OFFICE OF SPECIAL MASTERS

No. 92-341V

(Filed: December 3, 1998)

* *		
CHRISTIAN RHYS VANT ERVE, a minor, by and through his next friends RON VANT ERVE and CATHY VANT ERVE,	*	
Petitioners,	*	**TO BE PUBLISHED**
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	
* *		

A. Leroy Tolliver, Atlanta, Georgia, for petitioners.

Michael Milmoe, Department of Justice, Washington, D.C., for respondent.

DECISION ON REMAND

HASTINGS, *Special Master*.

This is an action seeking an award under the National Vaccine Injury Compensation Program (see 42 U.S.C. § 300aa-10 *et seq*.[(1)]). For the reasons stated below, I conclude that the petitioners are not entitled to such an award.

I

BACKGROUND FACTS

Christian Vant Erve (hereinafter "Christian") was born on April 27, 1989. His parents are Ron Vant Erve and Cathy Vant Erve, who filed this petition on his behalf. In his initial two months of life, Christian appeared to be a generally healthy infant. On June 23, 1989, Christian was given a "DPT" inoculation (diphtheria, pertussis, tetanus--also known as "DTP"). Two days later, on June 25, he experienced two episodes in which his left arm and leg shook. These episodes continued on the following day, June 26, and later that day Christian displayed major movements of all extremities, prompting his parents to take him to a hospital emergency room. There he was diagnosed to be suffering from seizures. (See, *e.g.*, Pet. Ex. 7, pp. 3, 13.[(2)])

Christian has not been diagnosed as suffering from further seizures since June of 1989. However, those seizures turned out, tragically, to be the first symptoms of a terribly devastating neurologic disorder. Over the months following the seizures, questions arose concerning whether Christian's development was normal, and by late in the following year the concern became serious. At a neurologist visit on December 7, 1990, the physician noted that "of main concern is developmental delay." (Pet. Ex. 14, p. 5.) Developmental delay was noted throughout 1991, and worse yet, on October 9, 1991, Christian's neurologist noted the child to have "tightness of both heel cords," which was interpreted to be "early evidence of spastic diplegia," a very serious neurologic condition. (Pet. Ex. 14, p. 7.) The neurologist added that he suspected that the diplegia was a product of central nervous system dysfunction, the cause of which was not obvious. (*Id.*)

Since that time, Christian, tragically, has continued to suffer from severe physical and mental problems. He still suffers from spastic diplegia, a condition involving severe stiffness of limbs on both sides of his body, which makes him unable to perform almost any motor function. He also suffers from significantly diminished mental, visual, and hearing capacities, with all these deficits likely resulting from his neurologic condition.

II

APPLICABLE STATUTORY SCHEME

Under the National Vaccine Injury Compensation Program (hereinafter "the Program"), compensation awards are made to individuals who have suffered injuries after receiving certain vaccinations. There are two separate means of establishing entitlement to compensation. First, if a person suffered an injury listed in the "Vaccine Injury Table" found at 42 U.S.C. § 300aa-14(a), and the first symptoms of such injury occurred within a time period after vaccination prescribed in that Table, then that injury may be *presumed* to qualify for compensation, unless it is affirmatively shown that the injury was due to some factor other than the vaccination. § 300aa-13(a)(1)(A); § 300aa-11(c)(1)(C)(i); § 300aa-14(a); § 300aa-13(a)(1)(B). Second, compensation may also be awarded for injuries not listed on the Table, but entitlement in such cases is dependent upon proof by a preponderance of evidence that the vaccine *actually caused* the injury. § 300aa-13(a)(1); § 300aa-11(c)(1)(C)(ii).

One vaccine listed in the Vaccine Injury Table is the "DPT" inoculation, and two of the "Table Injuries" listed for that vaccine are "residual seizure disorder" and "encephalopathy" (*i.e.*, brain injury). § 300aa-14 (a)(I)(B) and (D). The Table further provides that to qualify the vaccine recipient for an award, either such injury must have been first manifested within *three days* of the inoculation. *Id*.

III

PROCEDURAL HISTORY

On May 14, 1992, the petitioners filed this action, seeking a Program award on account of Christian's devasting neurologic condition. On September 8, 1992, respondent filed her "Respondent's Report," recommending against a Program award for Christian. After settlement attempts proved unsuccessful, an evidentiary hearing, concerning the issue of whether petitioners are entitled to a Program award on Christian's behalf, was held on May 26, 1994. At that hearing, three medical experts testified for petitioners--Dr. Jan Mathisen, a pediatric neurologist who has treated Christian since 1989; Dr. Marcel Kinsbourne, another pediatric neurologist; and Dr. Roy Strand, a pediatric neuroradiologist. Two medical experts testified for respondent--Dr. Arnold Gale, a pediatric neurologist, and Dr. Charles Fitz, a pediatric neuroradiologist.

At the hearing, the petitioners' chief contention was that Christian had suffered the two "Table Injuries" noted above--*i.e.*, "residual seizure disorder" and "encephalopathy." Respondent disputed both points, but also argued that even if Christian had suffered those Table Injuries, such injuries were nevertheless not compensable because they were "due to factors unrelated to the administration of the vaccine." See § 300aa-13(a)(1)(B). Respondent contended that Christian's injuries were due to a prenatal injury to his brain.

On June 21, 1994, I filed my written Ruling concerning the entitlement issue, explaining my reasoning for concluding that petitioners *did* qualify for a Program award on Christian's behalf. See *Vant Erve v. Secretary of HHS*, No. 92-341V, 1994 WL 325426 (Fed. Cl. Spec. Mstr. June 21, 1994). I concluded that Christian did in fact suffer both of the alleged Table Injuries, and that respondent had failed to carry her burden of demonstrating that such injuries were caused by a prenatal injury.

For nearly three years thereafter, the parties made efforts to settle the "damages" issue. Then, in June of 1997, respondent requested that the entitlement issue be reopened. I denied the request. See *Vant Erve v. Secretary of HHS*, No. 92-341V, 1997 WL 383144 (Fed. Cl. Spec. Mstr. June 26, 1997). However, on review, Judge Bruggink of this court reversed that ruling, concluding that the entitlement issue should be reopened. *Erve v. Secretary of HHS*, 39 Fed. Cl. 607 (1997). After remand of the case to me, both sides submitted additional expert reports, additional medical records were filed, and an evidentiary hearing was held on September 18, 1998. Drs. Gale and Fitz testified once again for respondent, but this time only Dr. Mathisen testified for petitioners.

IV

RESOLUTION OF KEY FACTUAL DISPUTE

On remand, respondent has offered a theory somewhat different from the one advanced before me in 1994. In 1994, respondent's two experts theorized that all of Christian's neurologic symptoms, including his seizures, were the product of a static injury to his brain that occurred during his prenatal period. Now, with the advantage of considerably more evidence than was available in 1994, they argue that all of Christian's neurologic problems are the result of a progressive, dysmyelinating, metabolic disorder of genetic origin. Petitioner's sole expert on remand, Dr. Mathisen, disagrees. For the reasons to be set forth below, I found respondent's experts to be substantially more persuasive than petitioners' expert, and I hereby find that Christian's neurologic condition is, more probably than not, the result of a progressive, dysmyelinating, metabolic disorder.

A. Theory of respondent's experts

Respondent's experts rely principally upon the results of four "MRI"--*i.e.*, magnetic resonance imaging--procedures performed upon Christian's brain in 1991, 1992, 1993, and 1997. Dr. Fitz, a pediatric neuroradiologist, explained that these four images unquestionably show progressive deterioration of the white matter in Christian's brain. In particular, he noted that the images show that the cerebellum and brain stem portions of Christian's brain have shrunk to a significant degree. (See, *e.g.*, 2-Tr. 26.[(3)]) He characterized this white matter deterioration as the result of a process of "dysmyelination;" this means that the protective sheath around the brain's white matter, which is made of a substance called myelin, is defective because the myelin that the body produces is itself abnormal and defective. (See 2-Tr. 35-36, 96.) Dr. Fitz opined that Christian's progressive, dysmyelinating disorder is also correctly characterized as a "metabolic" disorder, meaning that it results from an error in Christian's metabolism. (*E.g.*, 2-Tr. 9, 32, 34, 62-63.) He further explained that in his opinion an injury produced by a pertussis vaccination, or any other type of static injury, could not have produced the condition in Christian that has been

demonstrated by the progressively worsening MRI images. (2-Tr. 11-13, 69-70.)

Dr. Gale, the neurologist, expressed agreement with Dr. Fitz's analysis. He emphasized that a single, "static" injury could not result in the series of progressively worsening MRI images. (2-Tr. 98, 218-219.) He also explained that *any* progressive dysmelination process would *necessarily* result from a *metabolic* disorder. (*E.g.*, 2-Tr. 97-100, 213-214.)

Moreover, both of respondent's experts argued strongly that because of the MRI results, it is not even a close case, as to which experts could reasonably disagree, as to whether Christian has a progressive metabolic disorder. They expressed bewilderment at Dr. Mathisen's statements of disagreement with their conclusion on this point. (*E.g.*, 2-Tr. 32, 54, 77, 80-81, 84-85.) They both acknowledged that based upon the evidence available to them to date, they cannot specify the *exact type* of metabolic disorder that Christian has, but they have no doubt that all of his tragic neurologic symptoms are due to a progressive metabolic disorder. (*E.g.*, 2-Tr. 34, 100.)

B. Dr. Mathisen's theory

Dr. Mathisen does not disagree that the MRI images show that Christian has a disorder of his white matter. (2-Tr. 154, 174, 200, 206-207.) But he argues that this disorder is neither progressive nor metabolic in origin. (*E.g.*, 2-Tr. 206-207.) He continues to argue that the DPT vaccination caused the white matter injury. (*E.g.*, 2-Tr. 140-141, 172.) As the basis for his opinion, Dr. Mathisen seems to rely chiefly upon the assertion that Christian has not regressed *clinically*--meaning in his neurologic abilities and disabilities assessed by personal examination by a neurologist--to the extent that one might expect by looking only at the MRI results. He acknowledges that Christian suffered some decline in function during the early years of his disorder (*e.g.*, 2-Tr. 142, 182), but argues that Christian's clinical exams have been fairly stable for the last two years (2-Tr. 155-156, 181). Dr. Mathisen purports not to dispute Dr. Fitz as to what can be seen on the MRI images, but argues that the clinical evidence should be given greater weight than the MRI results in making a final analysis of Christian's case. (2-Tr. 139, 157-59, 165-67.) He argues that there does not exist enough evidence to find it "more probable than not" that Christian has a metabolic disorder.

C. Comparative analysis

I find the analysis of respondent's experts to be far more persuasive than that of Dr. Mathisen. I will discuss, in turn, the principal reasons for that conclusion.

1. Evidence of clinical regression

One important reason is that an examination of the record tends to support the view that even based upon Christian's *clinical* picture, upon which Dr. Mathisen purports to place such emphasis, Christian's condition appears to be a *progressive* one, rather than a static one as Dr. Mathisen asserts. To be sure, Dr. Mathisen has acknowledged that in the first few years of his life Christian displayed "some evolving neurologic condition" (2-Tr. 140) and showed "some early decline" (2-Tr. 182). But these are such gross understatements that they strike me as disingenuous. Actually, the medical records of Christian's disorder--including Dr. Mathisen's own records--contain *very clear* evidence of *significant* developmental decline and regression.

I will review the relevant records on this point in chronological order. First, on March 6, 1992, Dr. Mathisen himself wrote that--

some of Christian's development seems to be slowing down a bit. He was eating fairly well last year but

this has stopped. * * * Cruising appears to be present but is not as well developed as it has been previously. He is having a much harder time holding his spoon. * * * In summary, Christian * * * has had some evidence of mild developmental regression.

(Dam. Ex. C, p. 13.) Similarly, on April 22, 1993, Dr. Mathisen wrote that Christian--

has been having * * * more significant neurologic problems. * * * Previously he could sit up well but, at this time, is not able to do so. He is having a greater difficult time with his tone increasing * * *. * * * His eating has decreased. * * * [He has a] degree of regression that we are seeing.

(*Id.* at 8.) On April 30, 1993, another physician described Christian as experiencing "worsening physical performance." (*Id.* at 32.)

On June 7, 1993, Dr. Mathisen wrote of Christian's "progressive worsening of spasticity and swallowing difficulties." (Dam. Ex. C at 47.) On September 16, 1993, the same physician wrote of Christian's "history of * * * worsening neurologic status, with decreased sitting, increased drooling," added that Christian's ability to walk with a walker "is now decreased," and also remarked upon Christian's "history of progressive neurologic deterioration in the last several weeks and months." (*Id.* at 48.) On December 13, 1994, Dr. Mathisen wrote that Christian's examination that day was "notable for progressive difficulties with back support, increased drooling and poor hand usage," and that there "has been some concern recently of developmental regression, especially in muscle groups involving his back." (*Id.* at 2.) Dr. Mathisen added on the same day that Christian "seems to have more fisting and difficulty with back control," summarizing that the child "appears to have a little bit of worsening of some of his neurologic features." (*Id.* at 4.)

The medical record quotation set forth in the last two paragraphs, thus, constitute irrefutable evidence of *significant* regression of Christian's *clinical* neurologic condition from late 1991 through late 1994.[(4)] Further, the record contains less direct, but still compelling, evidence of *additional* clinical regression in the following two years. That is, between February of 1995 and September of 1996, petitioners submitted in this Program proceeding four different "life care plans," in which each succeeding plan generally reflected *increased* needs for Christian's care. As Judge Bruggink pointed out, it is apparent that this sequence of plans resulted from continual worsening of Christian's condition during that time period. 39 Fed. Cl. at 609, fn.6.

In this regard, I acknowledge that I have found no evidence in the record of further clinical regression since the September 1996 life care plan. But the medical records for this recent period are sparse, with virtually no records since February of 1998 because Dr. Mathisen has not seen Christian since then. (2-Tr. 169.) Moreover, Dr. Gale explained why even in the context of a clearly progressive, metabolic disorder, one might expect little or no clinical evidence of additional regression in Christian in recent years. (2-Tr. 95.) Thus, my conclusion is that the overall record *does* show a pattern of significant *clinical* regression in Christian that corresponds to the progressive brain deterioration that Drs. Fitz and Gale have observed on the MRI images.[(5)] This pattern strongly supports the theory of respondent's experts over that of Dr. Mathisen.

2. Credibility of Dr. Mathisen

A second major reason why I have been persuaded by Drs. Fitz and Gale, rather than Dr. Mathisen, is that based upon the overall history of this case, I simply have come to question the candor of Dr. Mathisen's testimony. I have come to suspect that at this point he is merely striving to provide an opinion supportive of this most unfortunate child, rather than expressing his most candid scientific opinion. In this regard, I

have already noted above (p. 5) that I found his gross understatements as to Christian's early regression to be somewhat disingenuous. I also note that in the recent evidentiary hearing, Dr. Mathisen's answers were often so roundabout and unresponsive that I received the impression that he was simply avoiding a "straight answer" to the question. (See, for example, 2-Tr. at p. 173, lines 11-23; *id*. at p. 200, line 7, through p. 201, line 16.)

Further, it must be noted that some of the statements made by Dr. Mathisen in the first evidentiary hearing, held on May 26, 1994, seem suspect in light of other statements that he had made in medical records that actually were already in existence prior to that hearing, but had not at that time found their way into the evidentiary record of this Program proceeding. For example, during the 1994 hearing Dr. Mathisen stated that "on occasion we do see that on follow-up MRI scans that there is progressive white matter changes, or progressive brain atrophy if there is a specific perinatal event. And we did not see any of that." (1-Tr. 106.) However, prior to this testimony, Dr. Mathisen had already written, in reference to the April 1993 MRI scan, that the "MRI scan was quite abnormal with evidence of both brain stem and cerebellar atrophy with relatively minimal changes involving the cerebral cortex. The findings were thought to be more compatible with an underlying genetic disorder involving the brain stem and cerebellar region." (Dam. Ex. C at 7.) As Judge Bruggink has pointed out (39 Fed. Cl. at 614), on this point Dr. Mathisen's 1994 testimony seems quite inconsistent with his 1993 letter. Moreover, as noted above, in documents dated April 22, June 7, and September 16 of 1993, Dr. Mathisen pointed out evidence of recent *regression* in Christian. Yet in his testimony on May 26, 1994, though he hadn't seen Christian again since he wrote down those statements in 1993, Dr. Mathisen opined that Christian was *not regressing*. (1-Tr. at 131.) Based on both these points, an inference can be made that Dr. Mathisen's 1994 testimony was not candid, and that certainly casts doubt upon the credibility of his 1998 testimony as well.[(6)]

3. Points of petitioners' counsel

In weighing the expert testimony, I have carefully considered the arguments made by petitioners' counsel. For example, petitioners have stressed that the theory now advanced by respondent's experts is different from the one that they adopted in 1994, when they thought that Christian probably had periventricular leukomalacia ("PVL") and cerebral palsy. But respondent's experts have been forthright in acknowledging this change, explaining that the much greater amount of evidence now available justifies their current conclusion. (See, *e.g.*, 2-Tr. 29-31, 122-23.) Moreover, it is worthy of note that in retrospect, it is now clear that of the two pediatric neuroradiologists who examined the images available in 1994, the analysis of Dr. Fitz in fact was superior. It turns out that, as Dr. Fitz then testified, there *were* slight abnormalities in Christian's brain (which have since become much worse). Dr. Fitz, of course, was incorrect in 1994 as to the *cause* of the abnormalities, but he was correct that abnormalities *did exist*, in contrast to the opinion of the neuroradiologist who then testified for petitioners, Dr. Strand.

In short, I do not find that the fact that Drs. Fitz and Gale were not completely correct in 1994 is sufficient reason for me to credit Dr. Mathisen's view over their opinions in 1998. On the basis of the information now available, the analysis of the respondent's experts now seems substantially more convincing.

Similarly, petitioners have pointed out that a number of statements or diagnoses appearing in the medical records, made by physicians *other* than Dr. Mathisen, are not completely consistent with the current analysis of Drs. Fitz and Gale. (See, *e.g.*, 2-Tr. 50, 52-57, 123-127.) For example, there are references to "demyelination" in Christian's brain, rather than the "dysmyelination" process that respondent's experts have identified. However, most of those references are in the records of the early years of the disorder, when there existed much less information about Christian's condition. Moreover, Dr. Mathisen *did not* follow up on any of these points and attempt to explain exactly how Drs. Gale and Fitz might now be

wrong in their current analysis. For example, while petitioners' counsel made much of the record references to *de*myelination, in his testimony Dr. Mathisen ultimately did *not* dispute that, at least based upon the MRI images, it would be reasonable to conclude that Christian suffers from *dys*myelination. (2-Tr. 176.) In short, I have carefully examined the medical records, and do not find any evidence that dissuades me from concluding that the current analysis of Drs. Fitz and Gale is likely correct.

4. Summary and conclusion

In summary, I note that the theory of Drs. Gale and Fitz seems to fit the overall record of Christian's condition much more so than that of Dr. Mathisen. In addition, I have strong doubts about the candor of Dr. Mathisen. And at the recent hearing, Drs. Gale and Fitz simply seemed to be much better able to explain and defend their theory, in a straightforward and lucid fashion, than was Dr. Mathisen. Accordingly, based upon the whole record,[(7)] I find, as a matter of fact, that it is substantially "more probable than not" that Christian's entire history of neurologic dysfunction, including his seizures as well as his devastating array of additional neurologically-related disabilities, has been the result of a progressive, dysmyelinating, *metabolic* disorder.

V

IMPACT OF FACTUAL FINDING

In this case, petitioners have advanced three basic theories of proof: (1) that Christian suffered a "Table Injury seizure disorder;" (2) that he suffered a "Table Injury encephalopathy;" and (3) that his neurologic condition was "actually caused" by his vaccination. In this section of this opinion, I will discuss the impact that my factual ruling, made in the previous section, has upon each of these potential theories of proof.

A. "Actual causation" theory

Taking the theories in reverse order, it is first clear that petitioners have failed to demonstrate "actual causation." My key factual finding in this case, as explained above, is that it is *not* likely that Christian's DPT vaccination caused his tragic neurologic condition, but instead it is "more probable than not" that his condition is due to a progressive, dysmelinating, metabolic disorder. Moreover, even setting aside the fact that respondent has successfully shown a specific cause for Christian's condition, I note that petitioners have *never* in this case even come close, in my view, to supplying any substantial evidence, beyond unexplained expert assertions, supporting the proposition that the DPT vaccination *actually caused* Christian's condition. Rather, petitioners' only *substantial* theory, all along, has been that they are entitled to *a statutory presumption* of causation because of the temporal relationship between Christian's vaccination and the onset of his neurologic symptoms. Thus, petitioners clearly have *not* successfully demonstrated "actual causation" in this case.

B. "Table Injury encephalopathy" theory

Next, it is also clear that my factual finding means that petitioners have failed to make a meritorious case concerning their "Table Injury encephalopathy" theory. A bit more discussion is required on this point, however.

As noted above, if a person suffered an injury listed in the Vaccine Injury Table found at 42 U.S.C. § 300aa-14(a), and the first symptom of such injury occurred within a time period after vaccination also

specified in that Table, then that injury will be *presumed* to qualify for Program compensation. § 300aa-13(a)(1); § 300aa-11(c)(1)(C)(i); § 300aa-14(a). As also noted above, in my 1994 Ruling in this case I concluded that Christian did suffer an "encephalopathy," as that term is defined at *part (A)* of § 300aa-14(b)(3), and that the first symptoms of that "encephalopathy" were exhibited during the three-day period following his DPT immunization. (1994 WL 325426 at *7-9.)

On this remand, respondent does not contest my 1994 findings that Christian's neurologic disorder falls within the "encephalopathy" definition provided at *part (A)* of § 300aa-14(b)(3), and that the first symptoms of that disorder occurred within the three-day period post-vaccination. Nevertheless, my factual finding that Christian's disorder is a "metabolic" disorder still knocks Christian's disorder out of the category of a "Table Injury encephalopathy," for a *different* reason. That is, *part (B)* of § 300aa-14(b)(3) provides *another* requirement for a "Table Injury encephalopathy." That provision states as follows:

(B) If in a proceeding on a petition it is shown by a preponderance of the evidence that an encephalopathy *was caused by* infection, toxins, trauma, or *metabolic disturbances* the encephalopathy shall not be considered to be a condition set forth in the table.

§ 300aa-14(b)(3)(B) (emphasis added). In this case, Drs. Gale and Fitz explained that the fact that Christian has a "metabolic disorder" means that his disorder is the product of a "metabolic disturbance."[(8)] (2-Tr. 67, 100-102.) Accordingly, as a result of my factual finding set forth in part IV of this Ruling above, it follows that Christian has *not* suffered an encephalopathy falling into the "Table Injury" category.[(9)]

C. "Table Injury seizure disorder" theory

As also noted above, in my 1994 Ruling I found that Christian suffered, in addition to his "Table Injury encephalopathy," a "Table Injury residual seizure disorder" as well. (1994 WL 325426 at *3-4.) On this remand, respondent has not contested that ruling either.[(10)] Respondent argues, rather, that this "Table Injury seizure disorder" suffered by Christian is not compensable under the Program because it was "due to factors unrelated to the administration of the vaccine." § 300aa-13(a)(1)(B). And, once again, my factual finding set forth above--*i.e.*, that Christian's entire neurologic disorder, including his seizure disorder, was due to a progressive, dysmyelinating, *metabolic* disorder--means that respondent's argument is meritorious. In other words, respondent has successfully demonstrated that Christian's Table Injury seizure disorder was caused by a factor unrelated to his vaccination--namely, a metabolic disturbance.

As a final point in this regard, I note that in the proceedings before Judge Bruggink, petitioners apparently argued that respondent's current theory would not satisfy the statutory requirement for a "factor unrelated," because the cause now advanced by respondent is allegedly an "idiopathic" one. See 39 Fed. Cl. 6134 fn.17; § 300aa-13(a)(2)(A). However, on remand, petitioners have not raised such an argument. Moreover, I have considered that potential issue on my own, and I find it clear that the "factor unrelated" advanced by the respondent in this case clearly does *not* suffer from the alleged deficiency of being an "idiopathic" factor. I acknowledge that, in general, the existing case law is somewhat murky as to *exactly how specific* respondent must be in order to successfully demonstrate a "factor unrelated" that is not "idiopathic." See, *e.g.*, *Koston v. Secretary of HHS*, 974 F.2d 157 (Fed. Cir. 1992); *Whitecotton v. Secretary of HHS*, 17 F. 3d 374, 377-78 (Fed. Cir. 1994), *rev'd on other point*, *Shalala v. Whitecotton*, 514 U.S. 268 (1995); *Hanlon v. Secretary of HHS*, 40 Fed. Cl. 625 (1998); *Plavin v. Secretary of HHS*, 40 Fed. Cl. 609 (1998). However, in this case, the fact that Christian's condition was caused by a *metabolic disturbance* clearly means that the "factor unrelated" is *not* impermissably "idiopathic." That is, the statute *specifies* four types of "factors unrelated"--*i.e.*, "infection, toxins, trauma * * *, or metabolic

disturbances"--that appear to *automatically* pass the "idiopathic" test. § 300aa-13(a)(2)(B). In other words, § 300aa-13(a)(2)(B) indicates that if the respondent demonstrates that a Table Injury was caused by *one of the specified four causes*, such cause should *not* be considered an "idiopathic" factor. See, *e.g., Knudsen v. Secretary of HHS*, 35 F. 3d 543, 548-49 (Fed. Cir. 1994), in which the court of appeals held that if the respondent demonstrates that an *infection* caused the Table Injury, that is enough for a "factor unrelated" showing, with no need to demonstrate *exactly which virus* caused the infection.

In this case, because respondent has shown that Christian's entire neurologic disorder, including his seizure disorder, was probably caused by a *metabolic disturbance*, respondent has clearly made a successful "factor unrelated" showing, passing the "idiopathic" test.

VI

CONCLUSION

The story of Christian Vant Erve's neurologic disorder is obviously a tragic one. Moreover, it is unfortunate that the procedural history of this Program proceeding has played out in an unusual fashion that will undoubtedly add additional disappointment and heartbreak to the difficulties that this courageous family has already had to bear. Congress, however, did not design the Program to compensate all families with tragically injured children. Rather, compensation is limited to those whose factual circumstances fit within the Program's statutory requirements. And, unfortunately for the Vant Erves, under the evidence available at this time I cannot find that Christian's case fits within those requirements. Therefore, I conclude that petitioners are not entitled to a Program award.

It is not clear under either the statute or the Rules of this court whether the filing of this "Decision on Remand" automatically triggers a new 30-day period for seeking review under Appendix J, Rules of the United States Court of Federal Claims, Rule 23. It is *arguable* that in the absence of a motion for review filed within 30 days of the date of this Decision, the Clerk of this court should automatically enter judgment in accordance herewith. But that is not completely clear. The parties and/or the Clerk may wish to seek guidance from the chambers of Judge Bruggink as to the appropriate procedure at this time.

George L. Hastings, Jr.

Special Master

1. The applicable statutory provisions defining the Program are found at 42 U.S.C. § 300aa-10 *et seq*. (1994 ed.). Hereinafter, for ease of citation, all "§" references will be to 42 U.S.C. (1994 ed.).

2. Petitioners filed 18 numbered exhibits with the petition. "Pet. Ex." references will be to those exhibits. Respondent on May 2, 1997, filed a volume containing numerous additional medical records, divided into "Damages Exhibits" C through S. "Dam. Ex." references will be to those exhibits.

3. "2-Tr." references are to the transcript of the hearing held on September 18, 1998. "1-Tr." references are to the transcript of the hearing held on May 26, 1994.

4. Note that Dr. Gale also interpreted these medical records as *clinical* evidence of a *progressive* disorder. (See 2-Tr. 82, 85-89, 91-92.)

5. See also the observation of Judge Bruggink:

Dr. Mathisen's own records make two facts abundantly clear. First, Christian's clinical course since 1992 has been characterized by *significant and continuous regression* in his psychomotor development. This is supported by the observations documented in his academic and rehabilitation therapy records.

39 Fed. Cl. at 610 (emphasis added).

6. I note that at the 1998 hearing, Dr. Mathisen was invited to explain this seeming contradiction. His attempt to do so did nothing to enhance his credibility. (See 2-Tr. at p. 196, line 12, through p. 197, line 25.)

7. I note that the opinions of Drs. Kinsbourne and Strand still are part of the record before me, even though they did not testify during the *remand* proceeding. Accordingly, I have not ignored their opinions. But in the final analysis I cannot give those opinions any *significant* weight, since those opinions, given in 1994, werre based upon a scant subset of the evidence that is now available concerning Christian's disorder.

8. It is not absolutely clear whether, under § 300aa-14(b)(3)(B), it is a *petitioner's* burden to show that an encephalopathy was *not* "caused by infection, toxins, trauma, or metabolic disturbances," or instead it is *respondent's* burden to show that an encephalopathy *was* so caused. In this case the legal point is irrelevant, however, since I have ruled that respondent clearly *did* demonstrate that Christian's encephalopathy was caused by a metabolic disturbance.

9. I also note that even if part B of § 300aa-14(b)(3) did not exist, the ultimate result here would be no different. In that case, petitioners *would* have successfully demonstrated a "Table Injury encephalopathy." However, even in that case Christian's "Table Injury encephalopathy" would still *not* be compensable under the Program, for the same reason that Christian's "Table Injury seizure disorder" is not compensable--because it has been shown to be "due to factors unrelated to the administration of the vaccine." See part V(C) of this Decision.

10. In this regard, I note that as to the issue of whether Christian suffered the "residual effects" of his *seizure disorder* for at least six months, in retrospect my statutory interpretation (see 1994 WL 325426 at *3-4) now seems to me to probably have been somewhat overexpansive. However, that point is now moot, since respondent has successfully made a "factor unrelated" showing as to the seizure disorder Table Injury.