STATE OF FLORIDA
DIVISION OF ADMINISTRATIVE HEARINGS

JODY WORKMAN AND BRIAN WORKMAN, on behalf of and as parents and natural guardians of ALYSSA WORKMAN, a deceased minor,	)	
Petitioners,	)	
vs.	)	Case No. 03-4418N
FLORIDA BIRTH-RELATED NEUROLOGICAL INJURY COMPENSATION ASSOCIATION,	)	
Respondent.	)	

FINAL ORDER

Pursuant to notice, the Division of Administrative Hearings, by Administrative Law Judge William J. Kendrick, held a final hearing in the above-styled case on August 31, 2004, in Inverness, Florida.

APPEARANCES

For Petitioners: Brian Workman, pro se
Jody Workman, pro se
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For Respondent: Donald H. Whittemore, Esquire
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STATEMENT OF THE ISSUES

At issue is whether Alyssa Workman, a deceased minor, qualifies for coverage under the Florida Birth-Related Neurological Injury Compensation Plan.

PRELIMINARY STATEMENT

On November 25, 2003, Jody Workman and Brian Workman, on behalf of and as parents and natural guardians of Alyssa Workman (Alyssa), a deceased minor, filed a petition (claim) with the Division of Administrative Hearings (DOAH) for compensation under the Florida Birth-Related Neurological Injury Compensation Plan (Plan).

DOAH served the Florida Birth-Related Neurological Injury Compensation Association (NICA) with a copy of the claim on November 26, 2003; and on February 27, 2004, NICA filed a Motion for Summary Final Order, and on March 10, 2004, an Amended Motion for Summary Final Order; predicated on the opinion of their medical experts that Alyssa did not suffer a "birth-related neurological injury," as that term is defined by the Plan. § 766.302(2), Fla. Stat. (2001)[1] By Order of May 4, 2004, NICA's motions were denied, and on May 13, 2004, NICA filed its Response to Petition for Benefits, and averred that "the instant claim is not compensable as the injury does not meet the definition of a 'birth-related neurological injury' as defined in Section 766.302(2), Florida Statutes." By Notice of Hearing dated

May 14, 2004, a hearing was scheduled for August 31, 2004, to resolve whether the claim was compensable.

At hearing, Jody Workman, Brian Workman, Sharon Richardson (Alyssa's paternal grandmother), and Peggy Lane (Alyssa's maternal grandmother), testified on Petitioners' behalf, and Petitioners' Exhibits 1, 2, and 3A-3K were received into evidence.[2] Respondent called no witnesses, but offered Respondent's Exhibits 1-7, which were received into evidence.[3]

The transcript of the hearing was filed September 30, 2004, and the parties were accorded 10 days from that date to file proposed final orders. Both parties elected to file such proposals (Respondent on October 11, 2004, and Petitioners on October 18, 2004), and they have been duly considered.

FINDINGS OF FACT

Preliminary findings

1. Petitioners, Jody Workman and Brian Workman, are the natural parents and guardians of Alyssa Workman, a deceased minor. Alyssa was born a live infant on May 30, 2002, at Seven Rivers Community Hospital, a hospital located in Crystal River, Florida, and her birth weight exceeded 2,500 grams.

2. The physician providing obstetrical services at Alyssa's birth was Rose Sobel, M.D., who, at all times material hereto, was a "participating physician" in the Florida Birth-Related

Neurological Injury Compensation Plan, as defined by Section 766.302(7), Florida Statutes.

Coverage under the Plan

3. Pertinent to this case, coverage is afforded by the Plan for infants who suffer a "birth-related neurological injury," defined as an "injury to the brain or spinal cord . . . caused by oxygen deprivation or mechanical injury occurring in the course of labor, delivery, or resuscitation in the immediate postdelivery period in a hospital, which renders the infant permanently and substantially mentally and physically impaired." § 766.302(2), Fla. Stat. See also §§ 766.309 and 766.31, Fla. Stat.

4. In this case, Petitioners are of the opinion that Alyssa suffered a "birth-related neurological injury" because, in their view of the evidence, she suffered an injury to the brain caused by oxygen deprivation occurring in the cause of labor, delivery, or resuscitation, that ultimately produced severe seizure activity and which, together with the medications required to abate her seizure activity, led to her death. In contrast, NICA is of the view that the evidence fails to support the conclusion that Alyssa suffered a "birth-related neurological injury" since there was no competent proof to support a conclusion that, more likely than not, Alyssa suffered an injury to the brain caused by oxygen deprivation in the course of labor, delivery, or

resuscitation, or that her seizure activity was caused by such an injury, as opposed to another etiology.

Alyssa's birth and postnatal course

5. At or about 6:00 a.m., May 30, 2002, Mrs. Workman, with an estimated delivery date of May 21, 2002, and the fetus at 41 2/7 weeks' gestation, presented to Seven Rivers Community Hospital, for augmentation of labor. At the time, Mrs. Workman's membranes were noted as intact, and vaginal examination revealed the cervix at 2 centimeters dilation, effacement at 50 percent, and the fetus at -2 station. Uterine contractions were noted as mild, occasional (approximately every 7 minutes), and external fetal monitoring revealed a reassuring fetal heart rate, with a baseline in the 130-beat per minute range.

6. At about 6:45 a.m., Petocin augmentation was started, and Mrs. Workman's labor slowly progressed until 4:37 p.m., when complete cervical dilation was noted, and Alyssa was delivered at 5:03 p.m. Of note, in so far as the Labor Progress Chart reflects, as well as the fetal monitor strips and Anesthesia Report, external fetal monitoring continued until approximately 5:00 p.m., and continued to reveal a reassuring fetal heart rate. Of further note, at 9 pounds 1 1/2 ounces (4,125 grams), Alyssa was large for gestational age (LGA), and delivery was complicated by a shoulder dystocia, which was relieved with suprapubic pressure and McRoberts maneuver, and a nuchal cord x1, around the

neck, which was relieved as Alyssa's neck was delivered.[4] Alyssa's mouth and nose were suctioned while her head was in the perineum.

7. At delivery, Alyssa was depressed (cyanotic, floppy, with no respiratory effort), and was placed in a radiant warmer, where her oropharynx was suctioned, and she was given blowby oxygen; however, she failed to respond, and a code blue was called for the resuscitation team. On arrival of the team, Alyssa's oropharynx was again suctioned and she was given bag/mask ventilation, with first gasp noted at 3 minutes and sustained respiration noted at 4 minutes. (See Labor and Delivery Summary and New Born flow sheet.) Apgar scores were recorded as 2 and 8, at one and five minutes, respectively.[5]

8. Contrasted with the conclusion one would draw from the Labor and Delivery Summary and New Born flow sheet, discussed supra, Petitioners offered proof, through the testimony of Mr. and Mrs. Workman, Mrs. Richardson and Mrs. Lane, that Alyssa's first gasp (at 3 minutes) followed the start of resuscitative efforts by the team, and did not represent an elapsed time from her birth. Petitioners also offered proof through the same witnesses, that in their opinion the team did not start resuscitative efforts until three or four minutes after birth. According to Petitioners, this delay caused brain injury

to Alyssa, which resulted in the seizures or epileptic activity discussed infra.[6]

9. Here, it is unnecessary to resolve whether three or four minutes elapsed following Alyssa's delivery before the team began resuscitative efforts with bag/mask ventilation, as advocated by Petitioners, or whether that activity began earlier, as one would conclude from the delivery records, since the proof fails to support the conclusion that, more likely than not, any oxygen deprivation Alyssa may have suffered caused brain injury. In so concluding, it is noted that Alyssa's one-minute Apgar score documented a reassuring heart rate; her five-minute Apgar score documented sustained respirations; her newborn assessment, at 5:30 p.m., was grossly normal; her newborn course was without significant incident; and she was discharged with her mother on May 31, 2002, at one day of age. It is also noted that, except for two episodes of startle reflex (at 3 months and 4 months of age), which may evidence seizure activity, but is also common in children, and a suspected delay in gross motor skill development, noted at 9 months of age, Alyssa's early development was appropriate; and an MRI of the brain done March 23, 2003, discussed more fully infra, was normal. Finally, and most importantly, the record is devoid of any expert medical testimony or other competent proof that would support a conclusion that, more likely than not, Alyssa suffered a brain injury caused by

oxygen deprivation during labor, delivery or resuscitation. See Wausau Insurance Company v. Tillman, 765 So. 2d 123 (Fla. 1st DCA 2000)("Because the medical conditions which the claimant alleged had resulted from the workplace incident were not readily observable, he was obliged to present expert medical evidence establishing that causal connection."); Ackley v. General Parcel Service, 646 So. 2d 242 (Fla. 1st DCA 1995)(determining cause of psychiatric illness is essentially a medical question, requiring expert medical evidence); Vero Beach Care Center v. Ricks, 476 So. 2d 262, 264 (Fla. 1st DCA 1985)("[L]ay testimony is legally insufficient to support a finding of causation where the medical condition involved is not readily observable.") For similar reasons, the proof failed to support the conclusion that the seizures Alyssa subsequently experienced were related to a brain injury that occurred during labor, delivery, or resuscitation.

Alyssa's subsequent development

10. Except as heretofore noted, Alyssa's development was appropriate until approximately 10:10 p.m., March 22, 2003, when, at 9 months of age, she evidenced sudden onset of seizure activity, preceded by vomiting, and was transported (by her family) to Seven Rivers Community Hospital, and then by helicopter to Shands, where she was admitted at 1:55 a.m., March 23, 2003, and remained until April 16, 2003, when she was

discharged to her parents' care. Alyssa's hospital course was documented in her Discharge Summary, as follows:

> ADMISSION DIAGNOSIS: Status Epilepticus (complex partial)[7]
>
> DISCHARGE DIAGNOSIS: Same; Focal motor seizures.
>
> SERVICE: Pediatric Neurology.
>
> * * *
>
> PROCEDURES: Endotrachial Intubation, Pentobarbital Infusion with continuous EEG monitoring, MRI of the brain, EEG, ICC line placement, Chest x-rays.
>
> * * *
>
> HOSPITAL COURSE: Alyssa Workman is a 10-month-old, white female who was admitted to Shands Teaching Hospital due to intractable seizures manifested by unresponsiveness, head/eye deviation to the right and right hemibody clonic activity. Hospital course will be presented by pertinent problems and subsequent hospital outcomes.
>
> (1) Neurology. The patient was initially admitted in status epilepticus. Alyssa had failed IV loading doses of Ativan, Fosphenytoin, and Phenobarbital. Alyssa failed continuous IV midazolam infusion. She was placed in a pentobartital coma [to abort status epilepticus] and subsequently intubated. She was discontinued from the continuous pentobarbital infusion after electrographic seizure activity in the left temporal/parietal region was suppressed for greater than 24 hours with the initiation of phenobarbital. Tegretol and Topamax were added to the regimen to allow for discontinuation of Phenobarbital. Due to continued focal motor clonic activity of the right upper extremity with preserved mental status, Alyssa was transferred to the

Epilepsy Monitoring Unit for further classification of her seizures. Continuous EEG monitoring demonstrated brief, semi-rhythmic delta frequency discharge in the left parietal region without rhythmic spike/wave discharge. MRI of the brain was performed on three occasions; the first two studies were normal, the last demonstrated evidence of diffuse atrophy.[8] She was discharged home with AED regimen of Tegretol, Topamax, and clonazepam. The patient will follow-up in Neurology Clinic.

(2) Infectious disease. The patient underwent a rule out sepsis to determine if an infectious disease agent could possibly be causing the etiology of her disorder. The patient was afebrile throughout the remainder of her hospitalization and received a sepsis workup. She also received ten days of ceftriaxone. Blood, CSF and urine cultures were all insignificant. HSV and Bartonella studies were obtained and were negative. . . . Her last CBC was obtained on 03/28/2003 and was within normal limits.

(2) FEN. The patient was initially maintained on NG feeds secondary to intubation and poor p.o. intake. At discharge, the patient was tolerating a p.o. regular diet without any complications and requiring no maintenance IV fluids. Her last BMP was obtained on 04/14/2003 and was within normal limits.

(3) Respiratory. The patient was intubated secondary to status epilepticus from 03/24/2003-04/01/2003. Following extubation, the patient has been stable on room air. At discharge, the patient is stable on room air and requiring no supplemental oxygen.

(4) Gastroenterology. The patient had serial LFTs measured to determine secondary effects of anti-epileptic medications. Her last LFTs were within normal limits.

> (5) Metabolic. A metabolic workup was undertaken to determine if this could possibly be causing the patient's myoclonic jerks. The patient had an ammonia level of 24 that was within normal limits and a serum amino acids that were within normal limits. Urine organic acids are pending at discharge. CSF lactate and glycine are pending at discharge. A pyruvate level was obtained and was less than 0.10 which is low. This will be followed up in Neurology Clinic.
>
> CONDITION: Stable.
> DISPOSITION: Home.
>
> * * *
>
> DISCHARGE MEDICATIONS: Tegretol 100 mg p.o. t.i.d., Topamax 25 mg p.o. qam, 50 mg p.o. q.p.m. and clonazepam 0.125 mg p.o. b.i.d.
>
> FOLLOW-UP: Follow-up will occur in the Pediatric Neurology Clinic with Dr. Suhrbier . . . .

11. Following discharge from Shands, Alyssa was followed at the Pediatric Neurology Clinic (by Doctors Paul Carney and David Suhrbier) for intractable epilepsy manifested by right upper extremity focal motor seizures, which spread to the trunk and lower extremity. During this time, Alyssa followed a number of medication trials with varying success, but the seizures persisted and on July 24, 2003, she again presented to Seven Rivers Community Hospital in status epilipticus. There, despite Ativan and Dilantin loading, the hospital was unable to break her seizures, and she was transferred to Shands on July 25, 2003, for further management.

12. Alyssa remained at Shands until August 8, 2003, when she was discharged. Alyssa's hospital course was documented in her Transfer Summary (from the pediatric intensive care unit to the floor nursery) of August 8, 2003, shortly before her discharge, as follows:

> Alyssa is a 14 mo[nth female with] known [history of] idiopathic[9] intractable[10] epilepsy (now being called Lennox-Gastant Syndrome) that initially presented to Seven Rivers Hospital on 7/24/03 [with] focal status epilepticus . . . SRH unable to break [seizures with] Ativan and Dilantin load (had prolonged course in their ER) before being transferred to STH [Shands Teaching Hospital] for further m[anagement] . . . . Once here needed to be placed in pentobarbital coma to achieve burst suppression and lysis [abatement] of seizures . . . . Intubated while in PB coma . . . . [W]as extubated on 8/3/03 and is currently on RA [room air] and stable. Had HD [hemodynamic] instability initially [with] PB induction that required Dopamine infusion, but was quickly weaned and remained HDS (hemodynamically stable) for the rest of her course . . . . Alyssa's neuro exam is not normal and is notable for almost constant myoclonic m[ovement] of head and extremities . . . . She is globally hypotonic.

Principal diagnosis at discharge was generalized seizure disorder, with a secondary diagnosis of developmental delay.

13. While hospitalized, Alyssa underwent a repeat MRI of the brain (with and without contrast) which was "completely normal."[11] The MRI was performed on August 5, 2003, and reported, as follows:

FINDINGS:

Clinical Indication: Intractable seizures.

Comparison: MRI of the brain 7/22/03

* * *

Findings:

The brain density is appropriate for a young child. There is minimal retained interstitial water in the cerebral white matter. Brain formation is normal. Myelination is appropriate for age with evidence of myelination in corticospinal tracts, visual pathways and corpus callosum. Ventricular size and sulcal pattern are within normal limits. There is no evidence of mass lesion, hydrocephalus, intra or extra axial fluid collection to account for symptoms, as questioned. There is no evidence of neural migration disorder. The paranasal sinuses and oto-mastoid air cells are normally developed and aerated without evidence of acute or chronic mucoperiosteal thickening of intrasinus fluid.

Incidentally noted is extracranial soft tissue swelling in the high left parietal convexity region.

MR venography demonstrates all the major dural sinuses to be patent. There is no evidence of narrowing.

Comparison with the prior exam demonstrates no apparent interval change.

IMPRESSION:

Negative MRI and MRV of the brain.

14. Alyssa was discharged on a regimen of oral steroids, Topamax, and Klonopin, but nevertheless developed near continuous

focal clonic activity of the left hemibody with convulsive activity lasting for hours at a time. Purposeful use of the left upper extremity and lower extremity significantly declined along with her cognitive function. Keppra was added to the regimen without significant improvement, and on September 8, 2003, Alyssa was readmitted to Shands for the purpose of further diagnostic investigation and treatment of her seizure disorder.

15. During the course of her hospitalization, Alyssa was administered intravenous immunoglobulin (IVIG) as a potential therapy for her intractable seizure activity, which significantly reduced the frequency of her convulsive attacks. Further diagnostic testing to identify an idiology for Alyssa's seizure activity was unrevealing, and she was discharged on September 16, 2003.

16. Following discharge, Alyssa had a progressively declining course, her seizures persisted, and on October 22, 2003, she presented at Shands' Pediatric Neurology Clinic for evaluation by Dr. Suhrbier. Dr. Suhrbier summarized the results of his evaluation, as follows:

> On today's evaluation, Alyssa is somnolent but arousable. She does not significantly fixate on targets or track purposefully. Pupils are mildly dilated and sluggishly reactive. Optic discs are sharp. Extraocular movements are conjugate but demonstrate intermittent left beating nystagmus . . . . Alyssa does not vocalize . . . . Neurologic: Deep tendon reflexes are

> 3/4 in the upper and lower extremities with absent Babinski sign. Motor examination: Severe hypotonia with minimal spontaneous movement.
>
> * * *
>
> Impression: 17-month female with intractable epilepsy, progressive developmental delay, evidence of hepatocellular dysfunction, and failure to thrive with oral motor dysfunction. Currently, she is in a stuporous state which I fear may represent non-convulsive status epilepticus.
>
> Plan: 1) We will obtain comprehensive metabolic panel . . . today in clinic. 2) Will transfer to EEG laboratory for STAT EEG. 3) We will admit to the Pediatric IMC for clinical observation. We will consult GI for input regarding etiology of elevated liver enzymes as well as assistance with nutritional status.

17. Alyssa was admitted to Shands later that day, and discharged on November 15, 2003. Alyssa's hospital course was described in her Discharge Summary, as follows:

> DIAGNOSIS: (1) Seizures. (2) Pancreatitis. (3) Developmental delay. (4) Feeding intolerance.
>
> * * *
>
> ADMISSION HISTORY AND PHYSICAL: Alyssa is an 18-month-old Caucasian female with a history of intractable epilepsy and developmental delay who presented to the Neurology Clinic on 10/22/03 with a six-day history of acting sleepy, not smiling, not crying, and "looking drugged" per father. The father notes that the patient had a fever and vomiting six days prior to admission and he felt that Alyssa had the flu. However, she had never "perked up" after the illness though the fever had

been gone for several days. The patient had also displayed several of her clinical seizures which involve left-sided jerks. The father noticed approximately eight of these seizures during the past six days, much fewer than her usual. In the Neurology Clinic on the date of admission, Alyssa was found to be in nonconvulsive status epilepticus. She was sent to the Emergency Room and given Ativan times two and loaded with phosphenytoin 20 mg/kg. She was still having synchronized spiking wave discharges, so was sent to the PICU for further care including pentobarbital coma. The father describes Alyssa's baseline activity at home as being "flacid." She cannot sit on her own, crawl or hold a bottle, has approximately two words, will open her eyes and fix, and has been NG fed due to failure to thrive.

HOSPITAL COURSE: Alyssa was admitted to the Pediatric ICU. She was placed in a pentobarbital coma, intubated, and vital signs were monitored closely . . . . For further details of hospital admission, please refer to the systems evaluation as follows: (1) Respiratory: When pentobarb, was stopped, Alyssa was weaned from the ventilator without difficulty. She needed frequent suctioning of her secretions. At the time of discharge, Alyssa had no elicited gag reflex. She had frequent episodes of desaturation requiring suctioning and oxygen. At the time of discharge, suctioning device and home oxygen had been obtained for father. (2) Cardiovascular: Alyssa briefly required pressors while on the pentobarb in pentobarb coma, however, her blood pressure remained stable when off of pentobarb. She did have occasional episodes of poor peripheral perfusion requiring normal saline boluses, but otherwise was clinically stable. (3) Neurological System: Alyssa was weaned from the pentobarb coma on 10/30/03 due to blood pressure instability. A brain MRI was obtained which demonstrated generalized and focal atrophy. The focal atrophy was noted

in the hippocampal regions. White matter distribution showed a normal pattern of arborization but a generalized decrease in volume . . . . Ophthalmology consult was obtained which showed normal eye anatomy. Muscle biopsy was performed by Peds Surgery which showed no evidence of mitochondrial enzyme deficiency by histopathology; however, enzyme studies were still pending at the time of this summary. A whole blood sample was sent to Athena Diagnostics for mitochondrial DNA evaluation which was still pending at the time of this discharge. A liver biopsy was obtained by Peds Surgery which showed histologic evidence of TPN changes but no specific abnormalities. Again, further studies are pending at the time of this summary . . . . At the time of discharge, Alyssa was requiring approximately one dose of Ativan every 24 to 48 hours. There was no evidence of subclinical seizure activity at the time of her discharge. She was sent home on Celontin, Mebaral, and pyridoxine as well as p.r.n. rectal Diastat. (4) GI System: Alyssa was kept on hyperalimentation for the majority of her hospitalization. On 11/07/03 an NJ tube was placed and feeds as Tolerex (a low-fat formula), were begun at trickle rates. Alyssa tolerated this trickle formula very well and her lipases came down to a low of 200 at the time of her discharge. However, as feeds were increased toward maintenance fluid goals, Alyssa developed abdominal distention and had several episodes of vomiting associated with some respiratory distress. Abdominal distention usually resolved with holding the feeds. Alyssa's father requested that an NG tube be placed, however, staff felt that this was against Alyssa's best interest since she had no gag reflex and would . . . be unable to protect her airway. A NJ tube was replaced and feeds were restarted with Boost in the hopes that she would tolerate this formula more readily. However, Alyssa again developed abdominal distention. On the day of discharge feeds were held and abdominal distention resolved.

> The father became very agitated with the feeding regimen and felt that he could feed Alyssa at home with more success. He was advised about the risk of dehydration and aspiration. However, he still chose after lengthy discussions with Dr. Pineda, Pediatric GI, and Pediatric Neurology to sign Alyssa out AMA [against medical advice]. He was told absolute minimum fluid goals for each day and was also reassured that he could bring Alyssa back at any time if he had problems at home . . . .

18. The brain MRI noted in the Discharge Summary was performed October 26, 2003, and reported, as follows:

> FINDINGS:
>
> History: Patient with intractable epilepsy presenting with increasingly poor control of seizures.
>
> * * *
>
> Compared to a prior study of 8/5/2003 patient now appears to have a fairly large collection of fluid posteriorly in the deeper layers of the scalp. No obvious blood products are seen. There is no enhancement. Perhaps this is a area of prior scalp injury related seizures which was previously a deep scalp hematoma which now has turned into a seroma. There is no communication of this with intracranial structures and there is no evidence of skull injury.
>
> The study as on prior exam shows a generally atrophic appearance of the brain which can be caused in part by seizure medications. No focal abnormalities are present. There is a general diminished volume of white matter although there does appear to be considerable myelination present. The white matter which is present appears relatively normal with there being some extension into the subcortical regions and arcuate bundles so

that while the white matter volume may be diminished in its overall pattern development is not abnormal and this may just reflect generalized brain atrophy.

The temporal pole poles of both lateral ventricles are dilated. The hippocampal formations appear atrophic bilaterally more on the right side. The parahippocampal gyri also appears small and white matter volume in this region is diminished. It is uncertain whether this is a primary or secondary effect of the patient's seizure disorder. The fornices are small and the mamillary bodies are difficult to identify.

Contrast was given and there is no evidence of abnormality that would explain the patient's symptoms. There is no abnormal meningeal or brain enhancement.

Diffusion weighted images of the brain show possible areas of minimal predominantly cortical and subcortical areas of restricted diffusion in the posterior temporal parietal regions. This is a nonspecific finding and quite subtle it could be related to generalized prolonged seizure activity. No altered signal intensity on T2-weighted images is seen in these areas.

IMPRESSION:

Really no change from prior imaging studies except for a collection of fluid in the deep layers of the scalp probably due to a resolving scalp hematoma presumably related to scalp trauma due to seizures. No evidence of acute blood products within this collection.

Other findings described above. Their relationship to the patient's seizure disorder is uncertain. No definite abnormalities seen on diffusion weighted images . . . but subtle changes reflecting seizure activity could be present.

19. On November 20, 2003, Alyssa suffered cardiorespiratory arrest. EMS was contacted and she was transported to Seven Rivers Community Hospital, during which CPR was initiated (total CPR time greater than 2 hours) including intubation. At the hospital, Alyssa was found to have pulseless ventricular fibrillation, with arterial blood gas pH of 7.068. Thereafter, Alyssa was transported to Shands by helicopter, where she died at 10:37 p.m., November 20, 2003. A postmortem examination at Shands included the following observations:

> NEUROPATHOLOGIC DIAGNOSES:
>
> 1. Diffuse organizing and organized hypoxic-ischemic encephalopathy (see note).
>
> 2. Diffuse cerebral white matter loss (see note).
>
> 3. Widespread myofiber atrophy/hypotrophy in skeletal muscle.
>
> NEUROPATHOLOGIC NOTE:
>
> This 18 month old female, with a history of nuchal cord and asphyxia at birth (Apgars reported 2 and 8), suffered subsequent developmental delay which apparently left her flaccid and unable to sit, crawl, or hold her bottle. She was reportedly admitted to hospital on 10/22/03 in non-convulsive status-epilepticus, for which she was intubated and placed in Phenobarbital coma. Following extubation on 11/2, she apparently continued to be in some respiratory distress, having difficulties with aspiration and requiring frequent suctioning. She was reportedly discharged against medical advice on 11/15. Apparently during the evening of

11/19, the parents reported that she had become somnolent and was experiencing mild respiratory distress with abdominal distention, then, early in the morning of 11/20, she had to be emergently transported, with CPR, to hospital, and was then transferred to Shands-UF via helicopter, where she was reported to have had spontaneous respirations but was hypothermic. During the evening of 11/20, her condition continued to deteriorate and, after discussing the infant's poor prognosis with family, maximal support was discontinued and she died. At autopsy, the infant's brain exhibited histologic evidence of marked and diffuse hypoxic-ischemic encephalopathy, with features suggesting between 10 and 20 days duration. In addition, there were changes in hippocampus, which suggested the possibility of at least one superimposed more recent (less than a few days old) hypoxic-ischemic event. All of these changes had also been associated with gross evidence of an underlying diffuse white matter loss, presumably causing the moderate, apparently "*ex vacuo*" (in the absence of demonstrable CSF obstruction) ventricular enlargement. It should be noted that diffuse white matter injury of this type could occur in the setting of perinatal hypoxia. Although it appears that this infant was weak and flaccid prior to her admission on 10/22, the widespread myofiber atrophy/hypotrophy in skeletal muscle seen post-mortem appeared similar to that in a biopsy (during life) and was probably not due to denervation (i.e., "lower motor neuron"). The myofiber atrophy may have represented disuse-like effects due to loss of upper motor neuron function as a result of the older (and possibly more recent) diffuse hypoxic-ischemic brain injury.

The cause and timing of Alyssa's neurologic impairments

20. To address the cause and timing of Alyssa's neurologic impairments, the parties offered a report of Postmortem Examination (Petitioners' Exhibit 1); a report by Dr. Suhrbier, dated November 18, 2003, and a discharge summary, dated November 15, 2003 (Petitioners' Exhibit 2); photographs of Alyssa (Petitioners' Exhibit 3A-3K); medical records related to Alyssa's birth and subsequent development (Respondent's Exhibits 1-5); an affidavit, curriculum vitae, and two reports by Michael Duchowny, M.D. (Respondent's Exhibit 6); and an affidavit and report by Donald Willis, M.D. (Respondent's Exhibit 7). Petitioners also presented the testimony of Mr. Workman, Mrs. Workman, Mrs. Richardson (Alyssa's paternal grandmother), and Mrs. Lane (Alyssa's maternal grandmother), as to the events surrounding Alyssa's birth and, in the case of Mr. Workman, his perception of a casual relationship between the events surrounding Alyssa's birth and her neurologic impairment. Notably, the medical records do not reveal an etiology for Alyssa's encephalopathy and epilepsy; no competent medical testimony was offered to support a conclusion that, more likely than not, Alyssa suffered an injury to the brain caused by oxygen deprivation during labor, delivery or resuscitation that resulted in neurologic impairment[12]; and the lay testimony of Mr. Workman, and any of his witnesses, regarding

a causal relationship between the events surrounding Alyssa's birth and her neurologic impairment is legally insufficient to support a finding regarding the cause or timing of Alyssa's neurologic impairment. See Wausau Insurance Company v. Tillman supra; Ackley v. General Parcel Service, supra; Vero Beach Care Center v. Ricks, supra. Consequently, while the proof suggests that Alyssa may have suffered oxygen deprivation during labor, delivery, or resuscitation, it fails to support the conclusion that, more likely than not, any oxygen deprivation she may have suffered caused brain injury, or resulted in neurologic impairment.

CONCLUSIONS OF LAW

21. The Division of Administrative Hearings has jurisdiction over the parties to, and the subject matter of, these proceedings. § 766.301, et seq., Fla. Stat.

22. The Florida Birth-Related Neurological Injury Compensation Plan was established by the Legislature "for the purpose of providing compensation, irrespective of fault, for birth-related neurological injury claims" relating to births occurring on or after January 1, 1989. § 766.303(1), Fla. Stat.

23. The injured "infant, her or his personal representative, parents, dependents, and next of kin," may seek compensation under the Plan by filing a claim for compensation with the Division of Administrative Hearings. §§ 766.302(3),

766.303(2), 766.305(1), and 766.313, Fla. Stat. The Florida Birth-Related Neurological Injury Compensation Association, which administers the Plan, has "45 days from the date of service of a complete claim . . . in which to file a response to the petition and to submit relevant written information relating to the issue of whether the injury is a birth-related neurological injury." § 766.305(3), Fla. Stat.

24. If NICA determines that the injury alleged in a claim is a compensable birth-related neurological injury, it may award compensation to the claimant, provided that the award is approved by the administrative law judge to whom the claim has been assigned. § 766.305(6), Fla. Stat. If, on the other hand, NICA disputes the claim, as it has in the instant case, the dispute must be resolved by the assigned administrative law judge in accordance with the provisions of Chapter 120, Florida Statutes. §§ 766.304, 766.309, and 766.31, Fla. Stat.

25. In discharging this responsibility, the administrative law judge must make the following determination based upon the available evidence:

> (a) Whether the injury claimed is a birth-related neurological injury. If the claimant has demonstrated, to the satisfaction of the administrative law judge, that the infant has sustained a brain or spinal cord injury caused by oxygen deprivation or mechanical injury and that the infant was thereby rendered permanently and substantially mentally and physically impaired, a

> rebuttable presumption shall arise that the injury is a birth-related neurological injury as defined in s. 766.303(2).
>
> (b) Whether obstetrical services were delivered by a participating physician in the course of labor, delivery, or resuscitation in the immediate post-delivery period in a hospital; or by a certified nurse midwife in a teaching hospital supervised by a participating physician in the course of labor, delivery, or resuscitation in the immediate post-delivery period in a hospital.

§ 766.309(1), Fla. Stat. An award may be sustained only if the administrative law judge concludes that the "infant has sustained a birth-related neurological injury and that obstetrical services were delivered by a participating physician at birth." § 766.31(1), Fla. Stat.

26. Pertinent to this case, "birth-related neurological injury" is defined by Section 766.302(2), to mean:

> injury to the brain or spinal cord of a live infant weighing at least 2,500 grams for a single gestation or, in the case of a multiple gestation, a live infant weighing at least 2,000 grams at birth caused by oxygen deprivation or mechanical injury occurring in the course of labor, delivery, or resuscitation in the immediate postdelivery period in a hospital, which renders the infant permanently and substantially mentally and physically impaired. This definition shall apply to live births only and shall not include disability or death caused by genetic or congenital abnormality.

27. As the proponents of the issue, the burden rested on Petitioners to demonstrate that Alyssa suffered a "birth-related

neurological injury." § 766.309(1)(a), Fla. Stat. See also Balino v. Department of Health and Rehabilitative Services, 348 So. 2d 349, 350 (Fla. 1st DCA 1997)("[T]he burden of proof, apart from statute, is on the party asserting the affirmative issue before an administrative tribunal.").

28. Here, the proof failed to support the conclusion that, more likely than not, Alyssa suffered an "injury to the brain . . . caused by oxygen deprivation or mechanical injury occurring in the course of labor, delivery, or resuscitation . . . which render[ed] . . . [her] permanently and substantially mentally and physically impaired." Consequently, the record developed in this case failed to demonstrate that Alyssa suffered a "birth-related neurological injury," within the meaning of Section 766.302(2), and the claim is not compensable. §§ 766.302(2), 766.309(1), and 766.31(1), Fla. Stat. See also Humana of Florida, Inc. v. McKaughan, 652 So. 2d 852, 859 (Fla. 5th DCA 1995)("[B]ecause the Plan . . . is a statutory substitute for common law rights and liabilities, it should be strictly constructed to include only those subjects clearly embraced within its terms."), approved, Florida Birth-Related Neurological Injury Compensation Association v. McKaughan, 668 So. 2d 974, 979 (Fla. 1996).

29. Where, as here, the administrative law judge determines that ". . . the injury alleged is not a birth-related neurological injury . . . he [is required to] enter an order [to

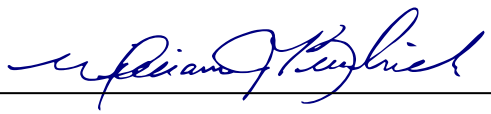
such effect] and . . . cause a copy of such order to be sent immediately to the parties by registered or certified mail." § 766.309(2), Fla. Stat. Such an order constitutes final agency action subject to appellate court review. § 766.311(1), Fla. Stat.

CONCLUSION

Based on the foregoing Findings of Fact and Conclusions of Law, it is

ORDERED that the claim for compensation filed by Jody Workman and Brian Workman, on behalf of and as parents and natural guardians of Alyssa Workman, a deceased minor, is dismissed with prejudice.

DONE AND ORDERED this 29th day of November, 2004, in Tallahassee, Leon County, Florida.



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Filed with the Clerk of the
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this 29th day of November, 2004.

ENDNOTES

1/ All citations are to Florida Statutes (2001) unless otherwise indicated.

2/ Given Respondent's objection, on the basis of hearsay, to Petitioners' Exhibit 1, a report of Postmortem Examination by the Department of Pathology, Shands at the University of Florida, the admissibility of the exhibit was taken under advisement. Upon reflection, the exhibit is received into evidence.

3/ Copies of Respondent's Exhibits 1-5 (medical records related to Alyssa's birth and subsequent development) were provided to Petitioners' post-hearing and, although they were accorded an opportunity to do so, they raised no objection to there receipt into evidence. Consequently, Respondent's Exhibits 1-5 were received into evidence. Respondent's Exhibits 6 and 7 are hearsay, and not otherwise admissible over objection in a civil action. Consequently, they were received into evidence subject to the limitations imposed by Section 120.57(1)(c), Florida Statutes (2003)("Hearsay evidence may be used for the purpose of supplementing or explaining other evidence, but it shall not be sufficient in itself to support a finding unless it would be admissible over objection in civil actions.")

4/ At hearing, Mr. Workman also noted Mrs. Workman's history of low lying placenta, and voiced concern of a placenta previa; however, the medical records reveal any low lying placenta she may have had resolved itself, as evidenced by a report of ultrasound examination on February 20, 2002, and the Obstetric Admitting Record of May 30, 2002, and there was no evidence of a placenta previa encountered during labor or delivery. (Respondent's Exhibit 2).

5/ The Apgar scores assigned to Alyssa are a numerical expression of the condition of a newborn, and reflect the sum points gained on assessment of heart rate, respiratory, effort, muscle tone, reflex irritability, and color, with each category being assigned a score ranging from the lowest score of 0 through a maximum score of 2. As noted, at one minute, Alyssa's Apgar score totaled 2, with heart rate being graded at 2, and respiratory effort, muscle tone, reflex irritability and color being graded at 0. At five minutes, Alyssa's Apgar score totaled 8, with heart rate, respiratory effort, and reflex irritability being graded at 2 each, and muscle tone and color being graded at 1 each.

6/ Petitioners' witnesses, given the circumstances of birth, expressed somewhat different opinions regarding the time that elapsed from Alyssa's birth until resuscitative efforts were started by the team, with the most frequent expressed estimate being the 3 or 4 minutes noted by Mr. Workman. (Transcript, page 15) That estimate was also the figure used by Petitioners in paragraph 2 of their Proposed Final Order, where they state:

> . . . Witnesses testified that Alyssa layed on the warmer several minutes before a code blue was called, and then immediately the resuscitation team came in and began to bag and mask Alyssa . . . .

Paragraph 2 of Petitioners' Proposed Final Order continues, and concludes as follows:

> It then took her three minutes for first gasp and four to sustain respiration. This means Alyssa was not getting any oxygen for three to four minutes.

To the extent Petitioners are suggesting that Alyssa did not get any oxygen for three to four minutes following the onset of resuscitative efforts, their opinion is rejected. At best, the testimony reflects a delay in the onset of spontaneous respiration and not a lack of oxygenation.

7/ "Status epilepticus" is defined by Dorland's Illustrated Medical Dictionary, Twenty-eighth Edition (1994), hereinafter referred to as Dorland's Dictionary, as

> 1. a continuous series of generalized tonic-clonic seizures without return to consciousness, a life-threatening emergency. Called also *convulsive s. epilepticus*.
> 2. any prolonged series of similar seizures without return to full consciousness between them; the two major types are *convulsive s. epilepticus*, which is life-threatening, and *nonconvulsive s. epilepticus*, which is serious but not usually life-threatening.

8/ The first MRI of the brain was performed on March 23, 2003, and reported, as follows:

> FINDINGS: Clinical Indication: Seizure. This study was performed to evaluate for intracranial abnormality.
>
> * * *
>
> Findings:
>
> The brain density is appropriate for a young child. There is minimal retained interstitial water in the cerebral white matter. Brain formation is normal. Myelination is appropriate for age with evidence of myelination in corticospinal tracts, visual pathways and corpus callosum. There is no evidence for malformation of cortical development or foreign tissue lesion. Ventricular size and sulcal pattern are within normal limits. There is no evidence of mass lesion, hydrocephalus, intra or extra axial fluid collection to account for symptoms, as questioned. The paranasal sinuses and oto mastoid air cells are normally developed and aerated without evidence of acute or chronic mucoperiosteal thickening of intrasinus fluid.
>
> IMPRESSION: Negative enhanced MRI of brain for seizure.

The second MRI of the brain, also read as normal, was performed on March 27, 2003, and reported as follows:

> Examination shows midline structures are normally localized. Ventricular system and sulci are normal. No abnormal areas of increased or decreased signal are identified. There is normal brain morphology. There is normal degree of myelination for age. There is no evidence of diffusion abnormality. Hippocampal regions are normal in appearance.
>
> Examination of the calvarium vault demonstrates soft tissue swelling and probable blood products in the extracalvarial soft tissues. This is localized to the occipital region in the midline. This is a

new finding in comparison to prior study. Of note there is no evidence of subjacent brain injury.

IMPRESSION:

1. Normal MR examination of the brain for age. This remains stable in comparison with study of 4 days prior.

2. A focal area of extracalvarial soft tissue swelling/injury occipital region. This is a new finding.

The third MRI of the brain, read as abnormal, was performed on April 8, 2003, and reported as follows:

FINDINGS:

Clinical Indication: 10 month-old female with right-sided focal seizures. This study was performed to evaluate for a structural abnormality.

Comparison: March 27, 2003

* * *

Findings:

The prior MR had demonstrated a normal ventricle size and sulcal size. The current exam demonstrate dilatation of both consistent with an atrophic process. However, whether this . . . apparent atrophy is . . . related to the state of hydration, effective current medications vs. chronic progressive atrophy is not clear.

IMPRESSION:

The current study was obtained to evaluate for a focal abnormality in the left temporal parietal region. This area had focal EEG findings. The current MR demonstrates no

focal abnormality within this area. There are global apparent atrophic changes as described above.

9/ "Idiopathic" is defined as "of the nature of an idiopathy; self-originated; of unknown causation," and "idiopathy" is defined as "a morbid state of spontaneous origin; and neither sympathetic nor traumatic." Dorland's Dictionary.

10/ "Intractable" is defined as "resistant to cure, relief, or control." Dorland's Dictionary.

11/ Respondent's Exhibit 1 (Staff Notes, August 5, 2003).

12/ As noted in Endnote 3, the affidavits of Doctors Duchowny and Willis are not competent proof to support a conclusion regarding causation.

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NOTICE OF RIGHT TO JUDICIAL REVIEW

A party who is adversely affected by this final order is entitled to judicial review pursuant to Sections 120.68 and 766.311, Florida Statutes. Review proceedings are governed by the Florida Rules of Appellate Procedure. Such proceedings are commenced by filing the original of a notice of appeal with the Agency Clerk of the Division of Administrative Hearings and a copy, accompanied by filing fees prescribed by law, with the appropriate District Court of Appeal. See Section 766.311, Florida Statutes, and Florida Birth-Related Neurological Injury Compensation Association v. Carreras, 598 So. 2d 299 (Fla. 1st DCA 1992). The notice of appeal must be filed within 30 days of rendition of the order to be reviewed.