. Althen Prong One

Under Althen prong one, Petitioners must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. Andreu, 569 F.3d at 1375; Pafford, 451 F.3d at 1355-56. Petitioners' theory of causation need not be medically or scientifically certain, but it must be informed by a "sound and reliable" medical or scientific explanation. Boatmon, 941 F.3d at 1359; see also Knudsen, 35 F.3d at 548; Veryzer v. Sec'y of Health & Hum. Servs., 98 Fed. Cl. 214, 223 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both "relevant" and "reliable"). If a petitioner relies upon a medical opinion to support his theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. See Broekelschen v. Sec'y of Health & Hum. Servs., 618 F.3d 1339, 1347 (Fed. Cir. 2010) ("The special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories."); Perreira v. Sec'y of Health & Hum. Servs., 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) (stating that an "expert opinion is no better than the soundness of the reasons supporting it" (citing Fehrs v. United States, 620 F.2d 255, 265 (Ct. Cl. 1980))).

Lack of information about a specific mechanism to prove that a theory is sound and reliable by preponderant evidence does not preclude Petitioners from prevailing. Given the state of current scientific knowledge, there is no way that a petitioner could satisfy such a requirement. And requiring proof of such would require scientific certainty, which is a bar too high. See Knudsen, 35 F.3d at 549 (explaining that "to require identification and proof of specific biological mechanisms would be inconsistent with the purpose and nature of the vaccine compensation program"). However, there must be more than conclusory opinions or speculation. Special masters consistently reject "conclusory expert statements that are not themselves backed up with reliable scientific support." Kreizenbeck v. Sec'y of Health & Hum. Servs., No. 08-209V, 2018 WL 3679843, at *31 (Fed. Cl. Spec. Mstr. June 22, 2018), mot. for rev. denied, decision aff'd, 141 Fed. Cl. 138, aff'd, 945 F.3d 1362 (Fed. Cir. 2020).

Petitioners' expert cardiologist, Dr. Chang, opined that T.B.D. had acute hypersensitivity myocarditis, described as an inflammatory condition of the heart muscle which caused ventricular tachycardia or ventricular fibrillation and cardiac arrest. He explained that heart dysrhythmias and left ventricular dysfunction are manifestations of myocarditis. In his expert reports, Dr. Chang also described the mechanism as an inflammatory reaction of the heart muscle to vaccination. The medical literature cited by the parties generally described this form of immune-mediated myocarditis based on its histological findings on biopsy as an eosinophilic infiltration of heart muscle causing injury.

In support of his mechanism of hypersensitivity myocarditis, Dr. Chang cited medical articles, including case reports, and these, along with articles filed by Respondent, establish that acute hypersensitivity myocarditis has been associated with vaccinations in infants and children. The articles establish that a hypersensitivity immune-mediated reaction can occur after vaccinations.

Barton et al. explains that acute myocarditis can occur after vaccinations evidenced by histological findings that strongly support a hypersensitivity reaction. Thanjan et al. proposes hypersensitivity reaction as the mechanism of myocardial injury after vaccination. While this is "usually a retrospective circumstantial diagnosis," suspected by virtue of a temporal association between vaccination and onset of illness, histology from cardiac biopsies has provided evidence to support the mechanism. Resp. Ex. C at 4. More specifically, Barton et al. describes a type III hypersensitivity immune complex-mediated reaction. Kounis and Mplani also support a finding that myocarditis can be caused by a hypersensitivity reaction. And a more recent article from Frustaci et al. identified eosinophilic myocarditis as virus-negative immune-mediated myocarditis associated with drug hypersensitivity reactions, including reactions to vaccinations. In summary, the medical literature supports the mechanism of hypersensitivity as a cause of post-vaccination myocardial injury.

The vaccines at issue here include DTaP, Hep B, and IPV (polio). These vaccines have been reported in cases of an immune-mediated hypersensitivity mechanism after vaccination. Thanjan et al. reported a 17-year-old who developed myocarditis after DTaP along with other vaccinations. Dilber et al. described a 14-year-old with myocarditis after tetanus vaccination. Boccara et al. reported myocarditis in a 31-year-old after diphtheria, tetanus, and polio vaccinations. Relative to infants, Kumar et al. described a six-week-old who developed myocarditis after the whole cell pertussis DPT vaccination, two additional cases associated with the whole cell DPT vaccination (at ages two and three months of age), and an infant who developed myocarditis after diphtheria, polio, and tetanus vaccinations. Barton et al. explain that cases of post-vaccination cardiac injury "support an immune-mediated hypersensitivity mechanism." Resp. Ex. D at 4.

While case reports do not provide strong evidence of causation, based on Dr. Chang's expert report, and the cited medical literature, the undersigned finds that Petitioners have provided preponderant evidence that vaccinations, including those at issue here, can cause post-vaccination hypersensitivity myocarditis.

B. Althen Prong Two

Under Althen prong two, Petitioners must prove by a preponderance of the evidence that there is a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." Capizzano, 440 F.3d at 1324 (quoting Althen, 418 F.3d at 1278). "Petitioner[s] must show that the vaccine was the 'but for' cause of the harm . . . or in other words, that the vaccine was the 'reason for the injury.'" Pafford, 451 F.3d at 1356 (internal citations omitted).

In evaluating whether this prong is satisfied, the opinions and views of the vaccinee's treating physicians are entitled to some weight. Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326 ("[M]edical 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 trustworthy evidence since they are created contemporaneously with the treatment of the vaccinee. Cucuras, 993 F.2d at 1528. The Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano, 440 F.3d at 1325. Instead, Petitioners may satisfy their burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

The undersigned finds that Petitioners have provided preponderant evidence that the vaccines administered to T.B.D. caused myocarditis for four reasons: (1) T.B.D.'s clinical course and diagnostic studies were consistent with post-vaccination myocarditis, (2) T.B.D.'s treating physicians identified myocarditis as a differential diagnosis, (3) T.B.D.'s treating physician opined that further vaccinations were contraindicated; and (4) there was no alternative cause found for T.B.D.'s cardiac arrest.

First, T.B.D.'s clinical course was consistent with hypersensitivity immune-mediated myocarditis. Relevant diagnostic studies include cardiac MRI, echocardiogram, EKGs, and cardiac enzymes. Echocardiogram showed mild-moderate left ventricular hypertrophy. Cardiac MRI showed moderate septal hypertrophy. The MRI did not exclude myocarditis, noting that the T2 images were of poor quality. The MRI reviewing physician opined that he was unable to exclude myocarditis due to T.B.D.'s age.

In addition to heart muscle abnormalities seen on imaging studies, T.B.D. also had laboratory evidence of heart muscle injury (very elevated troponin and BNP levels). While some elevation of these enzymes were attributed to resuscitation efforts, Respondent's experts did not refute that these abnormal values were consistent with myocarditis.

Although T.B.D.'s myocarditis panel was negative, this result does not preclude a finding of hypersensitivity myocarditis. Although the actual study results are not in the hospital records, the procedure appears to test for viral and not immune causes of myocarditis. This understanding is supported by the records of geneticist Dr. Derek Wong who referenced the study as a "viral myocarditis panel" and noting it was negative. Pet. Ex. 10 at 87. It is also supported by the expert report from Dr. Ringel, who opined the "viral panel" done after T.B.D.'s arrest was negative. Resp. Ex. F at 4. Given these opinions, the panel ruled out viral causes of myocarditis (although Dr. Ringel explained that the test does not rule out all viral causes).

Further, T.B.D.'s EKGs showed heart rhythm abnormalities identified in the medical literature as associated with myocarditis.[60] As explained by Frustaci et al., the clinical manifestations of myocarditis include "life-threating arrhythmias and sudden death." Resp. Ex. O at 1. T.B.D. presented with cardiac arrest, VT, and ventricular fibrillation. During his hospitalization, he had SVT, non-sustained VT, premature ventricular contractions, atrial arrhythmias, ectopic atrial tachycardias, and right bundle branch block. His abnormal heart rhythm required treatment with amiodarone and other medications.

Lastly, as it relates to his clinical course, like most of the infants/children described in case reports of post-vaccination myocarditis, T.B.D.'s myocarditis resolved. By February 2016, T.B.D.'s echocardiogram had returned to normal. See Pet. Ex. 10 at 81 ("[P]rior subtle appearance of moderate left ventricular hypertrophy not present today."). In April 2016, Dr. Silka noted that T.B.D. was "clinically stable" with no arrhythmias. Pet. Ex. 5 at 7.

Secondly, T.B.D.'s physicians identified myocarditis as a differential diagnosis. On December 18, Dr. Kung considered myocarditis in her list of differential diagnoses due to the presentation of cardiac arrest and left ventricular hypertrophy. Pet. Ex. 8 at 89. The diagnosis of myocarditis was also considered by Dr. Badran, when she noted that T.B.D. had "significant improvement without anti-inflammatory treatment, will not give IVIG/steroids at this time." Pet. Ex. 8 at 92. IVIG and steroids constitute treatment for immune-mediated conditions, including myocarditis. See, e.g., Resp. Ex. O at 4 (noting IVIG was a therapy for immune-mediated myocarditis); Resp. Ex. N at 1 (noting that "most [hypersensitivity myocarditis] patients respond well to steroids").

The differential diagnosis of myocarditis is only referenced several times, and it was not mentioned as a discharge diagnosis, although the reasons for this are not documented. Regardless, T.B.D. improved without IVIG or steroids. At discharge, T.B.D.'s diagnosis was hypertrophic cardiomyopathy, and it was thought to have a genetic basis. Once genetic testing failed to show a genetic cause of T.B.D.'s cardiomyopathy, the etiology of his cardiac arrest was unknown. Ultimately, T.B.D. did not have a diagnosis relative to his cardiac arrest. In summary, T.B.D. had a differential diagnosis of myocarditis, but after a thorough workup, he had no diagnosis. The prior differential diagnosis of myocarditis was not revisited.

The third reason for the undersigned's finding as to Althen prong two is based on T.B.D.'s treating physician statements related to vaccine causation.

Dr. Silka documented conversations with Petitioners about vaccine causation of the cardiac arrest, although these statements and opinions primarily note a temporal association and he characterized the question of causation as speculative or as a possibility. See, e.g., Pet. Ex. 10 at 140 (noting "any cause/effect relationship would be speculative"). In June 2016, after whole exome genetic testing was negative, Dr. Silka noted the "possibility" that T.B.D.'s cardiac arrest

[60] Medical literature states that in addition to abnormal rhythms, myocarditis can cause EKG abnormalities, such as ST segment elevation. The case reports related to infants did not mention this clinical finding. There is no evidence in T.B.D.'s medical record about whether he had any ST segment abnormalities.

was an untoward reaction to the DTaP vaccine. Id. at 22. Dr. Silka agreed to provide an objective letter for this claim. See Pet. Ex. 21. In the letter, Dr. Silka referenced a possible adverse reaction as a "distinct possibility." Id. at 1. Thus, while Dr. Silka questioned vaccine causation, he did not offer an opinion that more likely than not, vaccinations caused T.B.D.'s cardiac arrest.

T.B.D.'s primary care physician, Dr. Hartstein, took a stronger position. She recommended that Petitioners hold off on vaccinations for T.B.D. pending the genetic testing results. Once these studies were complete and did not provide an explanation for T.B.D.'s cardiac arrest, Dr. Hartstein agreed with Petitioners that vaccine causation could not be ruled out. Pet. Ex. 5 at 18 ("[T]here's no way that I can say whether or not [T.B.D.'s] arrest was associated with his vaccinations."). Dr. Hartstein submitted a VAERS report for T.B.D.'s cardiac arrest following his two-month vaccinations. See Pet. Ex. 9 at 1-3. In January 2017, at T.B.D.'s 15 month visit, Dr. Hartstein noted that some vaccines, including DTaP were "contraindicated." Pet. Ex. 5 at 98. In May 2017, Dr. Hartstein provided a medical exemption for all vaccinations. She concluded that while it was unknown what triggered T.B.D.'s cardiac arrest, vaccinations were to be held to avoid a "possible trigger for further arrhythmias." Pet. Ex. 17 at 1.

Dr. Hartstein's statements and letter of exemption do not offer an opinion that vaccinations more likely than not caused T.B.D.'s cardiac arrest. Instead, she used the phrase "possible trigger." However, she did conclude that vaccines were contraindicated for T.B.D. and wrote a permanent exemption for vaccines. While standing on its own, this evidence is insufficient to establish causation, her opinion finding vaccines were contraindicated and her letter of permanent exemption adds to the weight of circumstantial evidence.

"A treating doctor's recommendation to withhold a certain vaccination can provide probative evidence of a causal link between the vaccination and an injury a claimant has sustained." Andreu, 569 F.3d at 1376; see also Kelley v. Sec'y of Health & Hum. Servs., 68 Fed. Cl. 84, 98, 100 (2005) (determining that the petitioner's treating physicians' reluctance to authorize the petitioner with further tetanus vaccinations was "robust" medical evidence of vaccine causation). A treating physician's recommendation against future vaccination is supportive of a petitioner's Althen prong two burden and "helps satisfy the second Althen prong." Michie v. Sec'y of Health & Hum. Servs., No. 19-453V, 2023 WL 10410004, at *7 (Fed. Cl. Spec. Mstr. Dec. 4, 2023).

The fourth reason is the fact that no alternative cause was found for T.B.D.'s cardiac arrest after an extensive workup, including whole genome testing for cardiac genetic mutations known to cause hypertrophic conditions and/or arrhythmias. Also, the metabolic studies were normal, cardiomyopathy panel was normal, viral myocarditis panel was normal, and MPS was ruled out.

While Petitioners are not required to eliminate all alternative causes, the lack of alternative cause "may be included as part of evidence to satisfy" Althen prong two. Ramsey, 2022 WL 17821368, at *6; see also Walther v. Sec'y of Health & Hum. Servs., 485 F.3d 1146, 1151 (Fed. Cir. 2007) (noting petitioners may use rely on "evidence eliminating other potential causes to help carry the burden on causation"). Here, Petitioners' evidence that no alternative

cause was found for T.B.D.'s cardiac arrest provides additional support for their prong two showing.

For all the reasons set forth above, the undersigned finds that Petitioners have proven Althen prong two by preponderant evidence.

C. Althen Prong Three

Althen prong three requires Petitioners to establish a "proximate temporal relationship" between the vaccination and the injury alleged. Althen, 418 F.3d at 1281. That term has been interpreted to mean a "medically acceptable temporal relationship." Id. Petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disease's etiology, it is medically acceptable to infer causation-in-fact." de Bazan, 539 F.3d at 1352. The explanation for what is a medically acceptable time frame must also coincide with the theory of how the relevant vaccine can cause the injury alleged (under Althen Prong One). Id.; Koehn v. Sec'y of Health & Hum. Servs., 773 F.3d 1239, 1243 (Fed. Cir. 2014); Shapiro, 101 Fed. Cl. at 542.

T.B.D. received his vaccinations on December 15, 2015, and suffered cardiac arrest approximately 23 hours later. Petitioners posited mechanism is acute hypersensitivity myocarditis, an immune-mediated process that injured the heart muscle causing heart dysrhythmias and abnormality of the left ventricle. Dr. Chang cited case reports where onset of myocarditis occurred one to four days after vaccination. In Dilber et al., onset occurred three days after tetanus vaccination; in Boccara et al., onset occurred four days a diphtheria, tetanus, and polio vaccination; and in Kumar et al., onset occurred three days after tetanus vaccination with the authors citing three additional infant cases where onset occurred within hours (24 hours and 72 hours).

Respondent's cardiology expert, Dr. Ringel, also provided medical literature from Barton et al. demonstrating onset of vaccine-associated myocarditis ranged from one to 30 days after vaccination. Several cases reported an onset of one day. "[C]ardiac manifestations of hypersensitivity myocarditis . . . typically appear[s] after the first week and up to [four] weeks postvaccination, but may occur as early as [one to four] days postvaccination." Resp. Ex. D at 4.

Moreover, Respondent's experts did not refute the fact that there was a temporal association between vaccination and onset (cardiac arrest) in this case.

Petitioners allege hypersensitivity myocarditis, and they have shown by medical literature that onset can occur as early as one day after vaccination. Respondent's experts do not refute this position. Therefore, the undersigned finds that Petitioners have proven Althen prong three by preponderant evidence.

VII. CONCLUSION

For the reasons discussed above, the undersigned finds that Petitioners have established by preponderant evidence that T.B.D. suffered a Vaccine Injury Table claim for encephalopathy.

In the alternative, the undersigned finds by preponderant evidence that the vaccinations T.B.D. received on December 15, 2015, caused acute hypersensitivity myocarditis, which triggered dysrhythmias and cardiac arrest. Cardiac arrest then caused T.B.D. to suffer a hypoxic ischemic neurological injury and encephalopathy. Accordingly, Petitioners are entitled to compensation.

A separate damages order will be issued.

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master