OFFICE OF SPECIAL MASTERS
No. 90-1703 V
(Filed: June 4, 1998)

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JOHN LURTZ and DONNA LURTZ, as Natural Guardians of PAUL WILLIAM LURTZ, a minor	*	
Petitioners,	*	**TO BE PUBLISHED**
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

* *

Jonathan F. Sobel, Beachwood, California for petitioners.

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

French, Special Master.

DECISION

This matter arises under 42 U.S.C. §300aa-1 et seq., the National Vaccine Injury Compensation Act. On September 27, 1990, petitioners filed their claim in this court alleging that as the result of a Diphtheria-Pertussis-Tetanus (DPT) vaccination administered on July 12, 1982, their infant son, Paul William Lurtz (hereinafter Paul), sustained an encephalopathy and a residual seizure disorder as defined by §14 of the Vaccine Act, and that his present neurological deficits are causally related to those injuries.

Respondent argues that the contemporaneous medical records are inconsistent with petitioners' claim and that a vaccine-related on-Table injury, therefore, cannot be established. Respondent claims in the alternative, that even if an on-Table injury did occur, Paul's neurological condition was significantly aggravated by an intervening catastrophic event unrelated to the vaccine and that his present condition, more likely than not, was triggered by a malignant viral illness, when Paul was seven years old.

PROCEDURAL BACKGROUND

Petitioners filed their initial action for a vaccine injury in the State of Ohio. The merits of their claim were never considered by the Ohio court, and the case was dismissed to permit filing in the Court of Federal Claims. The case was assigned to the undersigned special master on May 3, 1993. An evidentiary hearing, limited to factual issues, was held in Washington D.C. on June 30, 1994. The following witnesses appeared: Mrs. Donna Rebecca Lurtz, Paul's natural mother; John Martin Lurtz, Paul's natural father; and Mrs. Barbara Means, a neighbor who was an eyewitness of the alleged onset of Paul's symptoms. Respondent called no witnesses.

At the close of the June 30, 1994 hearing, the court indicated its intention to find facts favorable to petitioners' claim based upon the highly credible testimony of the fact witnesses and upon credible evidence documented in the medical records that support petitioners' claim of onset of Paul's injuries within the requisite 72-hour Table time frame.

At respondent's request, however, the record was held open to permit examination of interrogatories, affidavits, and deposition testimony prepared for the prior civil action and to permit respondent to reassess its position.[(1)] No evidence to impeach the testimony of the eyewitnesses was found, and the court now affirms its finding of an on-Table onset of symptoms. A detailed description of the basis for the court's factual findings is set forth in the Transcript of the June 30, 1994 proceedings (hereinafter Tr.) at 122-127.

Following the June 30, 1994 hearing on factual matters, the parties discussed the possibility of settlement based on litigative risk. Negotiations continued for many months without success. In the meantime, both parties began to pursue efforts to determine the level of damages during which time respondent raised new medical issues suggesting the possibility that factors other than the vaccine might be responsible for Paul's clinical course. A hearing on medical issues was held September 9, 1997 in Washington D.C. Dr. Gerald Erenberg testified on behalf of petitioners. Dr. Arthur Prensky testified for respondent. Both doctors are pediatric neurologists.

FINDINGS OF FACT

Paul Lurtz was born on May 6, 1982, the normal product of an uneventful pregnancy and delivery. His APGAR scores were 9 and 9 at one and five minutes respectively after birth. For the first two months of life, his development was normal and his health was good except for "gastrocolic" reflux causing stomach cramps and minor diarrhea.

At a well-baby check on July 12, 1982, in the office of Dr. James Jones of Mansfield, Ohio, two-month-old Paul was given his first DPT shot. About four hours later, his mother was sitting at the kitchen table having a cup of tea with Mrs. Barbara Means, a neighbor. Paul was sitting in a "pumpkin chair" similar to a light-weight car seat. He had a seat belt around his stomach leaving his arms and legs free. Paul let out a sudden strange yell and threw his head back. His arms and legs flew out and twitched, his eyes became glassy, and his face assumed a strange grimace involving the right side of his mouth. This episode lasted perhaps five to seven seconds. His mother placed her hand on him and spoke to him, but he showed no response and seemed to fall immediately into a sound sleep lasting from 45 minutes to an hour. Both Mrs. Lurtz and her neighbor saw the incident.[(2)] Mrs. Lurtz was puzzled and told her husband about it, but the incident was not reported to the doctor until later. Many similar episodes would be observed later, and this first incident would be recalled and explained.

The following events are well documented and were not challenged. Exactly one month later, on August 12, 1982, while his mother was getting him dressed, the infant started twitching dramatically and jerking rhythmically for a period of approximately 15 to 20 minutes. His head was thrown back, his eyes rolled back, and his body stiffened. His mother could not move his body nor bend his leg or his arm. Mother and Father rushed him to the hospital where his vital signs were checked, blood tested, a spinal tap taken, and EEG tests performed. Dr. Shakar identified this event as a grand mal seizure. When asked if there had been a history of fever, vomiting, diarrhea, or upper respiratory infection, Mrs. Lurtz answered, "no." When asked if the baby had ever done this before, Mrs. Lurtz also said "no."[(3)] She would later realize that she was mistaken. Thereafter, Paul's seizure disorder became full blown and is described now as "intractable."

The pertussis component of the vaccine was eliminated in subsequent DPT vaccinations. Paul received thereafter DT only. No other cause of his seizure disorder has ever been identified. He has continued to suffer from a mixture of seizure types including absence, petit mal, focal, grand mal, and episodes of status epilepticus;[(4)] some episodes have been severe enough to cause apnea. His seizures include the same type first observed on July 12, 1982. At one point Paul was having between 100 and 150 seizures a day. Mrs. Lurtz asked her pediatrician if the DPT could have caused Paul's seizures. According to her testimony, Dr. Burns told her that it was a great possibility but that they would never be able to find out for sure. The nature of his seizure disorder is apparently closely associated with an unfavorable prognosis, and, predictably, Paul continues to suffer multiple types of seizures and psychomotor deficits. [(5)]

In spite of the intractable nature of his disorder, Paul was able to make some gains. By the time he had reached a chronological age of seven years, he was able to function at the level of a two and one-half to three-year old child although development of speech was significantly affected. He was able to assist in his daily care, he recognized his caregivers, and he was able to communicate with them. That condition was drastically altered after an event that occurred on November 16, 1989. The nature of that event and its cause, is critical to the outcome this case.

On November 16, 1989, more than seven years after the onset of Paul's seizure disorder, a catastrophic episode of status epilepticus occurred that lasted approximately two hours and could be stopped only by placing the child in a sodium pentothal coma.[(6)] He remained hospitalized for two months, and when discharged, still remained in a severe coma. Although Paul had had episodes of status epilepticus before on many occasions, the event of November 16, 1989 lasted nearly three times longer than any previous occurrence and was accompanied by unusual symptoms not usually encountered with his seizure disorder, i.e., acute kidney failure with anuria, hepatic failure, and high elevations of CSF lactic acid and plasma lactic acid. Doctor Cruse wrote as follows:

Impressions and Recommendations: Paul clearly had an episode of acute encephalopathy associated with fever which by history was preceded by a viral-like illness with herpangina.[(7)] The precipitating cause for his status is not clear but surely is not unusual in children who are prone to seizures when they develop a febrile illness. . . . The atypical features about Paul's illness is [sic] not only his status epilepticus, surely he has had episodes of status before on many occasions in the past, but his acutely developing kidney failure with anuria[(8)] and hepatic failure are unusual. . . . At the time of discharge from the hospital Paul had findings of chronic stupor with significant regression in his neurological status as compared to prior to the onset of the status epilepticus. . . . Also, his MRI scan did show significant generalized cerebral atrophy which was not present on his initial CT or MRI scan.

Report of Dr. Robert Cruse of March 19, 1990, filed March 2, 1995 at 3-4.

The parties agree that after the November 16, 1989 event, Paul's neurological condition was significantly worsened. Nearly five years later, at the time of the June 30, 1994 factual hearing, Paul still remained in a level three or four coma.[(9)] He can hear, and on some days he has vision. On others days, he does not. He can move his arms back and forth, hold his head up a little sitting in a wheelchair, but he does not grasp or make any purposeful movements. He cannot swallow, takes many different medications, and is fed through a gastric tube.

STATUTORY PROVISIONS

Petitioners in vaccine cases are entitled to compensation for injuries and for any sequelae causally related to a covered vaccine. DPT is one such vaccine. To establish a residual seizure disorder, §14 of the statute requires petitioners to demonstrate: 1)that the injured individual had suffered no seizures prior to the onset of the first seizure; 2)that the first seizure following the DPT shot occurred within 72 hours; and, 3)that two or more seizures occurred within one year unaccompanied by fever in excess of 102 degrees F. §14(b)(2). The issue of sequelae, however, is a complicated one. A causal relationship between the vaccine and the initial injury is presumed if the facts support an on-Table onset of symptoms. That presumption does not extend to sequelae. Petitioners are required to prove sequelae of vaccine-related injuries by traditional litigation standards, i.e., by a preponderance of evidence.

Respondent may rebut the presumption of causation by establishing, by a preponderance of evidence, that the injury was caused by factors unrelated to the administration of the vaccine. The term "factor unrelated" may not include any "idiopathic, unexplained, unknown, hypothetical, or undocumentable cause, factor, injury, illness, or condition, but may include infection, toxins, trauma. . . or metabolic disturbances which have no known relation to the vaccine involved, but which in the particular case [is] shown to have been the agent or agents principally responsible for causing the . . . condition." §13(a)(1) and (2).

ISSUES

At the beginning of the September 9, 1997 hearing on medical issues, respondent conceded that based on the court's factual findings, all elements required to demonstrate a residual seizure disorder had been met. Transcript of September 9, 1997 (hereinafter Tr.II) at 7. Respondent, however, does not concede that Paul's present neurological condition is vaccine-related, arguing that the devastating episode of November 16, 1989 was caused, more likely than not, by an intercurrent, intervening viral infection unrelated to the vaccine that significantly aggravated his condition.

Because the experts agree that the event of November 16, 1989 was the source of an abrupt worsening of Paul's condition, the nature and cause of that event is the overwhelming issue in this case. Debate at the hearing of experts focused on two possible causes: Petitioners argue that the abrupt devastation in Paul's neurological status was causally related to the prolonged seizure, and is a sequela, therefore, of his vaccine injury. Respondent argues that it was caused by an unidentifiable viral infection.

EXPERT OPINION

Medical Records of Dr. Robert Cruse, treating physician:

Dr. Cruse was Paul's treating specialist during the extensive November 1989 - January 1990 hospitalization. Dr. Cruse was not called as a witness, but his discharge summary, dictated on October 9, 1990, is highly relevant. He reports that on November 16, 1989, Paul "clearly had an episode of acute encephalopathy" that by history was preceded by "a viral-like illness." He states further that multiple

cultures and tests for viral titers were unable to confirm the presence of the suspected virus.[(10)] Extensive efforts to determine any other origin of Paul's encephalopathy also failed. Muscle biopsy, skin biopsy, biotin studies, tests for organic acids (including carnitine), and convalescent monitoring for herpes titers, were all negative or normal with the exception of a positive culture for Herpes Stomatitis (generalized inflammation of the oral mucosa, that is, sores in the mouth), a mild sinus infection, and right middle lobe pneumonia. These conditions were treated with antibiotics.[(11)] No identifiable metabolic disorders were found. It was Dr. Cruse' opinion that liver and kidney problems were probably secondary to an anoxic insult (lack of oxygen during status epilepticus) and that development of coagulopathy (blood clotting), was believed to be secondary to the liver failure. Kidney and liver problems gradually improved, returned to normal, and "remained normal." Id.

According to Dr. Cruse, Paul's sudden devastating change in neurological status remained essentially unchanged thereafter. CT and MRI scans revealed generalized cerebral atrophy that was not apparent in prior imaging. Periodic EEGs thereafter were abnormal and remained abnormal. Dr. Cruse does not discuss further the cause of Paul's encephalopathy except in the following statement:

Precipitating cause for his status [epilepticus] is not clear but surely is not unusual in children who are prone to seizures when they develop a febrile illness.

Id.

Dr. Gerald Erenberg for Petitioners:

Dr. Gerald Erenberg testified that Paul's significantly worsened condition was "part and parcel" of the intractible seizure disorder sustained as the result of his vaccine injury. Based on the extensive evidence documented and reported by Paul's treating neurologist, Dr. Cruse, Dr. Erenberg concludes that claims of neither a viral nor a metabolic basis for the change in Paul's neurological condition can be sustained with any degree of confidence. Over the intervening years, Paul had suffered many episodes of status epilepticus, many of which, as in this instance, were triggered by fever. Dr. Erenberg would not be surprised if the seizure of November 16, 1989 had in fact been precipitated by fever, no matter what its cause, because fever is known to trigger seizures in children who suffer seizure disorders. He acknowledges that neurological devastation following the November 16 episode could "possibly" have been caused by a malignant viral illness, but he believes that likelihood is not great given the unsuccessful efforts to isolate a virus, or virus titers, or, in fact, any other cause of the prolonged seizure -- bacteria, toxins, trauma, or metabolic disturbances. Letter of April 10, 1995 filed April 17, 1995; See also May 22, 1996 letter filed June 6, 1996. He believes a more apparent and logical explanation for Paul's brain damage was the difficult to control November 16, 1989 seizure capable of causing the tragic outcome.

Dr. Erenberg does not deny that infection can induce fever which could trigger a seizure, and cause transient damage to the organs (liver and kidney). But because the actual presence of infection was not established by hard evidence, and because prolonged status epilepticus is itself capable of causing serious neurological damage, particularly in an at-risk patient who is already suffering from significant brain damage, the status epilepticus, he believes, must be considered the more likely source of additional brain damage. Besides, he argues, an infection alone would not have resulted in such a severe outcome. Paul had had many such infections in the past without sequelae.

Dr. Arthur L. Prensky, for respondent:

Dr. Prensky acknowledges that severe damage to the central nervous system, including organ damage,

can occur with status epilepticus if that event is sufficiently severe. Report of Dr. Prensky filed March 27, 1997. For the following reasons, he is of the opinion that the event was insufficiently severe to destroy the large amounts of brain tissue observed in subsequent MRI and CT scans, and that an infectious agent, therefore, must have caused the damage. First, severe oxygen deprivation (hypoxia) capable of destroying tissue was never proved. Second, hypoxia is often accompanied by cerebral edema, and cerebral edema was not observed in this case. Third, one might expect less edema if the damage is ongoing over several days as one would expect in the case of an underlying infection. He concludes, therefore, that the absence of edema and failure to prove conclusively the presence of severe hypoxia argue against petitioners' claim that the status epilepticus caused the damage. He postulates that the type of damage observed in Paul's case could have been caused by toxic, metabolic, or infectious agents which are often difficult to detect or identify. Id. For these reasons, Dr. Prensky is convinced that an infectious agent, probably viral, is the cause of the encephalopathic event that left Paul in a devastated condition.

DISCUSSION

As discussed earlier, the controversy in this case is not whether Paul is entitled to compensation for a vaccine-related injury. That issue has been determined and conceded based on the court's findings of an on-Table injury. It is necessary now to determine the extent of Paul's vaccine-related injuries, that is, whether his post November 16, 1989 condition is related also to his vaccine injury. Petitioners bear the burden of proof.

The experts for both parties are well qualified, their proposed explanations, logical, and both theories are well within medical possibility. Their conclusions, obviously, are conflicting. Moreover, it appears that neither theory can be ruled out with any degree of certainty. The following statements illustrate: When asked whether the encephalopathic event of November 16, 1989 was caused by the prolonged seizure or by a viral infection, respondent's expert, Dr. Prensky answered:

"Well, I don't think I can give an either or. . . . It's hard to piece together blame. How much is status [epilepticus], how much is virus . . . ?"

Tr.II at 75, 110.

Petitioners' expert, Dr. Erenberg admits: "No, I do not believe it would be possible to know with absolute certainty . . . . We are dealing with probability." Tr.II at 12-13.

The Vaccine Act, however, does not require "certainty" to prove causation. The Federal Circuit in Bunting v. Secretary of DHHS, held --

The standard of proof required by the Act is simple preponderance of evidence; not scientific certainty. . . . It is not plaintiff's burden to disprove every possible ground of causation suggested by defendant nor must the findings of the court meet the standards of the laboratorian.[(12)]

The court considered carefully the probability that either source, alone, is capable of causing aggravated brain damage, or, as respondent's expert hypothesizes, that both might be implicated: "[I]n all probability the two in concert did act in concert [sic]." Tr.II at 115. Dr. Prensky would ascribe the majority of the damage to a viral illness "either acting with the seizure to cause a rise in fever or some other way directly affecting the brain. . . ." Id. Petitioners' expert, Dr. Erenberg, would ascribe "90-plus percent" of the neurological outcome to the prolonged seizure and "ten percent" to infection." Tr.II at 68-69.

In ascribing weight to the respective theories of causation, the court considered two factors to be of paramount relevance: First, the relatively speculative nature of respondent's theory that a viral infection attacked the brain; and second, persuasive evidence supporting the damaging effect of prolonged status epilepticus. Although one cannot rule out the possibility of a viral etiology (indeed, petitioners' expert agrees that it is possible), the evidence to support that hypothesis is insufficient to establish its probability.

The treating physicians reported a "a viral-like" illness prior the the onset of status epilepticus. Report of Dr. Robert Cruse of March 19, 1990, filed March 2, 1995 at 3-4. But they could not identify or establish the presence of the suspected virus or any other infection sufficient to cause massive brain damage. According to petitioners' expert, whatever caused Paul's prior illness, it may have and probably did elevate the child's temperature, (a common event known to trigger seizures particularly those persons with seizure disorders), but the court agrees with Dr. Erenberg that its overall effect was likely confined to decreased platelet count, transitory liver disfunction, and (possibly, but indirectly), transitory kidney dysfunction.(13) That damage was not permanent. Any further effect, to be specific, the probability of damage to the brain, is simply conjecture based on equivocal clinical symptoms without a shred of empirical or pathological evidence. Petitioners' evidence is more persuasive.

The massive seizure, unlike the presence of virus, was not hypothetical. Paul suffered many seizures over the years; this seizure was frightening. By all measures, it was worse than any previous seizure event. Based on evidence in medical literature that a severe and difficult to control seizure such as the one Paul experienced on November 16, 1989, would have placed him greatly at risk of additional injury, the court is convinced that more likely than not, it was the November 16, 1989 incidence of status epilepticus that led rapidly to further damage to his already compromised central nervous system.(14)

I will address the court's considerations in further detail. I considered carefully Dr. Prensky's reasons for suspecting a viral rather than a seizure-related cause. Dr. Prensky finds too little evidence in the medical records of hypoxia at a level significant enough to account for the extensive damage. The emergency room doctors provided good response after Paul reached the hospital to minimize damaging hypoxia by supplying oxygen. No evidence is available, however, about what may have happened in this regard prior to his arrival at the hospital. Tr.II at 52. Moreover, hypoxia is not the sole arbiter of brain damage during status epilepticus, according to the medical literature. P.Ex.1 at S39,S41; P.Ex. 3 at 16,19; see also Jerome Engle, Jr., Seizures and Epilepsy, "Epileptic Brain Damage," (1989) at 276. (This reference was not filed in this case, but the court notes that Dr. Engle's treatise support's Dr. Erenberg's testimony relating to mechanisms of damage.) Mechanisms of damage are not entirely understood in convulsive disorders, but mechanisms of damage other than hypoxia are described in Dr. Erenberg's testimony, tr. II at 53, and also in the in the exhibits cited above -- release during status epilepticus of destructive chemicals, excitatory amino acids, disturbances of systemic homeostasis, changes in blood chemistries, changes in the blood-brain barrier, etc. These mechanisms are suspected of exacerbating brain dysfunction during prolonged seizures:(15) Tr.II at 52-53.

Dr. Jerome Engle, in his learned treatise, states as follows:

[T]here is incontrovertible evidence that epilepsy itself can cause structural damage in the brain (epileptic brain damage). . . . The mechanism of action does not appear to require cytotoxic agents, generalized motor seizures, or occurrence of systemic factors leading to hypoxia, ischemia, or hypothermia. . . . Most recent evidence indicates that the endogenous excitatory amino acids, particularly glutamate, mediate damage and [other chemicals] activate proteases and lipases and leads to mitochondrial dysfunction[,] . . . neuronal swelling and could also contribute to cell damage.

Jerome Engel, Jr., Seizures and Epilepsy, "Epileptic Brain Damage," (1989) at 276.

Status [epilepticus] seizures can and do kill patients and threaten survivors with permanent neurological disability or other serious complications.

Editor's Note, Emergency Medicine Reports, Volume 10, Number 9, April 24,1989, at 65. Hearing Exhibit 4.

[T]ime is brain . . . and in some high-risk patients, seizure activity must be aborted well before the traditionally accepted time limit of 60 minutes to prevent mortality and excessive morbidity.[(16)]

Emergency Medicine Reports, Hearing Exhibit 4, Id.[(17)]

Respondent's expert, Dr. Prensky, acknowledges that status epilepticus can produce brain damage and that the longer the status lasts, the more likely an individual is to get brain damage. He adds, however, that it may not necessarily happen in every instance.[(18)] Tr. at 84. The court takes note of the following statements by Dr. Jean Aicardi in his authoritative treatise (Dr. Prensky acknowledges that Dr. Aicardi is a highly respected expert in this field):

Convulsive status may end in death or may leave severe mental and/or neurological sequelae that appear to result, at least in part, from the convulsive activity itself, irrespective of its causes.

[C]onvulsive status is the major emergency in the therapy of epilepsy because of its life-threatening character and the high incidence of its sequelae.

Jean Aicardi, Epilepsy in Children: International Review of Child Neurology, Chapter 16, "Status Epilepticus," 1986, at 258, and 251 respectively.

CONCLUSIONS

Petitioners have met their burden. It is reasonable to conclude that in a child at great risk, the catastrophic seizure sustained at the age of seven, more likely than not, was "part and parcel" of his vaccine-related seizure disorder and cannot be separated from it. Respondent has failed to show that in this particular case, an infection was the agent principally responsible for causing Paul's condition. The court concludes that Paul Lurtz is entitled to compensation for his vaccine-related residual seizure disorder and that his present condition, more likely than not, is the sequela of that injury. The parties are directed to enter into discussion as to the amount of compensation required for Paul's future care and rehabilitation.

IT IS SO ORDERED.

E. LaVon French

Special Master

1. Documents from the prior civil action revealed no new evidence. Respondent, however, requested the court's reconsideration of discrepancies in medical records that might suggest an onset of symptoms four weeks after the date claimed. The court had not overlooked the controversial notations in making its factual findings. Nevertheless, the court agreed to a second review and concluded that the first seizure

occurred, in fact, on July 12, 1982 as claimed and that a grand mal seizure on August 12, 1982, four weeks later, was the second seizure. The court concluded further that the discrepant medical history recorded on August 12, 1982 did not take into consideration the earlier event of July 12, 1982 because petitioners had not recognized it to be a focal seizure. The court is not persuaded by respondent's argument that failure to find contemporaneous documentation of the July 12, 1982 episode requires the court to conclude that it did not occur. The weight of evidence supports a finding that the first seizure was not recorded in contemporaneous medical records simply because it was not identified as such nor reported at that time. Later medical records do, in fact, report onset of seizures on July 12, 1982, confirming onset as claimed.

2. Mrs. Lurtz' neighbor, Mrs. Means, testified that one could not get the baby's attention. He appeared as if he were in the "ozone," but she did not think of it as a seizure at that time. Tr. at 93. This incident is not described in contemporaneous medical records although it is mentioned in the later medical histories recorded by Paul's treating physicians.

3. Concerning the August 12 grand mal seizure, Mrs. Lurtz testified that she was unaware of any possibility that the incident she had observed on July 12, 1982, four weeks earlier, may have been a seizure or that it could be related in any way to the second, more dramatic incident that occurred on August 12, 1982. The second seizure, a grand mal seizure, was obvious and more consistent with what she considered a seizure to be.

4. Status epilepticus is defined as "rapid succession of epileptic spasms without intervening periods of consciousness." Dorland's Pocket Medical Dictionary, 24th ed. at 559.

5. It is prudent to explain the statement above by citing its basis. Many experts in vaccine cases have testified that mental retardation and other neurological deficits are likely to accompany intractable seizure disorders in young children, although not in every case. The outcome of convulsive status epilepticus (SE) is variable, however, a series of studies by renowned epileptologist, Jean Aicardi, found a high incidence of mental deficits following SE. Studies by Fujiwara, et al., found similar results and a high incidence of motor damage as well. In the Aicardi series, seizures occurring after SE were mainly of a type "usually associated with brain damage." Aicardi concluded: "Convulsive seizure may end in death or may leave severe mental and/or neurological sequelae that appear to result, at least in part, from the convulsive activity itself, irrespective of its causes." Jean Aicardi, Epilepsy in Children, Raven Press, N.Y., at 243, 258. According to Aicardi, poor prognosis in some types of seizure disorders is often associated with early age of onset. Id. at 6, 36, 52, and 160.

6. Prior to inducing coma, the doctors administered twenty-two (22) milligrams of valium over a period of 54 minutes. The seizure was controlled, however, only after the administration of the sodium pentothal. Thereafter, intermittent seizures continued to occur. Report of Dr. Prensky of March 17, 1997, filed March 27, 1997, at 1.

7. Herpangina is defined as "an acute infectious disease . . . affecting the mucous membranes of the throat . . . ." Dorland's Illustrated Medical Dictionary, 27th Ed. at 759.

8. Dorland's Illustrated Medical Dictionary, 27th Ed. at 759, defines anuria as "complete suppression of urinary secretion by the kidneys." at 107.

9. A four-stage coma scale (established by Huttenlocher (1972)) provides that "[a] child in stage 4 coma [the most severe stage] is flaccid, unresponsive to painful stimuli and has no deep tendon reflexes, pupillary reactions, or spontaneous respirations. In stage 3 coma the child has decerabrate posturing,

either spontaneously or in response to deep pain." Kenneth F. Swaiman, Pediatric Neurology, Principles and Practice 2nd Ed. Vol. 1, 187.

10. Letter to Dr. Burns of March 19, 1990, filed on March 2, 1995 at 3.

11. Neither expert proposes that the Herpes Stomatitis caused Paul's condition.

12. Bunting, 931 F.2d 867 at 873 (Fed. Cir. 1991)(citing Tinnerholm v. Parke Davis & Co., 285 F.Supp. 432, 440 (S.D.N.Y. 1968), aff'd, 411 F.2d 48 (2d Cir. 1969)).

13. See Tr. II at 26-28.

14. See Claude G. Wasterlain, et al., "Pathophysiological Mechanisms of Brain Damage from Status Epilepticus," Epilepsia, Vol. 34, Supp. 1, 1993, at S37, Hearing Exhibit 1. ("the degree of brain damage can increase with the duration of the seizure. . . .") ("Convulsive, tonic-clonic SE rapidly leads to severe brain damage." at S39).

15. See Eric Lothman, "The biochemical basis and pathophysiology of status epilepticus," May 1990 Neurology, 40, at 19, Hearing Exhibit 3. See also Claude G. Wasterlain, et al., "Pathophysiological Mechanisms of Brain Damage from Status Epilepticus, Epilepsia, Vol. 34, Supp. 1, 1993, at S49, Hearing Exhibit 1.

16. Paul's seizure persisted for a period of two hours.

17. Dr. Erenberg cites research in animal models to support his belief that damage can occur even if hypoxia is mild. See, e.g., medical articles filed as Hearing Exhibits 1, 2, 3, and 4. The court agrees with respondent that research relating to seizures in animal (primate) models, does not necessarily prove that the same effect would be expected in humans as well. His argument is cited here as being of mild interest only. The court does not rely upon animal research models in support of its decision.

18. See also Dr. Erenberg's testimony: "The longer the seizure is ongoing, the greater the risk of damage. . . ." Tr. at 19.