OFFICE OF SPECIAL MASTERS

No. 96-820V

(Filed: December 21, 1998)

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CHATIE BANTUG CRUZ (formerly known as Rosario Bantug and Rosario Bantug Cruz), an individual,	*	
	*	
Petitioner,	*	**TO BE PUBLISHED**
	*	
v.	*	
	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
	*	
Respondent.	*	
	*	
* *		

Lisa A. Roquemore, Costa Mesa, California, for petitioner.

Mark W. Rogers, Washington, D.C., for respondent.

ENTITLEMENT DECISION

GOLKIEWICZ, Chief Special Master

I. PROCEDURAL BACKGROUND

On December 30, 1996, petitioner filed a petition pursuant to the National Childhood Vaccine Injury Act of 1986 (hereinafter referred to as "the Act")[(1)] alleging that she contracted poliomyelitis on December 31, 1995, as a result of the oral polio vaccine ("OPV") her daughter, Samantha Cruz, received on October 13, 1995, at six months of age. Petition, filed 12/30/96, at 2. Medical records submitted with the petition contained numerous notations from her treating and consulting physicians that petitioner suffered from paralytic polio as a result of contact with her recently vaccinated child. Medical Records, filed 12/30/96 and 4/3/98.[(2)] Respondent's Rule 4 Report concluded petitioner should be denied compensation for failure to sustain her burden that she contracted paralytic polio; instead, respondent argued petitioner suffered from Guillain-Barre Syndrome. Respondent's Report, filed 3/31/97, at 3.[(3)] Thereafter, the parties submitted their respective expert reports; petitioner filed Dr. David C. Redfield's report on June 17, 1997 (hereinafter referred to as "Dr. Redfield's Rpt."); respondent filed Dr. Barry G. W. Arnason's medical

opinion as Exhibit A on August 15, 1997.

Following the submission of the above information, the court conducted a factual and expert hearing in San Diego, California, on April 30, 1998, to address the only issue in this matter, which is whether petitioner contracted poliomyelitis or suffered from Guillain-Barre Syndrome ("GBS"). Petitioner testified, as did her treating physician and expert, Dr. Redfield, in support of petitioner's claim that she suffered from paralytic polio as a result of contact with her recently vaccinated daughter. Dr. Arnason testified on behalf of respondent that petitioner instead suffered from GBS.[4]

Post-hearing briefs have been submitted by both parties and the record is now closed. After reviewing the entire record, and for the reasons set forth below, the court finds petitioner is entitled to compensation.[5] A full discussion follows.

II. FACTUAL BACKGROUND[6]

On April 17, 1995, petitioner gave birth to her daughter, Samantha Cruz. Pet. at 2. On October 13, 1995, at six months of age, Samantha received her oral polio vaccination. Pet. at 2.

On December 19, 1995, petitioner visited Green Hospital of Scripps Clinic complaining of lower outer quadrant pain and tenderness, left-sided headache, and a history of left-sided abdominal pain for 3 weeks and chest pain for 1 month; she denied, among other things, intermittent diarrhea. An examination revealed a temperature of 98.0ºF, with intact gait and cranial nerves. Her reflexes were noted at +2/4 bilateral in the upper and lower extremities. Petitioner was diagnosed with a probable migraine and chest and abdominal pain, possibly associated with a gastrointestinal infection. She was advised to follow up in 7-8 days with Dr. Sargeant. M.R. II at 64-65.

Petitioner complained six days later, on December 25, 1995, of fever, stomach cramping, a three week history of burning sensation on the left side, some diarrhea, and dizziness with sitting and walking. Petitioner's temperature was 98.4ºF. She was diagnosed with gastroenteritis and prescribed 2 liters of IV fluid and appropriate medication.[7] M.R. II at 89, 94. Dr. Sargeant assessed petitioner at her December 28, 1995, appointment with nonspecific abdominal and pelvic pain with loose stools and advised petitioner to continue medicating with immodium and pepcid. M.R. II at 58.

On January 1, 1996, petitioner again visited Green Hospital, this time complaining of a left-sided occipital headache with onset at 10 p.m., the evening before. By midnight New Year's Eve, the severe headache was accompanied by numbness in the left arm, left facial region, and left tongue, which persisted as the headache worsened. Petitioner also complained of an aching back, "pins and needles" on her left side, numb knees, a two week history of "rubbery" legs, difficulty swallowing, and neck stiffness. However, petitioner reported improvement of her previous 1-2 week history of stomach pain and diarrhea.[8] An examination revealed a temperature of 98.8ºF, a left facial droop, deviation of the tongue to the left side on extension, dysarthria[9], subjective decrease in sensation with the left deep tendon reflexes, and intact strength, muscle stretch reflexes, and sensation. Petitioner was diagnosed with complicated migraine, with plans to rule out cerebrovascular accident, cerebral ischemic episode, vasculitis, and/or acute/chronic central nervous system infection. Dr. Romine admitted petitioner for continued care and studies and prescribed analgesics for her headache. M.R. III at 16, 20-22.

On January 2, 1996, petitioner's condition worsened with diminished swallowing capabilities, significant drooling, almost complete paralysis of the tongue, difficulty with deep aspirations, significant dysarthria, left-sided facial weakness, meningismus, neck and back pain, intact reflexes with possible left upper extremity slightly diminished compared to right, intact sensation, mild to moderate proximal leg

weakness (right greater than left), and mild to moderate proximal arm weakness (left greater than right). Petitioner was diagnosed with asymmetrical progressive bulbar paresis and quadriparesis with pleocytosis. Guillain-Barre Syndrome and polio, among other causes, remained under consideration. M.R. III at 23-25, 26-29, 31, 33-34. Thereafter, petitioner's condition worsened and peaked over the next couple of days. The progression and specific symptoms of petitioner's paralytic illness are addressed in great detail in the Discussion portion of this decision and, therefore, will not be reiterated here to avoid unnecessary duplication.

Petitioner's condition slightly improved by January 17, 1996, and she was transferred to Scripps' Encinitas Acute Rehabilitation Unit for continued speech, physical, and occupational therapy. Dr. Redfield's primary discharge diagnoses were the following: poliomyelitis with bulbar paralysis and quadriparesis, staphylococcal urinary tract infection, and mild hypertension. M.R. III at 17-19. While at the rehabilitation hospital, petitioner continued to recover slightly from her neurological deficits and significantly from her functional disabilities. However, petitioner's bulbar problems showed no significant improvement. Petitioner was discharged home on February 29, 1996, nearly two months after her admission, with discharge diagnoses of quadriparesis, dysarthria, dysphagia presumptively secondary to poliomyelitis[(10)], and admitting borderline hypertension and tachycardia presumptively secondary to autonomic dysfunction (resolved)[(11)]. M.R. II at 2-3. She continues to experience residual facial and extremity weakness. Tr. at 210, 213; Petitioner's Prehearing Memorandum, filed 4/3/98, at 8.

III. DISCUSSION

A. Statutory Scheme

Causation in Vaccine Act cases can be established in one of two ways: either through the statutorily prescribed presumption of causation, or by proving causation-in-fact. A petitioner must prove one or the other in order to recover under the Act.[(12)] The Vaccine Injury Table lists certain injuries and conditions which, if found to occur within a prescribed time period, create a rebuttable presumption that the vaccine caused the injury or condition. The presumption may be overcome by an affirmative showing by respondent that the injury was caused by a factor unrelated to the administration of the vaccine.[(13)] To demonstrate entitlement to compensation in a causation-in-fact case (*i.e.*, off-Table), a petitioner must affirmatively show by a preponderance of the evidence that the vaccination in question more likely than not caused the injury alleged. §§11(c)(1)(C)(ii)(I) and (II); Grant v. Secretary of HHS, 956 F.2d 1144 (Fed. Cir. 1992). The Federal Circuit in Grant summarized the legal criteria required: "Causation-in-fact requires proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury. A reputable medical or scientific explanation must support this logical sequence of cause and effect." Grant, 956 F.2d at 1148 (citations omitted); see also Strother v. Secretary of HHS, 18 Cl. Ct. 816 (1989), aff'd without opinion, 950 F.2d 731 (Fed. Cir. 1991).[(14)] A petitioner claiming an injury of paralytic polio, following the administration of the OPV to another individual (*i.e.*, contact case or vaccine-associated community case), is not bound by a specific time frame within which that injury must occur. §14(a). However, to be afforded a presumption of causation, a petitioner must demonstrate by a preponderance of evidence that he or she suffered from paralytic polio following contact with an individual vaccinated with the oral polio vaccine. §§13(a)(1) and 14(a). This case is measured against these standards.

In this case, petitioner claims she contracted paralytic polio, or poliomyelitis[(15)] following contact with her daughter, Samantha, who received the OPV in October 1995. The date of Samantha's OPV administration is uncontested, as is that petitioner was responsible for Samantha's care. However,

respondent contests petitioner's Table injury claim of paralytic polio, and asserts petitioner suffers from Guillain-Barre Syndrome, most likely as a result of a *Campylobacter* infection. Guillain-Barre Syndrome is not a Table injury resulting from the oral polio vaccine. Therefore, the only issue in this case is whether petitioner suffers from paralytic polio, which would afford petitioner the presumption of causation, or GBS, which would result in the dismissal of petitioner's claim.

The court is persuaded, following an exhaustive review of the record, that petitioner has sustained her burden. In reaching this conclusion, the court first addresses respondent's vigorous contention that laboratory tests are dispositive in polio cases. Respondent's Closing Brief, filed 7/29/98, at 1; Respondent's Responsive Closing, filed 9/2/98, at 21. Thereafter, the court will address its rationale for finding that petitioner proved by a preponderance of the evidence that she suffers from polio, and thus is entitled to compensation.

B. Respondent's Claim that Laboratory Tests are Dispositive

Respondent argues that petitioner's claim "turns strictly upon laboratory reports and applicable medical literature," "is not tenable in light of the laboratory results," and "should be decided based upon the laboratory testing." R. Closing at 1, 5, 19-20. Respondent asserts the lab results are dispositive, "unequivocal[ly] diagnostic," and preclude petitioner's poliomyelitis diagnosis. R. Closing at 1-3, 32. In short, respondent argues that laboratory tests are determinative and that petitioner's clinical symptoms and doctors' diagnoses are irrelevant. In counsel's responsive closing, he revises this position and insists instead that whether "it is possible to diagnose polio in the absence of any serology test results . . . is not the question here. These tests were performed here and cannot simply be ignored. The issue therefore is whether polio is a plausible diagnosis when serology testing indicates no increase in antibody titers." R. Resp. Closing at 21. The difference between respondent's positions is subtle, and warrants further inspection. Because respondent neither submitted nor cited any literature directly supporting its view, the court independently examined respondent's argument, as well as petitioner's claim that the Centers for Disease Control and Prevention ("CDC"), the agency charged with the paramount duty of monitoring the existence of infectious diseases within our country, does not itself require a four-fold rise in the antibody titer to diagnose polio. Pursuant to this review, the court finds that positive laboratory test results are not an absolute requirement for diagnosing paralytic polio. The court's reasoning follows.

First, the role of laboratory tests in diagnosing polio is described in the literature using indefinite language, such as "can usually be identified,"(16) "is most readily established,"(17) "usually confirms the diagnosis,"(18) "is suggestive,"(19) "may be further supported,"(20) "can aid in diagnosis,"(21) "can be made,"(22) and "can be established."(23)

Second, even where the language appears unambiguous, as it arguably does in Exhibits D through F (see respondent's closing, filed 7/29/98, at page 6, for the specific provisions), these passages discuss laboratory tests *in conjunction with* diagnoses. In other words, these passages explain what physicians should expect when conducting or reviewing lab results, and/or what a diagnosis *based on the laboratory tests* might require. Simply put, the passages do not state that a patient's paralytic polio diagnosis depends *solely* on the lab results, without any consideration of the clinical manifestations, nor does the literature specifically convey that a non-positive result deems a polio diagnosis medically impossible.

Third, the court questions the conclusive nature of lab tests which often rely on timing, test conducted, and specimen tested. For instance, virus recovery from the cerebrospinal fluid ("CSF") is rare in poliovirus infections and the neutralizing antibody test "is expensive and cumbersome, requiring careful selection of serotypes for use as antigens. Serodiagnosis is generally reserved for critical situations in which the etiology is questionable." R. Exh. D at 822. Moreover, "isolation of [the polio] virus from fecal

specimens only must be interpreted more cautiously because symptomatic shedding from the bowel may persist for as long as 4 months." R. Exh. D at 822. Cerebrospinal fluid specimens "may be less helpful 2 or 3 weeks into the illness, when (in poliomyelitis) the cell count has returned to normal, but the protein elevation may persist." R. Exh. F at 811. In addition, "[i]nfectious virus particles in human fluids and tissues are usually few in number, and many viruses are easily disrupted and inactivated even at room temperature." R. Exh. E at 144. The California Department of Health Services' Viral and Rickettsial Disease Laboratory cautioned the following for the Enzyme Immunoassay and Polymerase Chain Reaction ("PCR") tests:

This [enzyme immunoassay] technique is currently a research procedure and has not been established as a diagnostic procedure. The presence of enterovirus IgM does not prove that an enterovirus infection is the cause of the patient's current illness.

. . . PCR for enterovirus is not a routine diagnostic service of this laboratory. We are currently evaluating the usefulness of this experimental, research technique as a diagnostic test, but the significance of the test results has not been determined and we are not currently enrolled in a proficiency testing program that covers PCR testing for this virus.

M.R. II at 187. More convincingly, the CDC, while recognizing the importance and critical nature of tests to rule out or confirm a diagnosis of paralytic poliomyelitis, also notes the limits of the testing:

The likelihood of poliovirus isolation is highest from stool specimens, intermediate from pharyngeal swabs, and very low from blood or spinal fluid. The isolation of poliovirus from stool specimens contributes to the diagnostic evaluation but does not constitute proof of a causal association of such viruses with paralytic poliomyelitis. Isolation of virus from the cerebrospinal fluid (CSF) is diagnostic but is rarely accomplished . . . Serology may be helpful in supporting or ruling out the diagnosis of paralytic poliomyelitis . . . A four-fold rise between the acute and convalescent specimens suggests poliovirus infection. Non-detectable antibody titers in both specimens may help rule out poliomyelitis, but may be falsely negative in immunocompromised persons, who are also at highest risk for paralytic poliomyelitis. In addition, neutralizing antibodies appear early and may be at high levels by the time the patient is hospitalized; thus, a four-fold rise may not be demonstrated.

D. Rebecca Prevots, PhD, MPH, Linda Quick, MD, MPH, Peter Strebel, MBChB, MPH, and Roland Sutter, MD, MPH & TM, Chapter 10: Poliomyelitis (visited Sept. 29, 1998) <http://www.cdc.gov/nip/manual/poliomye/poliomye.htm>.[(24)]

Fourth, and perhaps most persuasive, the CDC, despite its role in monitoring infectious diseases, does not itself require, *sine qua non*, positive laboratory tests to diagnose poliomyelitis. The CDC defines a probable case of poliomyelitis, for reporting purposes, as one meeting the clinical definition: "[a]cute onset of a flaccid paralysis of one or more limbs with decreased or absent tendon reflexes in the affected limbs, without other apparent cause, and without sensory or cognitive loss." D. Rebecca Prevots, PhD, MPH, Linda Quick, MD, MPH, Peter Strebel, MBChB, MPH, and Roland Sutter, MD, MPH & TM, Chapter 10: Poliomyelitis (visited Sept. 29, 1998) <http://www.cdc.gov/nip/manual/poliomye/poliomye.htm>. The case is further classified as "confirmed" if it "meets the clinical case definition and in which the patient has a neurologic deficit 60 days after onset of initial symptoms, has died, or has unknown follow-up status"; the case may be further classified based on epidemiologic and laboratory measures. D. Rebecca Prevots, PhD, MPH, Linda Quick, MD, MPH, Peter Strebel, MBChB, MPH, and Roland Sutter, MD, MPH & TM, Chapter 10: Poliomyelitis (visited Sept. 29, 1998) <http://www.cdc.gov/nip/ manual/poliomye/poliomye.htm>. However, a case is considered "laboratory confirmed" if it is "confirmed by one or more of the laboratory methods listed in the case definition under Laboratory Criteria for Diagnosis." Definitions of Terms Used in Case

Classification (visited Sept. 29, 1998) < http://www.cdc.gov/epo/mmwr/other/case_def/define97.html>. The CDC notes that "[s]ome clinical syndromes do not have confirmatory laboratory tests; however, laboratory evidence may be one component of a clinical definition . . . [on the other hand] [s]ome diseases require laboratory confirmation for diagnosis regardless of clinical symptoms, whereas others are diagnosed based on epidemiologic data." Case Definitions for Infectious Conditions Under Public Health Surveillance (visited Sept. 29, 1998) <http://www.cdc.gov/epo/mmwr/other/case_def/intro97.html>. Given the absence of a section entitled "Laboratory Criteria for Diagnosis" under the case definition for poliomyelitis, or any other language to that effect, the court concludes the CDC does not require laboratory confirmation (regardless of the clinical manifestations) to diagnose polio. D. Rebecca Prevots, PhD, MPH, Linda Quick, MD, MPH, Peter Strebel, MBChB, MPH, and Roland Sutter, MD, MPH & TM, Chapter 10: Poliomyelitis (visited Sept. 29, 1998) <http://www.cdc.gov/nip/manual/poliomye/poliomye.htm>; Poliomyelitis, Paralytic (visited Sept. 29, 1998) <http://www.cdc.gov/epo/mmwr/other/case_def/polio97. html>. Moreover, when discussing the use of lab results, the CDC uses inconclusive language, similar to that seen in respondent's literature: *e.g.*, "[s]erology *may be helpful* in supporting or ruling out the diagnosis of paralytic poliomyelitis"; "[a] four-fold rise between the acute and convalescent specimens *suggests* poliovirus infection." D. Rebecca Prevots, PhD, MPH, Linda Quick, MD, MPH, Peter Strebel, MBChB, MPH, and Roland Sutter, MD, MPH & TM, Chapter 10: Poliomyelitis (visited Sept. 29, 1998) <http://www.cdc.gov/nip/manual/poliomye/poliomye.htm> (Emphasis supplied). [(25)]

The court also finds no support for respondent's contention in the expert testimony. Dr. Redfield expressed repeatedly that poliomyelitis may be diagnosed absent supporting laboratory results. Tr. at 106, 168. Drs. Arnason's and Weibel's opinions, that without a four-fold increase proof is lacking, are contrary to the literature submitted, as discussed above. Tr. at 223; Respondent's Report Attachments at RE-5.

The court finds no conclusive support in the expert testimony or medical literature for respondent's notion, however clarified in the responsive closing, that positive results for the polio antibody or virus must exist for a petitioner to prevail on his or her paralytic polio claim in a contact case.[(26)] While finding that lab confirmation is not essential in diagnosing polio, the court is, however, concerned about the lack of confirmatory lab results in Ms. Cruz's case and agrees with respondent that her test results should not be ignored. Therefore, the lab results will be considered and weighed in context with the other evidence in this case.

C. Petitioner's Table Injury Claim of Paralytic Polio

1. Court's Conclusion

Petitioner must demonstrate she suffered from paralytic polio to be afforded a presumption of causation under the Act. §13(a)(1) and 14(a). This proof can come from laboratory testing, clinical signs and symptoms, the doctors' assessments, and the doctors' trained medical judgments. As discussed, the lab tests were not confirmatory. Unfortunately, the symptoms are inconclusive.

The record reveals that the symptoms associated with paralytic polio and GBS are similar, occurring in one as well as the other even if only in rare circumstances. Despite the similarities, the experts disagreed on the characteristic and significance of almost every symptom expressed; this case exemplified the classic battle between the experts. Unfortunately, the court cannot determine this case based on petitioner's symptoms alone. Simply stated, the comparison of petitioner's symptoms with the expert testimony and literature affords the court no absolute answer on which diagnosis is correct. For instance, petitioner experienced the following symptoms which may be associated with either diagnosis: gastrointestinal upset (with or without diarrhea), aches and pains, CSF protein level of 51, asymmetrical and bilateral paralysis, progression of the paralysis over a few days, peaking of the paralysis by day 5,

proximal muscle weakness, facial weakness, paresthesia, loss of reflexes, and normal EMG/NCV results (initially). In contrast, petitioner exhibited the following symptoms which are arguably rare for polio: diarrhea, absence of fever, and an inability to isolate the polio virus or detect the polio antibody through laboratory tests. The following symptoms petitioner experienced are considered uncommon or rare for GBS: presence of fever, meningismus, pleocytosis, presence of polymorphonuclear leukocytes in the CSF, paralysis commencing in the face, ptosis, swallowing and oral secretion difficulties, and progression of paralysis over hours.[(27)] Because the case cannot be resolved on the significance of petitioner's symptoms alone, the court must look to other factors such as the experience of the experts, the deference, if any, to be afforded the treating physicians, the support of petitioner's case through the literature, and the strength of respondent's arguments.[(28)] The court concludes, after an exhaustive review of the record, that petitioner has sustained her burden by a preponderance of the evidence. The court's reasoning follows.

First, while petitioner's symptoms may be related to either diagnosis, the medical records and literature support Dr. Redfield's foundation for polio. In addition, the court found Dr. Redfield articulate, knowledgeable, credible, and unwavering in his opinions. He expressed no biases. He testified largely consistent with the medical records and literature and offered information from memory when the medical records lacked details. Moreover, Dr. Redfield communicated with and examined petitioner on a daily basis. The court rejects respondent's contention that Dr. Redfield's intimacy with petitioner's condition is fully duplicated in the medical records such that respondent's expert is in the same position for re-diagnosing. Critical symptomology was viewed first-hand by Dr. Redfield and testified to accordingly. Dr. Redfield maintained his opinion, even in the face of concessions. He admitted inexperience in certain fields, but appeared to have slightly greater experience with polio, an infectious disease, than Dr. Arnason.[(29)] Dr. Redfield further admitted at trial that on petitioner's first day at the hospital, her symptoms were confounding. Dr. Redfield testified: "[T]he first day that I met Mrs. Cruz, she had an illness with few defining characteristics, and certainly no classic characteristics . . . [the symptoms she complained of and the examination conducted] allowed no specific classic characteristic diagnosis at all." Tr. at 42. More importantly, the court finds quite persuasive, and cannot emphasize enough, that two physicians diagnosed petitioner with polio, continued to do so over the course of her stay, *and ruled out numerous differential diagnoses, including GBS, despite knowing that certain laboratory results were negative for polio.*[(30)]

Second, respondent's case is weakened by her reliance on rare symptoms or variant forms of GBS to support Dr. Arnason's diagnosis. For instance, petitioner's white blood cell count of 15, per Dr. Arnason's own article, falls outside the *strongly suggestive* range and within the *variant* range. R. Exh. C at 1456. Petitioner's initial symptoms included weakness of the tongue; later she developed ptosis.[(31)] Dr. Arnason's literature asserts GBS weakness *rarely* begins with facial diplegia, ptosis is *uncommon* in GBS, and *less than 5% of the cases* involving facial weakness begin with the tongue. R. Exh. C at 1452, 1456. Petitioner's weakness rapidly progressed over several hours. Tr. at 42. Dr. Arnason opined he would look for events happening within hours or days to diagnose polio, such as a limb progressing from full strength to complete paralysis over hours or a day or so. R. Exh. A at 1; Tr. at 246. Dr. Arnason submitted swallowing and secretion problems are a feature of polio and *alert a physician to this diagnosis*; however, he neglected to address petitioner's swallowing and secretion complaints, stating simply that bulbar involvement may occur in a *variant form* of GBS. Tr. at 246-247; R. Exh. A at 1.

The reliance on these rare symptoms demonstrates the lengths respondent ventured to re-diagnose petitioner, which the court strongly questions. In numerous cases before this court, respondent has deferred, without exception in this court's memory, to the treating physician's diagnosis. In this case, petitioner presented respondent with extensive medical records which documented two treating physicians' opinions that petitioner had poliomyelitis. This case is unlike many others where the

petitioner's injury claimed is neither supported by, nor even mentioned in, the medical records. In those instances, respondent will meticulously examine the records to determine if petitioner's claims are supported. Respondent will closely scrutinize an expert witness claiming an injury that is not substantiated by the medical records, and in such cases, will seek her own independent expert to either confirm or reject petitioner's expert's opinion. This is the nature of litigation under the Program, and the court makes no criticism of the process in such cases. However, where, as here, the records are substantial, detailed, and replete with notations of the treaters' thought-processes and conclusions, the court questions respondent's, in essence, re-diagnosing petitioner. The court understands the CDC's conclusion was likely persuasive in directing respondent's defense of this case. However, their recommendation rests primarily on the polio laboratory results, which as shown above the CDC does not require.(32) In addition, the strength of any symptoms relied upon by the CDC physician panel, to find petitioner suffered GBS following a *Campylobacter* infection, lose their persuasiveness when pitted against Dr. Arnason's concession that petitioner's symptoms fit a polio diagnosis as well.(33) (34) Tr. at 223. Furthermore, respondent relies on *Campylobacter* results from petitioner's serum, even though Dr. Arnason's article cautions that diagnosis of the infection from serologic specimens is "less secure . . . since in older populations up to 50 per cent of individuals have serologic evidence of prior infection." R. Exh. C at 1441. Despite these criticisms, the court did not review Dr. Arnason's testimony lightly. This court has relied on his testimony in the past, and has found him to be extremely knowledgeable and highly persuasive. See Trojanowicz v. Secretary of HHS, No. 95-215V, 1998 WL 774338 (Fed. Cl. Spec. Mstr. July 1, 1998)(reissued for publication October 16, 1998). However, in this case, he simply failed to support his views.

Weighing all of the evidence, the court concludes petitioner has sustained her burden of demonstrating beyond a preponderance of the evidence that she sustained paralytic polio from the OPV administered to her daughter. While based on petitioner's *symptoms* alone, the evidence is arguably in equipoise, there is no question that polio is a medically supported diagnosis in this case. As noted, Dr. Redfield's testimony was persuasive and supported by the medical records and literature. When Dr. Redfield's testimony, the medical records, and the literature are viewed in light of the weak testimony provided by Dr. Arnason, the court is convinced by a preponderance of the evidence that petitioner's illness was polio, not GBS. The whole of the evidence persuades this court petitioner more likely than not sustained the Table injury of paralytic polio.

A discussion of the record follows and is presented only to demonstrate that the symptoms, at times, overlap with each diagnosis, and on other occasions, are rare for a particular diagnosis. As stated earlier, the symptoms are not determinative of the diagnosis, but are a piece of the medical puzzle. The discussion is arranged categorically by the symptoms expressed and encompasses first, a discussion of the expert's testimony; second, an examination of the applicable medical literature; and third, a review of petitioner's medical records.

2. Discussion of Petitioner's Symptoms

Initial Illness

Dr. Redfield testified that poliomyelitis may be preceded by an initial illness resulting in symptoms similar to a minor viral infection and lasting from days to a week or more. Tr. at 27, 29. He also agreed GBS may be triggered by a viral or bacterial infection, such as *Campylobacter*. Tr. at 35, 188. Dr. Redfield believed petitioner suffered from a "short term prodromal illness" prior to the onset of her paralysis, which lasted 1 or 2 weeks and consisted of "fever, sweats, loss of appetite, abdominal discomfort, and some diarrhea." Dr. Redfield's Rpt. at 2-3. This was followed soon thereafter by "difficulty swallowing, difficulty moving her tongue, left hand tingling, and ultimately left arm weakness associated with severe headache, and neck and back stiffness." Dr. Redfield's Rpt. at 2. Dr. Arnason

agreed that polio may be preceded by an illness, but stressed its febrile nature. R. Exh. A at 1.

The literature specifies that a nonspecific febrile illness may result following infection with the polio virus. R. Exh. D at 822. The illness may last 1-3 days, but without central nervous system localization. R. Exh. D at 822. The onset is similar to any acute infection and the clinical manifestations of the illness vary. R. Exh. E at 146; R. Exh. F at 808. The illness is followed by a short period of "wellness" (for approximately 2-5 days) before the abrupt onset of a major illness begins, characterized by a rise in temperature and aseptic meningitis. R. Exh. E at 146; R. Exh. F at 809. In GBS, an antecedent infectious illness occurs in at least 50% or more of the cases. R. Exh. C at 1438; R. Exh. G at 613. The illness begins during the first few weeks of the infection, and usually clears prior to the onset of the neuropathic symptoms. R. Exh. C at 1438; R. Exh. G at 613. The antecedent symptoms vary but have been described as a flu-like upper respiratory infection with fever; some patients (10-20%) have complained of a preceding acute dysenteric episode. R. Exh. C at 1438-1439. Like polio, an interval follows the initial symptoms, and then the neuropathic symptoms emerge; this interval varies and is usually 1-3 weeks and "occasionally . . . as long as 6 weeks." R. Exh. C at 1439. In the majority of GBS cases, the cause of the prodromal illness is unknown. R. Exh. C at 1439.(35)

Aseptic Meningitis

Dr. Redfield testified aseptic meningitis occurs in polio, not GBS, and "is completely variable in its clinical expression." Tr. at 29, 34, 102. Dr. Arnason generally concurred. Tr. at 242. Dr. Redfield opined petitioner presented to the hospital in the aseptic meningitis phase, demonstrated by her initial complaints and CSF results. Tr. at 42, 78, 81, 103, 198. Dr. Arnason avoided this assertion and focused instead on the various symptoms often associated with meningeal irritation, see *infra.*

Aseptic meningitis abruptly begins, with the recurrence of fever, 5-10 days following the end of the initial illness and usually 1-2 days before paralysis ensues.(36) R. Exh. D at 823; R. Exh. F at 809. The meningitis may be mild and self-limited, but usually begins with fever, headache, and stiff neck; aseptic meningitis is clinically indistinguishable from other enteroviral infections. R. Exh. D at 823; R. Exh. F at 809, 811.(37)

Gastrointestinal Upset

The experts disagreed on the characterization and significance of petitioner's gastrointestinal upset. Dr. Arnason identified petitioner's complaints as a "diarrheal illness" and rejected that this occurs with polio's febrile illness. R. Exh. A at 1-2. Dr. Redfield disagreed with this characterization since medical records indicated only a "couple of episodes of diarrhea" and "some diarrhea." Tr. at 110-111; Dr. Redfield's Rpt. at 2. Dr. Redfield further opined that diarrhea is an occasional, but not classic, feature of polio's prodromal illness, and related petitioner's gastrointestinal upset to polio. Tr. at 27, 189. Dr. Arnason related petitioner's diarrhea to a *Campylobacter* infection and corroborated his opinion with positive serology results and the literature which states the infection precedes 20% of GBS cases. R. Exh. A at 2.

The literature recognizes that 4-8% of polio cases include a history of anorexia, vomiting, abdominal pain, and intestinal upset, although it neither accepts nor rejects diarrhea as a symptom of the febrile illness nor further defines "intestinal upset." R. Exh. F at 808; R. Exh. H at 480. These symptoms can last hours to approximately 2 days, but are clinically indistinguishable from other viral illnesses. R. Exh. F at 808. In contrast, 10-20% of GBS cases follow an acute dysenteric episode or diarrheal episode, most resulting from a *Campylobacter* infection. R. Exh. C at 1439, 1441.

Petitioner's medical records report various episodes of gastrointestinal upset, including some stomach and

abdominal cramping and pain and anorexia. M.R. II at 2, 89, 94; M.R. III at 17, 20, 23, 26, 27, 30, 32. The severity of the diarrhea is unclear.[(38)]

Fever

Dr. Redfield associated feverishness or a low grade fever of 99ºF or more with a polio presentation. Tr. at 27, 67, 86, 144. Dr. Arnason ardently contended polio patients experience fever (100ºF or more) at onset and for days thereafter, making the presence of fever important in differentiating polio from GBS. Tr. at 226, 246, 250; R. Exh. A at 1. Dr. Redfield opined a polio patient's paralysis could continue even where medication reduces the fever. Tr. at 199; *compare* Tr. at 145, 148. Dr. Arnason generally agreed with this statement, but considered this atypical; he also rejected that the fever in polio is easily treatable. Tr. at 226, 251. Dr. Redfield opined the presence of fever in GBS cases is unusual. Tr. at 67, 144. Dr. Arnason, again, generally agreed, but noted a GBS patient may present with a past history of fever. Tr. at 251. Dr. Arnason further opined the weakness in GBS typically continues in the absence of fever. Tr. at 226. Turning to petitioner's case, Dr. Redfield cited her initial complaint of 1-2 weeks of fever and sweats, and her January 1, 1996 temperature of 99ºF, as evidence supporting his diagnosis. Dr. Redfield's Rpt. at 2; Tr. at 147. He further explained petitioner entered the hospital during the aseptic meningitis phase of her illness and was thereafter placed on antipyretics. Tr. at 142-143, 146. In contrast, Dr. Arnason concluded petitioner never suffered a fever throughout her course; consequently, he rejected the polio diagnosis. Tr. at 226.

The literature supports the presence of fever in 4-8% of polio cases, which lasts hours to days, and is clinically indistinguishable from other infections. R. Exh. F at 808-809, 811; R. Exh. H at 480. The fever reoccurs, in association with meningeal irritation, anywhere from 2-10 days after the initial illness symptoms end and may last 4-7 days before gradually subsiding.[(39)] R. Exh. D at 823; R. Exh. E at 146; R. Exh. F at 809. The fever may return to normal before paralysis *or while the paralysis is advancing.* R. Exh. E at 146. A higher fever (37-39ºC) is expected with aseptic meningitis and may occur with chilliness and other symptoms of meningeal irritation.[(40)] R. Exh. F at 809. The absence of fever at the onset of the neuritic symptoms is strongly suggestive of GBS; whereas, the presence of fever is a variant.[(41)] R. Exh. C at 1456. Incidently, The Merck Manual of Diagnosis and Therapy defines "fever" in infectious diseases as a "body temperature >37.8 C (100 F) orally or 38.2 C (100.8 F) rectally." The Merck Manual of Diagnosis and Therapy 6 (15th Ed. 1987).

Petitioner complained of a 1-2 week history of low grade transient fever and night sweats prior to her hospitalization on January 1, 1996. M.R. III at 17, 23, 26. Her temperature was recorded on December 19, 1995, at 98.0ºF and at 98.4ºF on December 25th and December 26th. M.R. II at 64, 89, 94. Petitioner's records indicate various temperature readings following her admission. For instance, on January 1, 1996, petitioner had a temperature of 98.8ºF and 99ºF and 37ºC and 37.4ºC. M.R. III at 16, 17, 219. Petitioner's temperature on January 2nd varied between 36.2ºC and 37.6ºC. M.R. III at 23 ("afebrile"), 27, 219, 249. Dr. Redfield noted on January 2, 1996, that fever co-existed with petitioner's progressive asymmetrical weakness. M.R. III at 34. Petitioner was listed as afebrile on numerous occasions thereafter,[(42)] but notably demonstrated, in line with the literature describing aseptic meningitis, a temperature equaling or exceeding 37ºC on January 3rd and January 5th through January 8th. M.R. III at 253, 260, 268, 274, 279. The records also indicate petitioner was self-medicating with Tylenol on December 25, 1995, and January 1, 1996. M.R. II at 89; M.R. III at 16. Petitioner was prescribed Tylenol along with other medications on January 1st and 2nd. M.R. III at 183, 187.

Aches and Pains

Both experts agreed, and the literature supports, that aches and pain may be a presenting complaint of

poliomyelitis. Tr. at 27, 246. Dr. Redfield associated headaches and neck pain and muscle soreness with the onset of aseptic meningitis, the second phase of poliomyelitis. Tr. at 28. He submitted petitioner initially complained of headache and neck and back pain. Tr. at 42; Dr. Redfield's Rpt. at 3. Petitioner's headache worsened over 1-2 weeks, culminating in a severe headache, in late December 1995, accompanied by left arm weakness. Dr. Redfield's Rpt. at 2.

Headaches and muscle pain occur in 4-8% of the polio cases, may last a few hours to 2 days, are not clinically distinguishable from symptoms in other illnesses, and the headaches become increasingly worse in the aseptic meningitis phase. R. Exh. E at 142-143, 146; R. Exh. F at 808-809. Muscle soreness or spontaneous muscle pain, most commonly in the neck and back, is specifically associated with aseptic meningitis; the pain may also be in one or more muscles in the neck, lumbar, flank area, abdominal region and/or limbs. R. Exh. E at 146; R. Exh. F at 809. In GBS cases, 30-55% of the patients experience muscular or neuropathic pain which "may be the presenting complaint and precede the onset of weakness by 1-2 days or, rarely, by several days." R. Exh. C at 1454 (footnotes omitted). The location of the pain is similar to polio and may be in the thighs, buttocks, and low back. R. Exh. C at 1454. GBS patients may also complain of diffuse headaches at onset. R. Exh. C at 1454.

Petitioner experienced headaches one to two weeks preceding her admission which worsened in the left occipital region shortly before her hospitalization on January 1, 1996, and continued throughout her stay. M.R. II at 64; M.R. III at 16, 17, 20, 26, 28, 30, 32, 34, 35, 41, 45, 50, 52.[(43)] Dizziness and unsteadiness initially accompanied the headaches. M.R. II at 89, 94; M.R. III at 26, 32. Petitioner also experienced muscle pain (myalgia) and neck and back pain 1-2 weeks prior to her hospitalization which continued after her admission. M.R. III at 16, 17, 26, 35, 41, 45, 48, 50, 52, 54, 59, 61, 64. Lastly, petitioner had a recent history of chest pain. M.R. III at 21, 30, 32.

Meningismus (Neck Stiffness)

The experts agreed neck stiffness is a symptom of aseptic meningitis. Tr. at 33, 246. Dr. Redfield reported that petitioner initially complained of back and neck stiffness in late December 1995, following her 1-2 week history of fever, sweats, loss of appetite, abdominal discomfort, and diarrhea; Dr. Redfield noticed the meningismus in petitioner's initial exam. Dr. Redfield's Rpt. at 2-3; Tr. at 42, 198.

Neck stiffness is a symptom of meningeal irritation and a signal for the onset of aseptic meningitis. R. Exh. D at 823; R. Exh. E at 142. Back stiffness may also occur with aseptic meningitis. R. Exh. F at 809. In GBS cases, neck stiffness is detected in about 10% of the cases at onset. R. Exh. C at 1454.

The medical records reveal that petitioner's admitting complaint included neck stiffness; evaluations by both Dr. Redfield and another consulting physician were positive for meningismus. M.R. III at 16, 24. Petitioner's meningismus continued from her admission date at least until January 8, 1996.[(44)] M.R. III at 16, 28, 32, 34, 41, 45, 49, 50.

Cerebrospinal Fluid Results

Both poliomyelitis and GBS diagnoses rely on an examination of three features of cerebrospinal fluid: the presence of white blood cells (*i.e.*, pleocytosis[(45)]) and polymorphonuclear leukocytes in the fluid and the level of protein achieved.

Pleocytosis: Dr. Redfield testified that pleocytosis is a classic presentation of aseptic meningitis, and

classified a *normal* white blood cell count as equal to or less than 5 cells in the CSF, *mild* as equal to or less than 50 cells, and *moderate* as 50-100 cells. Tr. at 33, 195. In contrast, he testified pleocytosis is absent or normal (*i.e.*, ≤5 cells) in GBS cases and only in 10% of the cases or less would one see the presence of cells. Tr. at 37, 89, 90, 165, 167. Dr. Arnason agreed pleocytosis occurs in polio, but expected a range of 50-250 cells based on the literature. Tr. at 227; R. Exh. A at 1. He reported that most GBS cases exhibit less than 10 cells in the CSF, but opined a count reaching 50 would still support a GBS diagnosis if other features were consistent with the illness. R. Exh. A at 2. Dr. Redfield claimed petitioner suffered from mild pleocytosis, which developed prior to her paralysis, consistent with her aseptic meningitis presentation. Tr. at 42, 81, 195.

The literature supports a finding of pleocytosis in aseptic meningitis which develops in the period before the paralytic onset. R. Exh. E at 144, 146; R. Exh. F at 809. However, no specific range for pleocytosis in polio is provided. In the court's review of the CDC's criteria for reporting, "[t]he CSF usually contains an increased number of leukocytes--from 10 to 200 cells/mm³ (primarily lymphocytes)."[(46)] D. Rebecca Prevots, PhD, MPH, Linda Quick, MD, MPH, Peter Strebel, MBChB, MPH, and Roland Sutter, MD, MPH & TM, Chapter 10: Poliomyelitis (visited Sept. 29, 1998) <http://www.cdc.gov/nip/manual/poliomye/poliomye.htm>.[(47)] In GBS cases, pleocytosis is absent or minimal. R. Exh. C at 1453; R. Exh. F at 811. Per Dr. Arnason's article, a count *strongly supportive* of a GBS diagnosis is ≤ 10 mononuclear leukocytes, or white blood cells, per cubic millimeter of fluid, although a variant still permitting diagnosis is 11-50 mononuclear leukocytes. R. Exh. C at 1456. In most cases, there are few if any lymphocytes, although a few GBS patients will reach 20-30 cells/mm³. A result casting doubt on a GBS diagnosis is >50 mononuclear leukocytes. R. Exh. C at 1456.

Petitioner's CSF results dated January 1, 1996, show a white blood cell count of 9 and 15 resulting from two separate samples which were described in the Discharge Summary as predominantly mononuclear. M.R. III at 17, 79. Dr. Redfield's January 2nd consultation report characterized the pleocytosis as mild. M.R. III at 26. The cell count of 15 falls within the CDC's accepted range, and outside of Dr. Arnason's "strongly suggestive" range for GBS; accepting Dr. Arnason's opinion requires the court to treat petitioner's illness as a *variant* case of GBS.[(48)]

Protein: Dr. Redfield testified a mild or minimal increase in the protein level is a classic presentation of polio-associated aseptic meningitis. Tr. at 33, 37, 165. In contrast, the experts agreed the protein level is usually normal in the first week in GBS cases but increases thereafter. Tr. at 38, 39, 226-227; R. Exh. A at 2. Dr. Redfield elaborated the level in GBS usually increases to 100 or more "later in the course" when "the patient is near maximally affected . . . after the weakness is fully developed, or as the weakness is fully developing." Tr. at 37, 165-166. He submitted that petitioner experienced a slight elevation later in her clinical course. Tr. at 82; Dr. Redfield's Rpt. at 2. He noted the CSF was examined on January 1, 1996, upon petitioner's admission, approximately seven days after the onset of her symptoms on or about December 25-26, 1995. Tr. at 166. Dr. Arnason opined petitioner's CSF protein results were "on the margin . . . taken very early in the course"; therefore, the lack of "albumino-cytologic dissociation is not unexpected."[(49)] Tr. at 226; R. Exh. A at 2.

The literature supports a moderate, slight, or minimal elevation in the protein level of a polio patient which may persist 2-3 weeks into the illness. R. Exh. E at 144, 146; R. Exh. F at 811. Again, the literature fails to provide a specific range for the protein elevation. In the court's review of the Centers for Disease Control and Prevention's criteria, "[t]he CSF usually contains . . . a mildly elevated protein, from 40-50 mg/100 ml." D. Rebecca Prevots, PhD, MPH, Linda Quick, MD, MPH, Peter Strebel, MBChB, MPH, and Roland Sutter, MD, MPH & TM, Chapter 10: Poliomyelitis (visited Sept. 29, 1998) <http://www.cdc.gov/nip/manual/poliomye/poliomye.htm>.[(50)] With severe paralysis, the protein level may further elevate to 100-300mg/dl. R. Exh. E at 146. In GBS cases, a strongly suggestive protein level

is one which elevates after the first week of illness, may continue to rise even as the patient stabilizes, and peaks at 4-6 weeks after the onset of the clinical symptoms. R. Exh. C at 1456-1457.

Petitioner's spinal tap results dated January 1, 1996, revealed a slightly or mildly elevated protein level of 51, just outside the CDC's range of 40-50. M.R. III at 17, 20, 79. The test was conducted approximately a week after petitioner's complaints began on December 25-26, 1995, and arguably during a time frame and with a result consistent with either diagnosis.

Polymorphonuclear leukocytes: Dr. Redfield testified the presence of polymorphonuclear leukocytes ("polys") in the cerebrospinal fluid indicates inflammation and is associated with aseptic meningitis. Tr. at 39, 195. Dr. Redfield also opined the presence of polys is rare in GBS cases. Tr. at 40, 91, 103, 195. Dr. Arnason admitted the presence of polymorphonuclear leukocytes in a GBS case "would force one to rethink" the diagnosis. Tr. at 227. However, he ignored their presence in petitioner's case and insisted her course fit within a GBS diagnosis. Tr. at 227.

The literature states polys are predominant in the early stages of polio, but persist only for a few days. R. Exh. E at 146; R. Exh. H at 486. However, the presence of polymorphonuclear leukocytes *casts doubt* on a GBS diagnosis per Dr. Arnason's article. R. Exh. C at 1456.

Petitioner's CSF results dated January 1, 1996, demonstrated the presence of polymorphonuclear leukocytes at 43%. M.R. III at 79.

Paralysis, Generally

Dr. Redfield testified paralysis in polio is characteristically unpredictable, can extend beyond 3-4 days, and may persist after fever suppression. Tr. at 24, 28, 29, 31-32, 86, 200; *compare* Tr. at 145. He contrasted this with GBS, where the weakness spreads successively over a period of up to 2 weeks; or in the alternative, develops and becomes complete after a variable period but definitely by 4 weeks. Tr. at 36, 88. Dr. Redfield explained the weakness in GBS cases is often associated with sensory loss; although a variant form of the illness can be similar to poliomyelitis, presenting with pure motor paralysis and weakness. Tr. at 35, 197. Dr. Redfield noted petitioner's admitting exam revealed left arm and right leg weakness which worsened throughout the day, over a 6-8 hour period. Tr. at 43; Dr. Redfield's Rpt. at 2-3. He further related petitioner's paralysis began 2-5 days after the onset of her illness, on or about January 2, 1996, and reached its maximum by January 4, 1996, *i.e.*, within 3-4 days, as the literature corroborates. Tr. at 81, 88, 112, 113. Dr. Arnason testified he would look for muscle twitching to diagnose polio and considered the period of worsening in polio relatively brief, also citing 3-4 days. Tr. at 246; R. Exh. A at 1.

One to two percent of polio patients suffer from paralysis which is often preceded by fever and a minor illness. R. Exh. D at 822-823. Patients may have cramping, muscle pain, spasms, and twitching. R. Exh. D at 823. The paralysis is usually developed within a few days (*e.g.*, 2-5 days) after its onset, and its extent and rapidity are highly variable. R. Exh. D at 823; R. Exh. E at 146; R. Exh. F at 809. In GBS cases, weakness is a major complaint although the severity of the motor weakness varies. R. Exh. C at 1452, 1456. The weakness may affect both motor and sensory parts of the peripheral nerve system. R. Exh. C at 1447. The weakness worsens over a period of days to weeks, but is complete in 90% of the cases within 4 weeks of the onset of the symptoms. R. Exh. C at 1449, 1452.

Petitioner was admitted January 1, 1996, complaining of numbness in the left arm, left facial region, left tongue, and knee. M.R. III at 16. Petitioner's symptoms began December 31, 1995, with persistent left hand tingling, unsteadiness, and right hand dysesthesia.[(51)] M.R. III at 20, 23, 26. The records reflect petitioner described a history of "rubbery" legs or low extremity weakness for two weeks preceding her

admission, although petitioner did not recall making this complaint. M.R. III at 20; Tr. at 206. Petitioner's limb strength and stretch were normal upon initial exam. M.R. III at 17, 21. Dr. Romine's January 2, 1996 examination revealed mild or moderate weakness proximally in petitioner's arms and legs, left greater than the right, and asymmetrical progression of bulbar paresis and quadriparesis. M.R. III at 31.

Asymmetrical or Symmetrical Nature of the Paralysis

The experts hotly debated the symmetry of petitioner's paralysis. Both agreed polio characteristically manifests itself *asymmetrically*, but disagreed on the extent of limb involvement generally and in petitioner's case. Tr. at 29, 86, 155, 239; R. Exh. A at 1. Dr. Redfield opined the paralysis presents variably, affecting one or all limbs; in the former instance, it typically affects one limb versus the other, or one portion of the limb but not the other parts. Tr. at 29, 155. Dr. Arnason opined that polio *more often* involves one limb, rather than both. Tr. at 239. Although he ultimately conceded that all four limbs may be affected in unusual circumstances, he nevertheless concluded four limb involvement more likely suggested GBS. Tr. at 222, 225, 239. Dr. Redfield opined that GBS patients present with ascending, *symmetrical* weakness. Tr. at 36, 86; Dr. Redfield's Rpt. at 6. Although he recognized, as Dr. Arnason testified, that symmetry is seldom absolute in GBS cases, Dr. Redfield offered, not entirely in conflict with Dr. Arnason's testimony, that the weakness affects both limbs. Tr. at 90, 225. Dr. Arnason offered that marked asymmetry suggests a diagnosis other than GBS. Tr. at 221. In assessing petitioner's case, Dr. Redfield vehemently maintained that petitioner demonstrated, as determined from daily tests, strikingly *asymmetrical* and *not ascending* paralysis. Tr. at 74, 75, 78, 87, 92; Dr. Redfield's Rpt. at 5 (criticizing Dr. Quick's bilateral characterization of petitioner's paralysis as misleading). While all four limbs were involved, Dr. Redfield related a 40-50% difference in one muscle group versus the other. Tr. at 156. He explained his conclusion of asymmetry:

[w]hat impressed me the most, was that there was a striking asymmetry to the weakness. Left side of face was weak, right side really was not that weak. Left arm was weak and she couldn't lift the arm at the shoulder. The elbow was a little better. The wrist was a little better. The hands were weak. The right arm was relatively much less involved . . . She could move it. Same thing on the leg . . . the right leg was profoundly weak, especially the so-called muscle groups near the trunk and the left was much, much lesser involved . . . she would have trouble, say, lifting her leg from the bed on the right, but she could lift it on the left. She could push with her foot on the left, but not on the right.

Tr. at 74-75. He further argued petitioner's clinical symptoms supported a polio finding, even if one accepts asymmetrical weakness is not atypical for GBS. Tr. at 78, 117. In Dr. Arnason's interpretation, bolstered by his reliance on the nurse's notes allegedly suggesting symmetrical paralysis worsening at day 9 or 10, petitioner's paralysis significantly involved all four limbs and was not asymmetrical. Tr. at 222; R. Exh. A at 2.[(52)]

The literature supports the experts' testimony that bilateral paralysis in all four limbs is possible in polio and GBS, but distinguishes the two diagnoses based on the degree of symmetry expressed. As the experts agreed, asymmetrical paralysis is the most characteristic feature of polio. R. Exh. F at 809. In line with Dr. Redfield's testimony, paralytic polio may affect some muscle groups, but not others, and any combination of limbs, including all four extremities; although one leg then one arm or all extremities is the most common. R. Exh. F at 809. As the experts' conversely testified, the weakness is usually symmetrical in GBS, affecting both limbs, although the symmetry itself is seldom absolute. R. Exh. C at 1452, 1456. In GBS, the weakness is usually first evident in the legs, but may begin in the arms, and

rarely as facial diplegia.[(53)] R. Exh. C at 1452. The weakness is further ascending. R. Exh. F at 811. Marked persistent asymmetrical weakness *casts doubt* on a GBS diagnosis. R. Exh. C at 1456.

Petitioner's medical records, including numerous evaluations by Dr. Redfield and Dr. Romine (a neurologist), chronicle asymmetrical, and at times bilateral, paralysis throughout petitioner's hospitalization. M.R. III at 16, 28, 31, 33, 35, 37, 41, 45, 50, 54, 247, 256. This asymmetrical characterization of petitioner's paralysis consistently appears in the records, despite notations of changes in the degree of weakness, and despite Dr. Arnason's suggestion that the nurses' Neurological Assessment Records reveal a more symmetrical interpretation. Incidentally, numerous nurses completed the Neurological Assessment Records and the records only ask for a description of petitioner's motor responses in limited terms, inquiring simply whether the motor responses are "strong," "weak," or "unable." M.R. III at 221-228. In assessing the ability of petitioner to separately raise either leg off the bed, the assessment choices are slightly more detailed: "Raises, briefly holds," "Raises, unable to hold," or "Unable to raise." M.R. III at 221-228. Dr. Redfield testified he had not reviewed the nurses' assessments, but again avowed that petitioner experienced a 40-50% difference between each limb's degree of weakness. Tr. at 160, Moreover, Dr. Redfield asserted that he would rely on the physician's findings where conflicts arose between the nurses' and doctor's assessments on evaluations conducted the same day. Tr. at 158, 160. Lastly, the court was unable to find any medical records specifically or consistently describing petitioner's weakness or paralysis as "symmetrical."

Progression and Peaking of Neurological Deficits

Dr. Redfield testified that polio is identified classically by rapid onset and progressive worsening of neurological deficits which peaks within days of the onset, halts, and then improves. Tr. at 29-30, 71, 86, 198. Dr. Arnason described the period of worsening as lasting 3-4 days, and opined one would look for events happening within hours or days, such as a limb progressing from full strength to complete paralysis over hours or a day or so. R. Exh. A at 1; Tr. at 246. Dr. Redfield contrasted this clinical picture with GBS, wherein the weakness classically evolves slowly, over days to weeks, but is complete by four weeks. Tr. at 35, 36, 71, 117. Dr. Redfield noted the literature accepts that a variant form of GBS may progress rapidly, over a few days, but he emphasized the symmetrical and distal nature of the weakness in such a case. Tr. at 197, 198. Dr. Redfield ardently testified petitioner's weakness rapidly progressed within hours, peaked by the third hospital day, and reached maximum paralysis by January 4, 1996. Tr. at 42, 47, 68, 71, 74, 113, 114, 149, 154; Dr. Redfield's Rpt. at 3. She thereafter exhibited no new complications. Tr. at 69. Dr. Redfield corrected his report in the records that petitioner's paralysis peaked between days 7 and 9; he offered that he failed to read his records carefully before preparing his report and the medical records show petitioner began to improve on January 5, 1996. Tr. at 114. Dr. Redfield testified this peak could still occur at day 7 through 9 and be consistent with polio, since in GBS the illness progresses over a few weeks. Tr. at 116, 117. Dr. Arnason added little to this issue, stating simply that petitioner's condition progressed too slowly to be polio. R. Exh. A at 2.

The literature supports that a polio patient's paralysis reaches maximum involvement typically within 2-5 days of the onset of the paralysis, although the rapidity is variable and occasionally cases progress from weakness to complete paralysis within a few hours. R. Exh. D at 823 (within a few days); R. Exh. E at 146 (3-5 days); R. Exh. F at 809 (2-3 days), 811 (3-4 days). Progression of the paralysis after the passage of this time frame is unusual[(54)], and the paralysis may halt with an afebrile condition or advance while the temperature is returning to normal. R. Exh. E at 146; R. Exh. F at 809, 811. In GBS cases, the worsening occurs over a period of days to weeks. R. Exh. C at 1449. Fifty percent of GBS cases have completely evolved by 2 weeks, 80% by 3 weeks, and 90% by 4 weeks. R. Exh. C at 1452, 1456.

Petitioner's Walk-In Patient history, dated January 1, 1996, describes petitioner's complaints of headache, left arm numbness, face twitching, left facial and tongue numbness, back ache, knee numbness, difficulty

swallowing, and mild neck stiffness. M.R. III at 16. Petitioner was thereafter admitted on January 1, 1996. By January 2, 1996, petitioner developed an increase in speech slurring and arm and leg weakness. M.R. III at 31. Dr. Romine described petitioner's condition on day two of her hospitalization as one of "asymmetrical progressive bulbar paresis and [q]uadraparesis." M.R. III at 31. Dr. Redfield also noted the worsening of petitioner's condition. M.R. III at 33-34. Petitioner's weakness continued on January 3rd; she was unable to elevate her arms and barely able to lift her legs from the bed. M.R. III at 35, 37. On January 4, 1996, Dr. Redfield described petitioner's condition as "becoming more stable" with no new complications. M.R. III at 41. This is in contrast to Dr. Romine's entry in the notes the same day indicating petitioner's "condition continues to worsen re: muscular function." M.R. III at 43. Thereafter, Dr. Redfield noted a slight increase in petitioner's left facial and limb weakness on January 5th--day 5 of her hospitalization; in contrast again, Dr. Romine expressed his belief that petitioner was "slightly stronger today in face, extremities, tongue same . . . hopefully she has reached maximum deficit at this time." M.R. III at 45, 47. Dr. Redfield's January 6th entry notes petitioner's condition is the same and states she "should stabilize this weekend." M.R. III at 49. Petitioner's course remained stable January 7th, but Dr. Redfield noted a possible slight increase in facial and upper extremity weakness on the 8th and the 9th. M.R. III at 50, 53, 54-55. By January 10th, Dr. Redfield noted no new complications and an increase in petitioner's right arm and lower extremity power; he assessed petitioner's syndrome was regressing. M.R. III at 57-58. Both treaters noted petitioner's slight improvement and more stable condition on January 11th. M.R. III at 59. The records, however conflicting, may be read to conclude, and therefore support either diagnosis, that petitioner's condition peaked somewhere between January 4th and 5th, followed by a stable course for two days until slight changes in petitioner's weakness on the 8th and 9th, with a return to a stable and continuously improving condition on January 10th and 11th, and thereafter.[(55)]

Proximal Versus Distal Muscle Involvement

The experts did not specifically contest the location of petitioner's weakness in the proximal or distal muscles. Dr. Redfield found petitioner's proximal muscles (those closest to the trunk) more affected, as expected with polio. Tr. 30, 42; Dr. Redfield's Rpt. at 4. Dr. Redfield expected GBS patients to present with both distal and proximal muscle involvement, with the involvement commencing in the feet and ascending both sides symmetrically. Tr. at 36; Dr. Redfield's Rpt. at 6. Dr. Arnason disagreed slightly, stating that while it is more common for GBS's onset to begin in the legs rather the arms, and more distally than proximally, variations exist. Tr. at 253-254.

The literature confirms polio usually affects the proximal muscles and GBS the distal muscles, but recognizes that both muscle groups may be affected in GBS, where the proximal weakness may predominate more frequently at the onset of the illness with distal muscle involvement becoming more predominant later in the course. R. Exh. C at 1447, 1452; R. Exh. F at 809.

Dr. Romine entered in his January 2, 1996 notes that petitioner suffered from proximal weakness; no specific reference to distal involvement is mentioned in the records. M.R. III at 31.

Swallowing and Oral Secretion Problems

Dr. Redfield opined patients with bulbar polio, which affects the nerves controlling the head muscles, classically present early in the illness with difficulty swallowing and handling oral secretions. Tr. at 30, 31. Dr. Redfield noted petitioner presented initially with tongue and swallowing problems, which progressed to drooling and speaking difficulties. Tr. at 32, 42, 77; Dr. Redfield's Rpt. at 2, 3. Dr. Arnason submitted swallowing and secretion problems are a feature of polio and *alert a physician to this diagnosis*; however, he neglected to address petitioner's complaints, stating simply that bulbar involvement may occur in a *variant form* of GBS. Tr. at 246-247; R. Exh. A at 1. Dr. Redfield opined

cranial nerve involvement in GBS cases only occurs after the onset of limb weakness. Dr. Redfield's Rpt. at 6.

The literature states bulbar involvement occurs in 6-25% of polio cases, and depending on the cranial nerves affected, may result in problems with swallowing, secretion pooling, and chewing, as well as weakness in the forehead, cheek, and lip muscles. R. Exh. D at 823; R. Exh. F at 810. Dr. Arnason's own literature states chewing and swallowing are only affected in *severe* GBS cases, and a *variant* form of the illness may involve dysarthria. R. Exh. C at 1453, 1456.

Petitioner complained upon admission that she developed difficulty moving her tongue on December 31, 1995, while attempting to eat. M.R. III at 20. On January 1, 1996, petitioner complained of left tongue weakness and difficulty swallowing, which worsened on January 2, 1996, accompanied by marked dysarthria and drooling. M.R. III at 16, 23, 26, 31, 32, 33. Petitioner's problems continued in the week following her admission; her speech began to improve, with therapy, on January 11, 1996, and thereafter. M.R. III at 35, 37, 41, 42, 43, 45, 47, 51, 53, 55, 59, 61, 64, 70.

Facial Weakness

The experts testified consistently with the literature that facial diplegia, while uncommon or even rare in polio, occurs in 50% of GBS cases.[(56)] Tr. at 86, 90, 137, 138, 142, 223, 254. However, both advanced that some polio patients will experience facial involvement; Dr. Redfield described this as unilateral or one-sided facial weakness. Tr. at 86, 137, 228. Dr. Redfield further accepted, as stated in Dr. Arnason's article, that some GBS patients also experience unilateral facial weakness, while those presenting bilaterally demonstrate asymmetric weakness. Tr. at 141. While the experts testified consistently with each other and the literature regarding facial weakness generally in polio and GBS, they disagreed specifically on the characterization of petitioner's facial involvement. Dr. Redfield rejected the medical records citing facial diplegia and contrasted them with others recording only left-sided facial weakness. Tr. at 87, 137, 138, 140, 142; Dr. Redfield's Rpt. at 2, 3. He further submitted petitioner had ptosis, an uncommon feature of GBS, and Dr. Romine, despite his notations of facial diplegia, affirmed petitioner's poliomyelitis diagnosis. Tr. at 137, 141. Dr. Arnason relied simply on Dr. Romine's notations to argue petitioner experienced symmetrical bilateral facial weakness. Tr. at 222, 228. However, Dr. Arnason's opinion is weakened by his own article which states bilateral facial weakness in GBS presents asymmetrically, not symmetrically, and by his concession that a finding of facial diplegia in petitioner's case would deem her case *rare* for GBS. R. Exh. C at 1452; Tr. at 255.

Fifty percent of polio cases will exhibit facial nerve paresis with weakness in the forehead, cheek, and lips; however, facial diplegia is very uncommon, even in bulbar polio cases. R. Exh. F at 810, 811. Dr. Arnason's medical literature, in addition to supporting the experts' testimony generally and asserting GBS weakness *rarely* begins with facial diplegia, further notes ptosis is *uncommon* in GBS, and *less than 5% of the cases* involving facial weakness begin with the tongue. R. Exh. C at 1452, 1456.

The medical records provide a mixed, and thus unclear, description of petitioner's facial weakness. At times Dr. Romine characterizes the weakness as "severe facial diplegia," "moderately severe bilateral facial weakness," or simply "bilateral facial . . . weakness." M.R. III at 31, 59; M.R. IV at 24. Dr. Redfield uses the term "diplegia" on several occasions, but the records are also replete with notations that petitioner's weakness is left-sided or left-sided more than right. M.R. III at 16, 28, 31, 35, 45, 53, 54, 246. The records further describe the weakness as both asymmetrical and symmetrical. M.R. III at 17, 21, 28. Lastly, and perhaps more importantly, ptosis is recorded, and the records reveal, as noted above, that petitioner initially presented with tongue weakness. M.R. III at 20, 35, 41, 45.

Sensory Disturbances and Paresthesia

Dr. Redfield agreed with the literature that sensory loss in polio is rare and would suggest another diagnosis, such as GBS; however, he clarified polio patients not infrequently report episodes of paresthesia, although the objective test reveals intact sensation.[(57)] Tr. 86, 88, 125, 126, 127, 131, 161, 200. In contrast, 80% of GBS patients exhibit some sensory loss, although a variant form of the illness can mimic poliomyelitis and lack sensory findings. Tr. at 35, 86, 125, 132, 197, 200. Dr. Arnason testified sensory symptoms, including "pins and needles" and tingling, are more indicative of GBS, but conceded "that an occasional [polio] patient will complain of subjective sensory things at the site where the paralysis is developing is documented as not common but happens." Tr. at 225, 228, 229. Dr. Redfield denied petitioner suffered loss of sensation as evident from her examinations. Tr. at 79, 87, 131, 132. While he acknowledged petitioner reported left hand tingling in late December, he did not recall paresthesia being a prominent complaint and noted a same day examination revealed intact sensation. Tr. at 127, 163, 164; Dr. Redfield's Rpt. at 2. Dr. Arnason simply supplied that petitioner's documented sensory symptoms "fit better with GBS." Tr. at 225.

The literature reports that sensory disturbances, while rare in polio, exist in 80% of GBS cases; paresthesia, however, while uncommon in polio, is nevertheless occasionally seen in the pre-paralytic major illness. R. Exh. F at 809, 811. GBS patients may describe subjective tingling, numbness, and burning, although the objective examinations reveal the sensory loss to be less than that subjectively reported, and sensory disturbances may be absent in the early course. R. Exh. C at 1453. A finding of a mild sensory symptoms or signs strongly suggests GBS; a sharp sensory level casts doubt on the diagnosis. R. Exh. C at 1456.

The medical records consistently document intact sensation throughout petitioner's hospitalization, even though the records may be internally inconsistent. M.R. III at 21, 24, 35, 41, 248, 297, 301. For example, one record reports "sensory, decreased sensation subjectively" on January 1, 1996, while another notes paresthesia, in the form of left hand tingling and "pins and needles," on the same date, but Dr. Romine's January 1st examination revealed intact sensation in petitioner's hands, feet, and face. M.R. III at 16, 20, 21. In an other instance, Dr. Romine's January 2, 1996 entry records a decrease in sensation, but Dr. Redfield's notes that same day show petitioner's sensation to be intact and preserved. M.R. III at 31, 34.

Loss of Reflexes

Both experts reported that petitioner ultimately experienced a loss of reflexes. Tr. at 42; Dr. Redfield's Rpt. at 2; R. Exh. A at 2. Dr. Arnason acknowledged polio patients lose reflexes in the areas involved. Tr. at 247.

The literature fails to adequately distinguish this symptom's role in either diagnosis. Polio patients suffer from diminished or absent tendon reflexes; the stretch reflexes are initially hyperactive but then become absent. R. Exh. D at 823; R. Exh. F at 809. Similarly, the tendon reflexes of affected areas are abolished in GBS patients, usually from the onset of the illness, with slight activity possibly occurring in mild cases and complete retention occurring rarely. R. Exh. C at 1452. A GBS diagnosis requires a finding of areflexia (loss of tendon jerks). R. Exh. C at 1456.

Petitioner's records reveal a subjective decrease in the deep tendon reflexes on January 1, 1996, and a finding of intact stretch reflexes. M.R. III at 16, 17, 21. On January 2, 1996, petitioner's deep tendon and muscle stretch reflexes were intact, but the left upper extremity was reportedly slightly diminished more than the right. M.R. III at 24, 31, 34. Dr. Redfield's January 3rd exam showed a decrease in petitioner's deep tendon reflexes; Dr. Romine recorded absent or trace muscles stretch reflexes. M.R. III at 35, 37. By January 4, 1996, petitioner's muscle stretch reflexes were absent and by the 6th, she had trace deep tendon reflexes. M.R. III at 43, 49. Dr. Romine's January 11, 1996 entry cites areflexia. M.R. III at 59. In

short, petitioner's deep tendon and muscle stretch reflexes gradually diminished.

Electromyography and Nerve Conduction Velocity Results

Electromyography: Dr. Arnason testified that GBS patients exhibit, not infrequently, a normal EMG in the first week of illness. Tr. at 236. In the second week, the EMG characteristically demonstrates widespread denervation; an abnormal EMG is consistent with GBS. Tr. at 236. Dr. Arnason noted petitioner's first normal EMG revealed little in the way of diagnosis; however, her second EMG showed widespread denervation consistent with GBS. Tr. at 236; R. Exh. A at 2. Dr. Redfield admitted he was unfamiliar with the second EMG and unable to interpret that study given his lack of qualifications. Tr. at 188. The literature states abnormalities in routine EMG studies in GBS patients include: "(1) multifocal conduction block; (2) markedly slowed nerve conduction velocities (NCV) with prolonged distal and F-wave latencies; and (3) varying degrees of denervation." R. Exh. C at 1463.

Dr. Romine's January 4, 1996 Electromyogram report states: "Except for minimal prolongation of the right median sensory latency, nerve conduction studies and repetitive stimulation study of the ulnar and facial nerves are normal. Specifically, there is no evidence to indicate diffuse polyneuropathy or abnormality of neuromuscular transmission." M.R. II at 205. Petitioner's second EMG, conducted by January 16, 1996, was described as abnormal, "with findings of widespread acute and chronic denervation with early signs of reinnervation consistent with a disease of lower motor neurons." M.R. III at 70.

Nerve Conduction Velocity: Dr. Redfield testified normal nerve conduction velocity studies are consistent with poliomyelitis. Tr. at 48, 72. He further noted petitioner's NCV was conducted at her maximal weakness with normal findings and Dr. Romine determined the result consistent with polio and inconsistent with GBS. Tr. at 47-48, 72, 92. In contrast, Dr. Redfield opined, the NCV results should be abnormal in GBS. Tr. at 48, 72. Dr. Arnason testified, in line with his article and the only literature on this issue, that about 80% of GBS patients demonstrate evidence of nerve conduction slowing or blockage during the course of the illness. Tr. at 91; R. Exh. C at 1456. The literature further states that "[c]onduction studies [in GBS] may not become abnormal until several weeks into the illness" and abnormalities in routine NCV studies in GBS patients include: "(1) multifocal conduction block; (2) markedly slowed nerve conduction velocities (NCV) with prolonged distal and F-wave latencies; and (3) varying degrees of denervation." R. Exh. C at 1456, 1463. Lastly, three of four features are required in a NCV test (with predominant process of demyelination) to diagnose GBS:

1. Reduction in conduction velocity in two or more motor nerves . . . 2. Conduction block or abnormal temporal dispersion in one or more motor nerves: either peroneal nerve between ankle and below fibular head, median nerve between wrists and elbow, or ulnar nerve between wrist and below elbow . . . 3. Prolonged distal latencies in two or more nerves . . . 4. Absent F-waves or prolonged minimum F-wave latencies.

R. Exh. C at 1457.

Petitioner's January 4, 1996 medical records reflect that Dr. Romine considered petitioner's nerve conduction velocities normal; therefore, no evidence existed for a GBS diagnosis. M.R. III at 43. However, the medical records are not specific enough for this court to independently contrast and weigh the information with the criteria outlined in Dr. Arnason's article.

Laboratory Test Results

The experts agreed an unequivocal acute polio diagnosis requires a four-fold increase in the antibody

titer. Tr. at 50, 176, 224. However, Dr. Redfield emphasized the CDC requires neither this *nor* virus isolation to diagnose polio. Tr. at 106, 167. He opined any increase in the antibody titer, including one *two-fold*, is consistent with an acute active infection; however, he admitted he did not know whether the laboratory physician considered the change from 16 to 32 an "increase."(58) Tr. at 57, 175, 176; Dr. Redfield's Rpt. at 4. Dr. Arnason deemed the change within the test's error range and insignificant for any diagnostic or suggestive purposes. Tr. at 224, 234. Dr. Redfield agreed no change in the antibody titer suggests a past infection, but maintained his diagnosis, based on petitioner's clinical course, even if petitioner experienced no antibody rise. Tr. at 117-118, 179. He noted petitioner may have been infected with the virus for weeks prior to her admission and speculated an earlier sample could have demonstrated a four-fold increase. Tr. at 53, 57. Dr. Arnason agreed the results confirmed petitioner's exposure, *at some time*, to the Type I polio virus, but implied the exposure occurred before Samantha Cruz's oral polio vaccination; he buttressed his opinion with respondent's counsel's proffer that a Philippine study showed 100% of persons age 20 and older tested positive for the polio antibody, especially Type I. Tr. at 124, 233, 240. When asked about the strength of his opinion, Dr. Arnason admitted he could not be certain petitioner fell into the study's parameters or suffered exposure prior to her daughter's receipt of the OPV. Tr. at 240.

In assessing the laboratory tests specifically, Dr. Redfield admitted more familiarity with the neutralization antibody test, finding it simple, direct, "time honored [and a] more sensitive test to detect [the] polio antibody." Tr. at 52, 180-181. Dr. Arnason simply testified the neutralization antibodies persist for life. Tr. at 232-233. Dr. Redfield admitted he had never performed and had limited knowledge of the complement fixation test for polio; he also criticized it for its complexity and dependency on test conditions.(59) Tr. at 54, 180-181, 185. Moreover, Dr. Redfield confessed he had never seen petitioner's complement fixation results until his review of petitioner's records for trial. Tr. at 185. Dr. Arnason also expressed, but without elaboration, his inexperience with and dislike for the complement fixation antibody test. Tr. at 240. He further maintained the complement fixation antibodies remain in the body for 3-5 years. Tr. at 232-233. Dr. Redfield agreed virus isolation from fecal specimens, another test form, is most expected soon after the onset of the infection, but noted virus excretion varies and isolation decreases once antibodies appear. Tr. at 167, 170, 173. Given this, Dr. Redfield opined that the polio virus will be detected through virus isolation in only 50% of samples taken by day 15 of the illness. Tr. at 167, 169, 170, 174. Dr. Arnason simply testified the virus infects the gastrointestinal system 1-2 weeks prior to the onset of the symptoms and is shed in the feces for weeks and even months thereafter. Tr. at 235, 241. Dr. Arnason described the polymerase chain reaction test (PCR) as sensitive and powerful in detecting an enterovirus, but acknowledged it may not have been performed in petitioner's case to specifically detect polio, given the disease's rarity; Dr. Redfield did not address this test. Tr. at 230, 231, 232. Dr. Redfield acknowledged his inexperience with interpreting *Campylobacter* test results, but nevertheless criticized them (and the CDC's use of them) for their lack of standardization and inconclusiveness, claiming other infections may produce positive IgM results. Tr. at 100-101, 190. He also denounced the Vanderbilt University laboratory's role given its research pension, and therefore bias, for documenting positive *Campylobacter* results. Tr. at 100-101, 102, 190-191. Dr. Arnason simply replied the CDC relies on the lab for important data and results regarding *Campylobacter*. Tr. at 221. Generally speaking, Dr. Arnason conceded laboratory tests are not infallible. Tr. at 241.

In the end, while Dr. Redfield accepted that *Campylobacter* is a trigger for GBS and the cause of most diarrheal illnesses in the country, and even conceded the bacteria's presence here possibly suggested an earlier infection, he continually rejected it as the cause of petitioner's illness. Tr. at 98-99, 100-101, 190; Dr. Redfield's Rpt. at 5-6. Particularly, Dr. Redfield relied on petitioner's aseptic meningitis and overall clinical course, the significance of the two-fold antibody rise (per his discussion with the lab physician), and his belief that petitioner was never immunized against polio. Tr. at 45, 50, 57, 79, 96, 112, 175. He buttressed his opinion with the lack of evidence that petitioner tested positive for the polio antibody before her illness or that another non-polio enterovirus infection was present. Tr. at 102, 112. While he

opined that virus isolation is desirable, in the absence of serological data, to absolutely diagnose polio, he countered only 50% of cases result in positive outcomes. Tr. at 168. In addition, Dr. Redfield noted petitioner's virus isolation, while taken within 15 days of her paralytic onset, may not have been taken within 15 days of the onset of her illness. Tr. at 170. Moreover, he concluded petitioner had measurable antibodies, which would have inhibited virus excretion and, therefore, virus isolation. Tr. at 175. While remaining steadfast in his diagnosis, however, Dr. Redfield's conviction was weakened by two serious concessions: first, that petitioner's results, when viewed alone, were consistent with an old polio infection; and second, that repeated negative results in an immunocompetent individual are inconsistent with an acute polio infection. Tr. at 175, 179-180.(60) Dr. Arnason concluded his assessment of the laboratory data by relying on the lack of virus isolation or measurable antibody increase, the lab's findings of an elevated IgM, petitioner's episodes of diarrhea, and *Campylobacter*'s relationship with GBS. Tr. at 221; R. Exh. A at 2. While Dr. Arnason conceded that petitioner's symptoms may fit either diagnosis, even if only in rare circumstances, he nevertheless maintained her case turns on the lack of laboratory support for a polio diagnosis and the confirmatory results for a *Campylobacter* infection. Tr. at 223.

Per the literature, and as agreed by the experts, a physician traditionally looks for a four-fold rise in the antibody titer to diagnose an acute viral infection. R. Exh. D at 822; R. Exh. E at 144. A positive but unchanging titer simply means the patient had the infection in the past, and it is unlikely the virus is the cause of the presenting illness. R. Exh. E at 144. After infection with the polio virus, the virus may be present in the oropharynx for 1-4 weeks(61) and shed in the feces for 1-18 weeks.(62) R. Exh. D at 821; R. Exh. E at 146; R. Exh. F at 811. Recovery of the virus from the cerebrospinal fluid is rare in poliomyelitis. R. Exh. D at 822; R. Exh. E at 144, 146; R. Exh. F at 807, 811. Virus particles in the blood are usually undetectable once symptoms appear and virus replication terminates with the appearance of neutralizing antibodies. R. Exh. D at 822. The neutralization antibody test can detect the antibodies for years after the infection. R. Exh. D at 822. The test is considered the most specific and useful means for detection, but has been criticized as expensive, cumbersome, and requiring careful testing. R. Exh. D at 822; R. Exh. F at 811. In GBS cases, the antecedent illness of *Campylobacter* infection may be diagnosed with recovery of the bacteria from stool; while recovery seldom occurs for more than 2-3 weeks after the infection, it has occurred as late as 2 months after the onset of neuritis. R. Exh. C at 1441. Diagnosis of the infection from serologic specimens is *"less secure . . . since in older populations up to 50 per cent of individuals have serologic evidence of prior infection."* R. Exh. C at 1441 (Emphasis supplied).

None of the laboratory tests conducted on petitioner's throat, blood, fecal, or CSF specimens isolated the polio virus or tested positive for the polio antibody. A virus culture from a throat sample taken January 2, 1996, revealed "[n]o virus isolated in tissue culture in 29 days." M.R. III at 86. Two routine blood cultures completed by January 6, 1996, showed "[n]o growth 5 days." M.R. III at 85, 86. Complement fixation test results on two sera samples taken January 3rd and 16th showed the antibody was not detected. M.R. II at 186. Although the court was unable to find the records reporting the neutralization antibody test results on the January 3, 1996 serum specimen, the attachments to Dr. Weibel's report notes a figure of "16" for Type I; the February 15th sample resulted in a figure of "32" for Type I, although the significance of the apparent change or increase is debatable. R. Rpt. Attachments at RE-12. Polymerase Chain Reaction (PCR) test results from the January 3rd serum specimen stated the nucleic acid was not detected but notes: "PCR for enterovirus is not a routine diagnostic service of this laboratory. We are currently evaluating the usefulness of this experimental, research technique as a diagnostic test, but the significance of the test results has not been determined and we are not currently enrolled in a proficiency testing program that covers PCR testing for this virus." M.R. II at 187. Similarly, the Enzyme Immunoassay test completed on serum drawn January 3, 1996, for the detection of Enterovirus IgM also showed no antibody detected and noted: "This technique is currently a research procedure and has not been established as a diagnostic procedure. The presence of enterovirus IgM does not prove that an enterovirus infection is the cause of the patient's current illness." M.R. II at 187. Stool specimens taken

January 3rd and 6th for a comprehensive virus culture to detect enterovirus both returned without virus isolation. M.R. III at 87. The California Department of Health Service's Viral and Rickettsial Disease Laboratory reported on January 26, 1996, that no virus was isolated from a stool specimen; the exact date of the specimen is unclear but was given before January 10, 1996. M.R. II at 185. Not surprisingly, a CSF culture conducted by January 3, 1996, revealed "[n]o organisms seen" and "[n]o growth 2 days." M.R. III at 85. A more comprehensive viral culture review of petitioner's CSF, completed by the end of January 1996, showed "[n]o virus isolated in tissue culture in 29 days." M.R. III at 85. Petitioner's convalescent serum sample, drawn March 11, 1996, reportedly failed to detect Type I-III polio antibodies under the complement fixation test. M.R. II at 117, 190; M.R. III at 10. The neutralization antibody test results on the convalescent serum was "32" per the CDC's attachments. R. Rpt. Attachments at RE-12.

Petitioner was also tested for *Clostridium botulinum* toxin and organisms, but neither was detected. M.R. II at 1; M.R. III at 86. The records reveal a negative *Clostridium difficile* toxin screen on a January 7, 1996 stool sample. M.R. III at 88. Petitioner's serological screen for the *Campylobacter* bacteria revealed her to be above normal in 2 Ig classes for the sera drawn January 3rd and 16th. R. Rpt. Attachments at RE-5, RE-15. Although no report accompanies the results, Dr. Weibel concluded the sera "demonstrated a high titer of IgA, IgG, and IgM antibody for Campylobacter antigens consistent with a recent infection." R. Rpt. Attachments at RE-5. Despite all these results and Dr. Redfield's several references to them, he maintained his polio diagnosis throughout. M.R. III at 53, 68.[(63)]

IV. CONCLUSION

This case exemplifies the classic battle between the experts, whereby the court is assigned the task of weighing the credibility of the opinions expressed. Where the experts' medical judgments diverged, Dr. Redfield cited support for his opinions in the literature and petitioner's medical records, making his testimony more persuasive, thereby, tipping the scales in petitioner's favor. In contrast, Dr. Arnason's opinions were thinly supported by variant symptoms, and ultimately not persuasive. On the whole, the court deems Dr. Redfield more credible. For the reasons set forth above, the court finds petitioner has met her burden of proving by a preponderance of evidence that she contracted paralytic polio following the administration of the oral polio vaccine to her daughter in October 1995. The court will issue a separate Order in this matter setting forth the schedule for the determination of the compensation matters.

Gary J. Golkiewicz

Chief Special Master

1. The statutory provisions governing the Act, as amended, are found at 42 U.S.C.A. §300aa-1 *et seq*. (West 1994 and Supp. 1998). Hereinafter, for ease of citation, individual sections of the Act will be cited without reference to 42 U.S.C.A. §300aa.

2. The court will reference the medical records by volume and page number, *e.g.*, M.R. II at 3. Petitioner filed Volumes II and III on December 30, 1996. The medical records petitioner filed April 30, 1998, will be referenced as Volume IV. The medical records contained at Volume I, filed December 30, 1996,

pertain to petitioner's prior medical history and are irrelevant for the purposes of this Decision.

3. Respondent's Rule 4 Report contained a Declaration from Robert E. Weibel, M.D., summarizing respondent's medical position and attaching information and a report from the Centers for Disease Control and Prevention, which reviewed petitioner's allegation that she contracted polio. The Declaration and the attachments are designated as pages "RE-1" through "RE-16." Dr. Weibel's report is at pages RE-1 through RE-6; the CDC documentation, which was completed by Dr. Linda Quick on May 21, 1996, is at pages RE-7 through RE-16. References to this information will be cited as "Respondent's Report Attachments, filed 3/31/97, at RE-#" or in the short form "R. Rpt. Attachments at RE-#."

4. In addition to the expert reports filed and testimony provided, respondent submitted medical literature at Exhibits C through H, filed January 15, 1998, and April 3, 1998. Petitioner did not file any medical articles, but relied on respondent's literature to support her claim.

5. It is not disputed that petitioner has met the other prerequisites to compensation as outlined in the Act at §11.

6. Because the analysis of petitioner's claim requires a detailed inspection of petitioner's medical history in the Discussion portion of this decision, the Factual Background section will be abbreviated. The court will conduct an in-depth review of petitioner's symptoms and hospital care later, in the context of the medical literature and expert testimony provided.

7. Aftercare instructions, at M.R. II at 90, list the diagnoses as URI (upper respiratory tract infection) and viral infection.

8. Petitioner also complained of intermittent fevers and night sweats with anorexia in the 1-2 weeks prior to her admission. M.R. III at 23.

9. Dorland's Illustrated Medical Dictionary defines **dysarthria** as "imperfect inarticulation of speech due to disturbances of muscular control which result from damage to the central or peripheral nervous system." Dorland's illustrated medical dictionary 516 (27th Ed. 1988).

10. Dorland's Illustrated Medical Dictionary defines **dysphagia** as "difficulty in swallowing." Dorland's illustrated medical dictionary 519 (27th Ed. 1988).

11. Dorland's Illustrated Medical Dictionary defines **tachycardia** as "excessive rapidity in the action of the heart." Dorland's illustrated medical dictionary 1659 (27th Ed. 1988).

12. Petitioners must prove their case by a preponderance of the evidence, which requires that the trier of fact "believe that the existence of a fact is more probable than its nonexistence before [the special master] may find in favor of the party who has the burden to persuade the [special master] of the fact's existence." In re Winship, 397 U.S. 358, 372-73 (1970) (Harlan, J., concurring), quoting F. James, Civil Procedure 250-51 (1965). Mere conjecture or speculation will not establish a probability. Snowbank Enter. v. United States, 6 Cl. Ct. 476, 486 (Cl. Ct. 1984).

13. Sections 14(a) and 13(a)(1) respectively.

14. A reputable medical or scientific explanation does not simply mean, however, any theory that a medical expert is willing to espouse. In construing the Federal Rules of Evidence, the Supreme Court held that it is the trial judge's responsibility to ensure that "any and all scientific testimony or evidence admitted is not only relevant, but reliable." Daubert v. Merrell Dow Pharmaceuticals, Inc., 113 S.Ct.

2786, 2795 (1993); see also Vaccine Rule 8(b) (The special master is obliged to consider "all relevant, reliable evidence . . . ."). Rule 702 provides that an expert witness may testify to his "scientific, technical, or other specialized knowledge . . . ." The term "knowledge," however, "connotes more than subjective belief or unsupported speculation." Daubert, 113 S.Ct. at 2795. Thus, the expert's proposition must have been "derived by the scientific method." Daubert, 113 S.Ct. at 2795. This requires that the proponent demonstrate that there is "some objective, independent validation of the expert's methodology." Daubert v. Merrell Dow Pharmaceuticals, Inc., 43 F.3d 1311, 1316 (9th Cir. 1995) (Kozinski, J.), on remand from 113 S.Ct. 2786 (1993). Factors relevant to that determination may include, but are not limited to:

whether the theory or technique employed by the expert is generally accepted in the scientific community; whether it's been subjected to peer review and publication; whether it can be and has been tested; and whether the known potential rate of error is acceptable.

Daubert, 43 F.3d at 1316; see also Daubert, 113 S.Ct. at 2796-97. The overall touchstone is "whether the analysis undergirding the experts' testimony falls within the range of accepted standards governing how scientists conduct their research and reach their conclusions." Daubert, 43 F.3d at 1316.

Respondent's counsel argues in his Closing Brief that petitioner's expert's testimony failed to meet the requirements of Daubert. The court categorically rejects this argument. The court finds Dr. Redfield testified in accordance with Daubert's medical principles and that his opinions were, in all respects, consistent with the methods and knowledge utilized by his peers. Respondent has not shown otherwise.

15. Dorland's Illustrated Medical Dictionary defines **poliomyelitis** as "an acute viral disease, occurring sporadically and in epidemics, and characterized clinically by fever, sore throat, headache, and vomiting, often with stiffness of the neck and back. In the *minor illness* these may be the only symptoms. The *major illness*, which may or may not be preceded by the minor illness, is characterized by involvement of the central nervous system, stiff neck, pleocytosis in the spinal fluid, and perhaps paralysis." Dorland's Illustrated Medical Dictionary 1327 (27th Ed. 1988).

16. "Enterovirus-caused poliomyelitis . . . *can usually be identified* through routine methods of virus isolation and determination of specific antibody titer changes." R. Exh. D at 822.

17. "In acute enteroviral infections, the diagnosis *is most readily established* by virus isolation from throat swabs, stool or rectal swabs, body fluids, and occasionally tissues." R. Exh. D at 822.

18. "Direct isolation of virus from affected tissues or body fluids in enclosed spaces

. . . *usually confirms* the diagnosis." R. Exh. D at 822.

19. "Isolation of an enterovirus from the throat *is suggestive* of an etiologic association because the virus is usually detectable at this site for only 2 days to 2 weeks after infection." R. Exh. D at 822.

20. "The diagnosis *may be further supported* by a fourfold or greater neutralizing antibody titer increase in paired acute and convalescent serum samples." R. Exh. D at 822.

21. "In acute-poliovirus infections, complement-fixing antibody titer determinations on acute and convalescent sera *can aid in diagnosis*." R. Exh. D at 822.

22. "The diagnosis *can be made* by a combination of virus isolation (inoculation of blood, nasopharyngeal washings, feces, CSF, or tissue suspensions into susceptible animals or tissue culture systems), serologic tests, and amplification of viral nucleic acids." R. Exh. E at 144.

23. "The diagnosis of poliovirus infections *can be established* by recovery of the virus from stool . . . , throat washings . . . , or rarely from the CSF or blood." R. Exh. E at 146.

24. The court's use of this CDC source is permitted by law. See Hines on Behalf of Sevier v. Secretary of HHS, 21 Cl. Cl. 634, 644 (Cl.Ct. 1990)(the court ruled "since the Special Master was not inclined to accept [petitioner's expert's] equivocal testimony on the incubation period of measles, he was not precluded from seeking further substantiation of a medical fact, such as the incubation period of measles"). The Federal Circuit affirmed the U.S. Claims Court on appeal, ruling the special master's use of a medical textbook to determine and take judicial notice of a medical fact was not fundamentally unfair even though the parties were not informed the source would be used, and the textbook material was not presented at hearing. The court found persuasive that petitioner failed to discredit or rebut the textbook's information on appeal to the U.S. Claims Court and that the special master based his decision on a number of factors and the textbook's information was not critical to his decision. See Hines on Behalf of Sevier v. Secretary of HHS, 940 F.2d 1518 (Fed. Cir. 1991).

25. Incidently, the World Health Organization's standard definition for a confirmed case of poliomyelitis is "acute flaccid paralysis (AFP) and at least one of the following: (1) laboratory-confirmed wild poliovirus infection, (2) residual paralysis at 60 days, (3) death, or (4) no follow-up investigation at 60 days." *Progress Toward Global Poliomyelitis Eradication, 1985-1994,* 273 JAMA ____ (May 10, 1995).

26. Even if the court were to accept that positive lab results offer certainty in the diagnosis, the Act and its interpretation thereof does not require a petitioner to prove the elements of his or her case to a medical certainty. See Shifflett v. Secretary of HHS, 30 Fed. Cl. 341, 344-345 (1994)(finding the Vaccine Act requires neither a contemporaneous nor a definitive diagnosis of paralytic polio to establish an OPV Table injury; the burden on the petitioner is merely to establish by a preponderance of evidence that paralytic polio occurred).

27. The court discusses in detail, *infra,* the similarities and inconclusiveness of the symptoms.

28. The court agrees that the "treating physician rule," as discussed by the parties in their closings, does not apply in vaccine cases. Instead, the court looks to the treating physicians' medical notations, reports, and testimony, in so far as they corroborate or reject the polio diagnosis and provide further information not specifically outlined in the medical records. The court is permitted to defer to the treating physician, or any other expert, where testimony presented is done so cogently, credibly, and persuasively. It is in this manner the court will review the treaters' remarks, specifically Dr. Redfield's, to assess what deference, if any, is to be afforded the treating physicians.

29. Dr. Redfield is board certified in internal medicine and infectious diseases, with a speciality in clinical infectious disease; he denies expertise in the fields of immunology, neurology, or polio. Tr. at 18-22, 105. Dr. Redfield trained in the late 1970's during his residency in communicable disease and rehabilitation hospitals, where he assisted in treating already-diagnosed polio patients. Tr. at 19. While he evaluated two polio cases later in his career, Dr. Redfield admits petitioner is his first bulbar presentation. Tr. at 21, 105-106. Dr. Redfield encountered about 12 GBS cases in his training, and has treated dozens of GBS patients throughout his career. Tr. at 22, 117. Dr. Redfield notes polio and GBS remain differential diagnoses to be considered in the treatment of patients. Tr. at 21-22.

The court accepted Dr. Arnason, a neurologist, as an expert in both neurology and immunology; he has four years of immunology training and has sat on the editorial boards of immunology journals. Tr. at 216, 217. Dr. Arnason authored a major paper and clinical articles on GBS; his expertise in the diagnosis and treatment of GBS is well-known to this court. Tr. at 218. Dr. Arnason admits his polio expertise and treatment of the illness is limited. Tr. at 218. As a medical student, Dr. Arnason trained in an infectious

disease hospital during a polio epidemic where he monitored bulbar polio patients' temperature and blood pressure; his contact began after the patients' prodromal phase, upon their admission to the hospital for emergency room assistance or respiratory support. Tr. at 218, 237. He has not observed any polio cases since. Tr. at 237.

30. This case was delayed initially by respondent's request for Dr. Redfield to submit a more detailed report, based on Dr. Weibel's statement that Dr. Redfield failed to consider GBS as a possible diagnosis. R. Rpt. at 4. A careful review of the records clearly shows Drs. Redfield and Romine considered GBS from the start, but ultimately rejected the diagnosis. M.R. III at 31, 34; Tr. at 98.

31. Dorland's Illustrated Medical Dictionary defines **ptosis** as "drooping of the upper eyelid from paralysis of the third nerve or from sympathetic innervation." Dorland's illustrated medical dictionary 1387 (27th Ed. 1988).

32. The CDC report lacks a discussion of its conclusions, but Dr. Weibel's synopsis verifies the CDC relied heavily on the laboratory test results for polio and *Campylobacter* in making its decision. R. Rpt. Attachments at RE-4. However, as discussed *supra*, the CDC does not require laboratory confirmation to diagnose polio.

33. Again, the CDC's conclusions are vague, although Dr. Weibel notes Dr. Quick was struck by the "bilateral" nature of petitioner's paralysis, which apparently lent support for the CDC's conclusion that petitioner suffered GBS in the presence of slightly elevated protein. R. Rpt. Attachments at RE-4. Notably, Dr. Quick apparently had little contact with Dr. Redfield or petitioner's other treating physicians during her review of petitioner's illness, and only spoke with petitioner months after her acute illness had subsided. Tr. at 94, 208-209. In addition, no one from the CDC testified on respondent's behalf or filed an affidavit in this case regarding the CDC panel's conclusions or foundation for their determination.

34. As evidenced by the following exchange between the court and Dr. Arnason at trial, Dr. Arnason conceded that petitioner's symptoms could support a polio diagnosis:

THE COURT: . . . Would we be here today if the serological reports had come out in favor of polio?

THE WITNESS: Not for a minute.

THE COURT: So, all the symptoms that we have here may be variants of GBS, depending on what side you're on, or they may be variants of polio, but really, the crux of this case comes down to, from the Government's side, is the lack of--lack of support on the serological.

THE WITNESS: That's the nub of this issue . . . I mean, the fact of the matter is, if there had been a four-fold, eight-fold, sixteen-fold increase in antibody, we would have said, yes, this lady had an acute infection with poliomyelitis . . .

Tr. at 223.

35. The initial illnesses in either polio or GBS may consist of several symptoms; therefore, the court will divert from its stated format of addressing the medical records here and will discuss petitioner's related symptoms in the relevant categories.

36. Other literature cites a period of "wellness" following the initial illness, lasting from 2-5 days before the onset of fever and meningeal irritation. R. Exh. E at 146; R. Exh. F at 809. However, Exhibit E also notes most cases follow the initial illness without a period of "wellness." R. Exh. E at 146.

37. Aseptic meningitis may consist of several symptoms; therefore, the court will divert from its stated format of addressing the medical records here and will discuss petitioner's related symptoms in the relevant categories.

38. On December 19, 1995, petitioner "denie[d] intermittent diarrhea." M.R. II at 64. Medical records from December 25, 1995, report "some diarrhea"; December 26th records note a "couple episodes of diarrhea"; and the discharge summary lists "transient diarrhea." M.R. II at 89, 94; M.R. III at 17. Later records make generalizations, such as "[f]or the preceding 1-2 weeks the patient has had problems with stomach pains and diarrhea which has mostly improved now" and "approximately one to two weeks prior to admission, the patient started having abdominal discomfort and diarrhea . . . [t]he symptoms persisted until December 31, 1995." M.R. II at 20; M.R. III at 23. See also M.R. II at 2, 58; M.R. III at 21, 26, 30, 32. Petitioner testified she only experienced diarrhea over a 2-3 day period which resolved with medication; she denied a three week history of diarrhea as Dr. Weibel suggested in his Declaration. Tr. at 204, 212; R. Rpt. Attachments at RE-2, RE-4, RE-5.

39. Respondent's Exhibit D states the fever reoccurs 5-10 days after the initial symptoms end; Exhibit F proposes 2-5 days. R. Exh. D at 823; R. Exh. F at 809.

40. Dr. Redfield testified 37ºC or 98.6ºF is a normal temperature. Tr. at 66, 67.

41. Dorland's Illustrated Medical Dictionary defines **neuritic** as "pertaining to or affected with neuritis." Dorland's Illustrated Medical Dictionary 1127 (27th Ed. 1988). **Neuritis** is further defined as "inflammation of a nerve, a condition attended by pain and tenderness over the nerves, anesthesia and paresthesias, paralysis, wasting, and disappearance of the reflexes. In practice, the term is also used to denote noninflammatory lesions of the peripheral nervous system." Dorland's Illustrated Medical Dictionary 1127 (27th Ed. 1988).

42. See M.R. III at 35, 45, 46, 48, 50, 52, 54, 56, 58, 59, 63, 65, 68.

43. Petitioner had at least a 9-month history of headaches prior to her hospitalization, although the pain worsened shortly before her hospital admission. M.R. III at 17.

44. Dr. Redfield stated in his expert report that petitioner's neck and back stiffness promptly resolved with the progression of petitioner's clinical course, but that statement conflicts with the medical records in so far as the meningismus continued at least until January 8, 1996, although it did not worsen. Dr. Redfield's Rpt. at 3; M.R. III as cited above.

45. Pleocytosis is the presence of white blood cells in the CSF and indicates inflammation. Tr. at 33, 35.

46. Dorland's Illustrated Medical Dictionary defines a **leukocyte** as a "white blood cell or corpuscle." Dorland's Illustrated Medical Dictionary 915 (27th Ed. 1988). **Lymphocytes** are "any of the mononuclear, nonphagocytic leukocytes [*i.e.*, white blood cells or corpuscles], found in the blood . . ." Dorland's Illustrated Medical Dictionary 963 (27th Ed. 1988).

47. Other literature supports that pleocytosis in polio is usually less than 100 cells/mm^3, although higher counts may be seen. See Gareth J. Parry, Myelopathies Affecting Anterior Horn Cells, *in* Peripheral Neuropathy 891 (Peter James Dyck, M.D. et al. eds., 1993).

48. Incidentally, Dr. Quick's report from the CDC fails to mention the results of the second tube of serum, which contained 15 cells; instead, Dr. Quick concluded petitioner's white blood cell count never arose above normal based solely on the first result which showed 9 cells in the CSF. R. Rpt. Attachments at

RE-9, RE-10.

49. Dorland's Illustrated Medical Dictionary defines **albuminocytological** as "pertaining to the level of protein as albumin in relation to number of cells present in cerebrospinal fluid." Dorland's Illustrated Medical Dictionary 43 (27th Ed. 1988).

50. The CDC's range is supported by other literature citing the same level, and noting this may gradually rise in the third week and again be normal by week six. See Joseph L. Melnick, Ph.D., Live Attenuated Poliovaccines, *in* vaccines 118 (Stanley A. Plotkin, M.D. and Edward A. Mortimer, Jr., M.D., eds., 1988).

51. Dorland's Illustrated Medical Dictionary defines **dysesthesia** as "impairment of any sense, especially of that of touch." Dorland's Illustrated Medical Dictionary 517 (27th Ed. 1988).

52. Interestingly, in evaluating whether an individual's paralysis was asymmetrical if one leg or arm was totally paralyzed and the other was 50% useless, Dr. Arnason expressed that such a case would fall on the borderline. Tr. at 239.

53. See the discussion of petitioner's facial weakness as it relates to Dr. Arnason's testimony, *infra*, at page 28.

54. The literature supports that muscle paralysis may extend, occasionally, up to 1 week after its onset in polio patients. R. Exh. F at 811.

55. Incidentally, Dr. Quick concluded petitioner's paralysis peaked and stabilized between days 5 and 7; Dr. Weibel opined this occurred day 8. R. Rpt. Attachments at RE-4, RE-10.

56. Dorland's Illustrated Medical Dictionary defines **facial diplegia** as "paralysis affecting both sides of the face." Dorland's illustrated medical dictionary 478 (27th Ed. 1988).

57. Dr. Redfield agreed the definition of "paresthesia" includes sensations of burning, pricking, tickling, and tingling. Tr. at 161.

58. Dr. Redfield also described the two-fold change in the antibody titer as a "trend" and "consistent with a trend." Tr. at 176, 178.

59. While noting his disagreement with the chart contained at Exhibit F, page 809, Dr. Redfield agreed the chart, as drawn, shows that a positive neutralization antibody test matched with a negative complement fixation test means the patient probably sustained a polio infection more than 3-5 years earlier, or alternatively, the tests were flawed. Tr. at 182-185. He noted, however, that the complement fixation antibody test is not relied on clinically to diagnose polio. Tr. at 185.

60. Dr. Redfield repeatedly voiced his partial disagreement with the chart contained at Exhibit H at page 483, and only accepted certain aspects of the chart per respondent's request, as one might hypothetically (*e.g.*, "Assuming the chart is accurate, what does the chart show?"). Tr. at 171, 174, 182-185. For instance, Dr. Redfield testified the presence of antibodies greatly reduces, contrary to the chart, the opportunity for virus isolation; however, he agreed the chart, *as drawn*, reflects isolation is maximum at the paralytic onset. Tr. at 171, 173. Respondent often misrepresented Dr. Redfield's testimony in this respect.

61. According to Dorland's Illustrated Medical Dictionary, the **oropharynx** is "that division of the

pharynx which lies between the soft palate and the upper edge of the epiglottis." Dorland's Illustrated Medical Dictionary 1191 (27th Ed. 1988). Exhibit D also states the virus may be isolated from throat cultures 2 days to 2 weeks *after infection*. R. Exh. D at 822. In contrast, another article states "[v]irus recovery from tonsillopharyngeal swabs has been reported only *infrequently* after the first week following *onset of the major illness*." R. Exh. H at 480 (Emphasis supplied).

62. Exhibit D also notes that virus recovery results from stool specimens should be interpreted cautiously since the virus may be excreted for as much as 4 months *after infection*. R. Exh. D at 822. Other literature specifies isolation from the feces may be "readily accomplished during the first 3 weeks after onset *[of the major illness*]." R. Exh. H at 480.

63. Urinary bladder dysfunction and respiratory problems are possible symptoms in either diagnosis, but neither played a significant role in the experts' analysis of petitioner's case. Per Dr. Redfield's testimony and the literature, and contrary to Dr. Arnason's testimony, 25% of adult polio patients experience urinary bladder problems. Tr. at 68, 92, 255; R. Exh. D at 823; R. Exh. F at 809. In GBS, bladder dysfunction is uncommon; transient bladder paralysis is a variant form of the illness and persistent bladder or bowel dysfunction (or dysfunction occurring at onset) casts doubt on a GBS diagnosis. Tr. at 68, 92, 92, 255; R. Exh. C at 1453, 1456. Petitioner was catheterized briefly for bladder retention; Dr. Arnason testified this treatment assists little in the diagnosis. M.R. III at 35; Tr. at 255. Respiratory problems, including affected heart rate and blood pressure, can arise in polio and GBS patients. Tr. at 30, 247. Respiratory dysfunction is frequent in adult polio patients. R. Exh. D at 823. The presence of tachycardia, arrhythmias, or hypo/hypertension strongly suggest a GBS diagnosis. R. Exh. C at 1456. Antecedent respiratory infections have been reported by up to 50% of GBS patients. R. Exh. G at 614, 616. Petitioner's medical records report some respiratory problems as her clinical course progressed, but no serious complications; petitioner also had an upper respiratory infection prior to the onset of her symptoms, which was self-limited. Tr. at 68, 247; Dr. Redfield's Rpt. at 3; M.R. III at 23, 35, 45.

The experts also touched briefly on the recovery expectations of polio and GBS patients, but failed to specifically focus on petitioner's case in the discussion. The literature indicates that recovery is highly variable in either illness. R. Exh. C at 1452, 1456; R. Exh. D at 823; R. Exh. E at 145, 146; R. Exh. F at 811.