Analysis of causes that led to Baby Robert's respiratory arrest and death in August of 2000

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Abstract

Brian Herlihy is a 32-year-old, white man accused of and arrested for killing Baby Robert by vigorous shaking in August of 2000. Robert was a 4½-month-old infant who suffered from respiratory arrest while at Brian's apartment on the morning of August 2, 2000. That day, Robert's mother arrived at Brian's apartment shortly after 0900 and asked him to watch the baby for a short time. He had cared for the baby on five occasions in the past for a few hours per day. On August 3, 2000 Brian was arrested based on verbal communications between the treating physicians and the police while the baby was still alive in the hospital. The treating physicians told the police that the baby was suffering from injuries caused by shaking. Baby Robert died August 10, 2000. I evaluated the medical evidence in this case using differential diagnosis. My findings clearly show that baby Robert died as a result of adverse reactions to medications and vaccines that were given to him by the healthcare providers. Brian Herlihy is innocent and should be released from prison. Also, the diagnosis of shaken baby syndrome is a theory that should be re-evaluated and is not supported by science in this case.

Keywords: Shaken Baby Syndrome (SBS), adverse vaccine reactions, vaccine-related death

1. Introduction

Brian Herlihy is a 32-year-old, white man accused of and arrested for killing Baby Robert by vigorous shaking in August of 2000. Robert was a 4½-month-old infant, who suffered from respiratory arrest while at Brian's apartment on the morning of August 2, 2000. That day, Robert's mother arrived at Brian's apartment shortly after 0900 asked him to watch the baby for a short time. He had cared for the baby on five occasions in the past for a few hours per day. On August 3, 2000 Brian was arrested based on verbal communications between the treating physicians and the police while the baby was still alive in the hospital. The treating physicians told the police that the baby was suffering from injuries caused by shaking. Baby Robert died August 10, 2000.

Brian Herlihy's jury trial was held in the Eighth Judicial Circuit in Alachua County, Florida on September 10, 2002. His trial lasted sixteen days (Case No. 01-2000-CF-2753-A). The State claimed that Baby Robert was perfectly fine and that absolutely nothing was wrong with him when his mother brought him to Brian's shortly after 0900 on August 2, 2000. The State asserted that while Baby Robert was alone with Brian, he suffered from violent shaking which ultimately resulted in fatal neurological damage and his death. The State furthermore claimed that Brian punished Baby Robert because the baby was crying and that had annoyed, maddened, and frustrated him.

In addition, the State alleged that Baby Robert was never lethargic or anxious from the time of his birth until the morning of August 2, 2000. Brian entered a plea of not guilty. He stated that he took very good care of the baby and that he never harmed him in any way. However, in September of 2002, Brian was convicted of involuntary manslaughter in the death of Baby Robert and sentenced to 15 years in prison.

Brian Herlihy and his family requested that I evaluate the medical evidence in Baby Robert's case in order to find the factual cause(s) that led to Robert's respiratory arrest and death in August of 2000. I evaluated Robert's case by reviewing the following material: (1) the medical records of the mother during her pregnancy with him, (2) Robert's medical records, autopsy report, adverse reactions to medications and vaccines given to Robert, (3) trial documents and testimonies of expert witnesses, and (4) the medical literature pertinent to this case. I used differential diagnosis to evaluate the contribution of agents relevant to this case and the possible synergistic actions among agents in causing Robert's respiratory arrest, bleeding in the subdural space and retina, pathologic changes in the brain and other tissues, and his death.

I present my review of the mother's medical records during her pregnancy with Robert in Section 2. Section 3 contains a review and analysis of Baby Robert's medical records from birth on March 22, 2000 up until the time of his respiratory arrest on August 2, 2000. I also elaborate upon and explain the adverse reactions to vaccines given to Robert in this section.

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Section 4 illustrates the clinical events that occurred during Robert's eight-day stay in the hospitals following his respiratory arrest, and my analysis of these events.

Furthermore, my review and analysis of the medical examiner's autopsy report are presented in Section 5. In Section 6, I define the pathogenesis of Robert's illnesses and their contributions to his respiratory arrest. I also describe adverse reactions to corticosteroids in infants. My review and analysis of the testimonies given by the State's expert witnesses are presented in Section 7. Section 8 contains my conclusions and recommendations.

Robert's mother was involved in a serious car accident on January 10, 2000 when she was at the 26th week of gestation. Her abdominal region was struck by the steering wheel and she experienced pain in her back, legs, and arms along with severe cramping. She was hospitalized at Alachua General Hospital (AGH) for about one week and released. Then she had premature labor at the 34th week of gestation. She spent eight days in labor and Robert was born four weeks premature with a broken collarbone on March 22, 2000.

Robert's mother was treated with betamethasone (corticosteroid) during the last week of her pregnancy and due to this treatment Robert was exposed to corticosteroid in utero. It seems that Robert's mother developed diabetes as a result of her treatment with corticosteroid because she was treated with the anti-diabetic drug micronase. Micronase is not recommended as a treatment in nursing mothers due to its risk of causing hypoglycemia in infants. However, she breast-fed Robert during her treatment with micronase.

Baby Robert suffered from serious health problems that resulted from his exposure to corticosteroid in utero and after birth. These include: gastrointestinal disturbance and reduction in food intake, polyurea, excessive weight gain, myopathy, neurological problems, brain atrophy, chronic subdural and retinal hemorrhage, vision problems, atrophy of the thymus, diabetes, and sinus and ear infections. These symptoms and lesions have been reported in infants treated with corticosteroids as I describe in Section 6. However, none of the physicians who evaluated this case ever addressed this issue.

Furthermore, Baby Robert was given six vaccines on May 9, 2000 and his vaccination with these six vaccines was repeated on July 19th. Premature babies are usually more susceptible to adverse reaction to vaccines than full term infants. Robert was born four weeks premature. Furthermore, vaccines should not be given to children treated with corticosteroid and other immunosuppressant agents. Robert suffered from severe thymic atrophy as a result of his treatment with corticosteroid and his thymus weight was less than 20% of normal for an infant of his age. Thymus weight is a very sensitive biomarker for exposure to corticosteroid.

The vaccines given to Robert increased his susceptibility to infections. The baby suffered from sinus and ear infections as shown by his cerebral CT scans taken on August 2nd. Also, DTP vaccines have been known to increase children's risk of developing neurological disorders, such as encephalopathy or complicated convulsion(s).

Baby Robert suffered from respiratory arrest on August 2, 2000 between 0920 and 0935 and the events that led to his respiratory arrest can be explained as follows: (1) Baby Robert suffered from a seizure prior to 0935 and his seizure resulted from a neurological problem and brain atrophy caused by his prenatal and postnatal treatment with corticosteroids. In addition, the vaccines received on July 19, 2000 might have also played a role in triggering the seizure. (2) The severe seizure caused the baby to vomit and thereby blocked his airway with fluids, which subsequently led to his respiratory arrest. The baby vomited a significant amount of formula like fluid. In addition, the paramedic used a vacuum to remove about 10 mL of formula fluid from his mouth and nose. The baby had been fed approximately 8 ounces of formula milk within 30 minutes prior to his seizure.

Baby Robert suffered from respiratory arrest for at least 60 minutes and that led to severe anoxia, which caused brain and cardiac damage. In addition, the baby suffered from a chronic subdural bleed and retinal bleed as a result of his treatment with corticosteroid. Corticosteroid given at high doses induces diabetes, hypertension, brain atrophy, and increases capillary fragility and abnormal vascular growth in the retina. Glucocorticoid causes hypertension and cardiovascular disease due to its capacity to promote sodium retention and increase blood pressure.

The cerebral CT scan taken on August 2, 2000 at 1028 showed that Robert had a multi-generation subdural bleed. The fresh bleed was estimated to be 20-25% of the total bleed. The occurrence of the fresh bleed in the subdural space on August 2nd can be explained by the synergistic actions of several factors that include: (1) the presence of previous vascular injury in the subdura which led to re-bleeding; (2) Robert suffered from a severe seizure that led to an increase in the intracranial pressure; (3) Robert had an elevated heart rate and that led to increased blood pressure. Robert's pulse rate was 172 at 0938 on August 2nd; and (4) Robert was given relatively large volumes of fluid by intravenous route and that can lead to an increase in the blood pressure.

The retinal bleed and other retinal vascular changes observed by Dr. Lawrence Levine on August 2nd can be explained by Robert's treatment with corticosteroid and diabetes. These conditions have been known to cause retinopathy and retinal hemorrhage as described in Section 6.

The medical examiner and the State's expert witnesses alleged that Baby Robert's respiratory arrest, neurological damage, and death were caused by violent shaking while he was at Brian Herlihy's apartment prior to 0937 on August 2, 2000. However, none of these physicians reviewed the baby's prenatal and postnatal medical records to learn about his pre-existing health problems, his treatment with corticosteroid, or his adverse reactions to corticosteroid and vaccines.

Review of the medical evidence in this case revealed that some of these physicians were aware that Baby Robert was suffering from chronic health conditions such as a chronic subdural bleed, brain atrophy, and sinus and ear infections. However, they did not make any attempt to investigate the links between the baby's chronic illnesses and his respiratory arrest on the morning of August 2, 2000. The following is a list of medical evidence that verifies that the State's expert witnesses conducted an incomplete medical investigation in this case and that they rushed to judgment in accusing Brian Herlihy. Their

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conclusions that the baby died as a result of shaking were based solely upon a theory and not on medical facts.

(1) The emergency teams, several physicians, and the medical examiner examined the baby on August 2-10 and they did not find any sign of injuries on the baby's head or body that was caused by trauma or abuse.

(2) The four cerebral CT scans taken from August 2-4 indicated that Baby Robert was suffering from a chronic subdural bleed. However, none of the physicians who testified for the State ever investigated the causes of the bleed. Furthermore, the medical examiner did not take a sample from the dura to be examined under the microscope in order to date the bleed. The data described in Section 6 of this report show that prenatal and postnatal treatments of infants with corticosteroid have caused hypertension, hyperatrophic cardiomyopathy, encephalopathy, and an increase in capillary fragility; these conditions can lead to subdural bleeding. Robert had been treated with corticosteroid.

(3) The treating physician, Dr. Dickison and the neuropathologist, Dr. Nelson were aware that Baby Robert suffered from brain atrophy but they did not investigate the cause(s) of the atrophy or the link between the atrophy and the baby's seizure and respiratory arrest that occurred on August 2nd. Dr. Dickison stated, "the baby had a smaller brain than the size of the skull, meaning that there was probably some atrophy or wasting of the surface of the brain or that the brain was not growing as rapidly as it should have been." Dr. Nelson also commented, "Robert's brain was an immature brain and it is inconsistent with a brain of a child of four and a half months of age."

It has been reported that premature infants treated with dexamethasone exhibited a 30% reduction in total cerebral tissue volume when compared to both control term infants and premature infants not treated with dexamethasone. Furthermore, dexamethasone administered postnatally to infants has demonstrated increased risk of neurologic impairment, neurodevelopmental disability, and the rate of cerebral palsy in preterm infants and later in survivors. Baby Robert was treated with high therapeutic doses of corticosteroid as indicated by the severity of his thymic atrophy.

(4) At autopsy, the lesions observed in Robert's brain consisted of edema and cell necrosis, which were caused by severe global anoxia and ischemia and not by trauma. The baby was not breathing well for at least 60 minutes. Brian found the baby was not breathing at 0937 on August 2nd. Additionally, Dr. Dickison found the baby was not breathing well at approximately 1100 because the baby was suffering from a severe seizure and his tongue was very stiff blocking the airways.

(5) Dr. John Hellrung, Baby Robert's pediatrician stated during Brian's trial that the baby was normal. However, his examinations showed that the baby suffered from excessive weight gain, polyurea, muscle weakness in the neck region, neurological and possible vision problems. The baby had poor head and neck control, decreased muscle tone in the shoulders and neck, and tight hip flexors. In addition, the baby's tracking with his eyes was not consistent following an object more than a hundred degrees. These symptoms have been reported in infants treated with corticosteroid.

(6) The medical examiner found that the weight of Robert's thymus was 4 grams, which is about 20% of normal. However, he stated that Robert's thymus was normal. The average thymus weight (g) in a white infant male at Robert's age (4-1/2-month old) is expected to be about 22.5g. The treatment with corticosteroid causes immune depression as measured by the reduction in the size and the functions of the lymphoid tissues. It is clear that the medical examiner overlooked an extremely important biological indicator that showed Baby Robert was suffering from severe adverse reactions to corticosteroid.

(7) Dr. Lawrence Levine examined the baby's eyes and found retinal hemorrhage, white spots in the back of the eye, (which he called "Purtscher's retinopathy"), and a crack in the back of the eye, (which he referred to as a choroidal rupture). He claimed that the above lesions were caused by trauma, but his examination of the eyes and eyelids did not reveal any sign of external injuries caused by trauma.

The findings of several studies in Section 6 show that the treatment of children and adults with corticosteroid caused retinopathy, hypertension, diabetes, and increased capillary fragility. Hypertension and diabetes are also known to cause retinopathy. The baby was treated with high doses of corticosteroid and suffered from diabetes. It is very clear that Dr. Levine overlooked crucial medical evidence that demonstrated the link between the baby's treatment with corticosteroids and the lesions found within the retina.

The extensive medical evidence presented in this report clearly shows that Baby Robert died as a result of adverse reactions to corticosteroid and vaccines. Brian Herlihy is innocent. The evidence also shows that Brian was wrongly convicted and imprisoned as a consequence of sloppy and incomplete medical investigations. I believe that the state of Florida has the responsibility to review the evidence presented in this report. The State should furthermore take immediate action to free him from prison. In addition, Brian should be reimbursed for all incurred legal expenses and he should also be compensated for his pain and suffering.

The objective of the State and physicians should be to focus on determining the factual causes that lead to the illness or death of a child so that they can prevent such problems from happening to other children. Accusing innocent people of abusing and killing children based on a faulty theory that has no medical or scientific evidence to support its claims will not prevent the death of other children by vaccines and adverse reactions to medications. However, it certainly places innocent people in prison and causes great suffering. It also costs taxpayers huge sums of money to pay for unnecessary trials and legal fees.

I spent approximately 280 hours evaluating the medical evidence in this case in order to find the factual causes of injuries and death and to write this detailed report. I have also evaluated three other alleged 'Shaken Baby Syndrome' cases from the US within the last 12-month period involving children who died as a result of adverse reactions to medications and vaccines. In all of these cases, either the parent or caretaker was imprisoned after being falsely accused of killing the baby in their care by shaking.

It is my hope that the state of Florida, the Federal Government, physicians, and our society will take the time to review

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the evidence presented in these cases. The government and American Medical Association have an obligation to act immediately as the theory behind the Shaken Baby Syndrome diagnosis must be re-evaluated. The theory itself is unsupported by science. Differential diagnosis must be used to solve complicated medical problems, as I have used in these cases in order to determine the factual causes of the presenting symptoms, illnesses and death.

2. Review of Mother's medical records during her pregnancy with Robert and after delivery

2.1 Mother's health condition during her pregnancy

Baby Robert's mother is a white female. She was 20-years old at the time of her pregnancy with Robert in July of 1999. She was born on August 26, 1979. During her pregnancy with Robert, she suffered from severe nausea for several months (September 21, 1999 through January 13, 2000) and she was treated with phenergan, (anti-nausea drug). Robert's mother was involved in a serious car accident on January 10, 2000, at the 26th week of gestation. She rear-ended a car that was making a turn in front of her and the front of her car was badly damaged. Her abdominal region was struck by the steering wheel and she experienced pain in her back, legs, and arms along with severe cramping. She was hospitalized at Alachua General Hospital (AGH) for about one week and was released [1,2].

Robert's mother only gained two-and-half-pounds during her entire pregnancy with Robert (Table 1). She reached her highest body weight of 120 pounds at the 34th week of gestation. She lost three pounds between the 34th and 35th week of gestation as a result of problems with her pregnancy, which was the result of her car accident a few weeks earlier (Table 1).

Table 1. Mother's body weight during her pregnancy with Robert

Date (mm/dd/yyyy)	Weeks of Gestation	Weight lbs. (kg)
	Pregravid	115.0 (52.2)
09/13/1999	9	110.5 (50.1)
09/30/1999	11	110.5 (50.1)
10/28/1999	15	107.5 (48.8)
12/27/1999	24	116.0 (52.6)
01/18/2000	27	115.0 (52.2)
01/24/2000	28	117.5 (53.3)
02/07/2000	30	117.5 (53.3)
02/21/2000	32	119.0 (54.0)
03/06/2000	34	120.8 (54.8)
03/14/2000	35	117.5 (53.3)

2.2 Mother's premature labor and her treatment with corticosteroid

I believe that the mother's car accident on January 10th induced premature labor. At 35 weeks of gestation, the mother had a premature contraction and she was treated with btamethasone (corticosteroid) to mature the baby's lungs [3]. Her labor was induced by medication and she was in labor for eight days. Baby Robert was born premature at the 36th week of gestation on March 22, 2000 with a broken collarbone. His Apgar scores were eight at one minute and nine at five minutes [4]. The baby and his mother stayed in the hospital for three days and were released.

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2.3 Mother's post delivery diabetes and her treatment with micronase

Based on blood and urine tests performed during the mother's pregnancy on January 24, 2000 and March 3, 2000 respectively, the mother's medical record indicates that she did not suffer from gestational diabetes during her pregnancy. However, her medical record shows that she was treated with an anti-diabetic drug, micronase (glyburide) after delivery. She was advised to stop taking micronase on June 8, 2000 [1]. It seems that Robert's mother developed diabetes following her treatment with corticosteroid. The baby also developed diabetes as a result of the corticosteroid treatment. Robert's mother took micronase during the period when she was breast-feeding Baby Robert.

Micronase (glyburide) is an oral blood-glucose-lowering drug of the sulfonylurea class. Glyburide appears to lower the blood glucose acutely by stimulating the release of insulin from the pancreas, an effect dependent upon functioning beta cells in the pancreatic islets. Single dose studies with micronase tablets in normal subjects demonstrate significant absorption of glyburide within one hour and it reached a high peak level at about four hours. The blood glucose lowering affects generally persist for 24 hours following a single morning dose of micronase in non-fasting diabetic patients [5:2496].

Some sulfonylurea drugs are excreted in human milk. In nursing infants, the potential for developing hypoglycemia exists, therefore, treatment of nursing mothers with micronase is not recommended during the breast-feeding period [6]. In addition, the safety of micronase in children has not yet been established [5].

3. Review of baby Robert's medical records from his birth on March 22 to August 2, 2000

3.1 Robert's treatment with corticosteroid and his exposure to micronase in milk

Baby Robert was born four weeks premature on March 22, 2000 and his mother was treated with betamethasone (corticosteroid) during the last week of her pregnancy [3]. Betamethasone was given to reduce the baby's risk of developing chronic respiratory disease. Robert's mother was in labor for eight days and the baby was born with a broken collarbone. The baby's birth weight was 5 pounds and 14 ounces. The mother and the baby stayed in the hospital for three days and then were released. The baby was noted to have some difficulty latching onto the mother's breast [1].

Three days following birth, Robert's mother noticed that the baby's lips and the inside of his cheeks were yellow. He suffered from mild jaundice. The baby was breast-fed between March 27 and June 8, 2000 and he was also fed formula milk as a supplement during this period. Robert's mother was taking the anti-diabetic drug, micronase, which is not recommended as a treatment in nursing mothers because of the associated risk of causing hypoglycemia in infants. However, she breast-fed Robert during her treatment with micronase [1]. Robert's mother was also put on the progesterone pill as birth control on May 9, 2000.

3.2 Robert's symptoms induced by his treatment with corticosteroid

My review of Baby Robert's medical records revealed that he suffered from serious health problems that directly resulted from his exposure to corticosteroid in utero and after birth [1, 3]. These health problems are detailed in the following subsections.

3.2.1 Polyurea

Baby Robert had polyurea as a consequence of his treatment with corticosteroid. On April 17, 19, and 26, 2000, his grandmother stated that she was changing wet diapers 8-10 times per day. Robert's grandmother was his principle caretaker during most of his life because his parents were working [7]. It is possible that the baby had diabetes at that time. On August 2, 2000 his blood glucose level was 317 mg/dL and he also suffered from glycosuria [8].

3.2.2 Gastrointestinal and feeding problems

On April 13 and 19, 2000, Baby Robert's grandmother stated that the baby was spitting formula milk frequently and he did not have a bowel movement from April 13th through the 17th. On June 6th she also stated that the baby was feeding poorly. On July 19th the baby was treated with mylocon to relieve his problem with intestinal gas. Treatment of infants with corticosteroid is known to cause gastrointestinal problems as described in Section 6 of this report.

3.2.3 Excessive weight gain

Baby Robert gained excessive weight between April 17th and August 2nd. He gained 10 pounds and 7 ounces (4.73 kg) in 136 days as shown in Table 2. His weight at four months and twelve days was about 3.5 times his birth weight. The approximate weight gain for an infant should be one ounce (28 g) per day and 2 pounds (900 g) per month during the first three months of life and 1.25 pounds (570 g) per month between three to six months of age [9]. The approximate weight gain for Baby Robert should not have been more than 7½ pounds (3.4 kg) during his life.

His abnormal weight gain was very obvious. On April 19th, his pediatrician reported that Robert gained 8 ounces in two days. On May 9, 2000, his body weight indicated that he had gained 23 ounces (652 g) in two weeks, which is twice the normal weight gain for a baby at his age. On May 9th, he was six weeks old. He was average in length, slightly below average weight, and slightly above average in head circumference (75%). However, on July 19, 2002, his height was in the 75th percentile, his weight was in the 80th percentile, and his head size was within the 85th or 90th percentile. He was already larger than the average baby at his age who was not born premature [10, 11].

The baby experienced rapid weight gain in spite of his feeding and gastrointestinal problems described above. His rapid weight gain is one of the signs of corticosteroid toxicity that

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was due to water and salt retention, and disturbance in fat, protein, and carbohydrate metabolism. His weight gain was not due to building muscle mass. Robert's serum creatinine values on August 2nd and 3rd, 2000 were less than 25% of normal values and they indicated that Baby Robert was suffering from a muscle wasting problem [8]. Muscle wasting and rapid weight gain are signs of adverse reactions of treatment with high therapeutic doses of corticosteroid.

Table 2. Robert's growth measurements from birth on March 22 through August 2, 2000

Date mm/dd	Age months	Weight lbs. + oz.	Weight (kg)	Height in.	Head Circumference cm.
03/22	Birth	5 lb +14 oz	(2.66)		
03/27		5 lb + 8 oz	(2.49)		
04/05	0.5	6 lb	(2.72)	20	35.5
04/17		6 lb + 9 oz	(2.98)		
04/19	1.0	7 lb + 1 oz	(3.20)	21.5	
04/26		7 lb +10 oz	(3.46)		
05/09		9 lb + 1 oz	(4.11)	22	39.0
06/06		12 lb	(5.44)		
07/19	4.0	15 lb +12 oz	(7.14)	25.5	43.5
08/02		17 lb + 8 oz	(7.94)	26.4	
08/10					43.1

3.2.4 Muscle weakness

Baby Robert exhibited muscle weakness in the neck region. His doctor's exam on May 9, 2000 revealed that he had poor, floppy neck control. The nurse also noted that the baby had tight hip flexors on June 6th. The baby had decreased muscle tone in the shoulders and neck. He still was not holding his head up in prone position on June 6th and July 19, 2000 [11]. His very low serum creatinine values measured on August 2nd and 3rd confirmed that the baby was suffering from muscle wasting illness.

3.2.5 Vision problems

On May 9, 2000 the pediatrician's nurse noted that his tracking with his eyes was not consistent, following an object more than a hundred degrees [11]. Treatment of infants with high therapeutic doses of corticosteroid has been known to cause retinopathy, encephalopathy, diabetes, hypertension, and increased capillary fragility; all of these problems can cause vision problems. At autopsy, Robert's thymus weight was about 20% of the normal weight for a baby at his age. Thymus weight is a very sensitive biomarker for the effect of corticosteroid. Exposure to high levels of corticosteroid causes thymic atrophy as described below (Section 3.3).

3.3 Vaccines given to Baby Robert and his thymic atrophy

Baby Robert was given six vaccines on May 9, 2000 and his vaccination with these six vaccines was repeated on July 19th as shown in Table 3. The compositions of these vaccines are presented in Table 4. Premature babies are generally more susceptible to adverse vaccine reactions. Robert was born four weeks premature. Furthermore, vaccines should not be given to children treated with corticosteroid and other immunosuppressant agents. Baby Robert exhibited severe thymic atrophy as described below.

At autopsy, Baby Robert showed severe thymic atrophy. His thymus weight was 4 grams, which was about 20% of normal [12]. The average thymus weight in a white infant male at three months and six months of age were found to be 20 and 25 g, respectively [13]. Baby Robert was 4½-months-old and his thymus weight should have been approximately 22.5 g. Treatment with corticosteroid causes immune depression as measured by the reduction in size and function of the lymphoid tissues.

Table 3. Robert's vaccination history

Date (mm/dd/yyyy)	Age (months)	Vaccines given
05/09/2000	1.5	DTaP (Diphtheria & Tetanus Toxoids and acellular Pertussis) OPV/IPV (Oral Polio vaccine) Hib (Haemophilus influenzae B) Hep B (Hepatitis B)
07/19/2000	4.0	DTaP (Diphtheria & Tetanus Toxoids and acellular Pertussis) OPV/IPV (Oral Polio vaccine) Hib (Haemophilus Influenzae B) Hep B (Hepatitis B)

Table 4. Compositions of vaccines administered to Baby Robert at two weeks prior to his respiratory arrest and seizure*

Vaccine	Compostion
DTaP	Each dose (0.5 mL) contains 0.625 mg aluminum; 25 Lf Diphtheria toxoid; 10 Lf tetanus toxoid; 25 µg pertussis toxin; 25 µg filamentous hemagglutinin; 8 µg pertacin; 2.5 mg 2-phenoxyethanol; 4.5 mg sodium chloride; and 0.1 mg formaldehyde.
Hepatitis B	Each dose (0.5 mL) contains 0.25 mg aluminum; 10 :g of hepatitis B antigen; 4.5 mg sodium chloride; 25 µg thimerosal (organic mercury); 0.49 mg disodium phosphate dihydrate; and 0.35 mg sodium dihydrogen phosphate dihydrate.
Haemophilus influenzae (Hib)	Each dose (0.5 mL of 0.4% sodium chloride solution) contains 10 µg of purified Haemophilus capsular polysaccharide.
Oral Polio Virus (OPV)	Each dose (0.5 mL of buffered solution) contains less than 25 µg of each of the antibiotics (streptomycin and neomycin) and attenuated poliovirus.

*Described in the Physicians' Desk Reference [5].

3.4 Adverse reactions to vaccines given to Baby Robert

Serious adverse reactions to the vaccines given to Baby Robert (Tables 3 and 4) requiring medical intervention (such as apnea and cardiac problems) are commonly observed in preterm infants. Baby Robert was born four weeks premature and he was suffering from severe immune depression as indicated by his thymus weight measured on August 10th. Vaccination is

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not recommended in children who have been treated with corticosteroids and other immunosuppressant compounds.

Furthermore, the authors of many well-documented studies concluded that the risk and benefit of vaccination in preterm infants should be evaluated prior to administering the vaccines. They also emphasized that preterm infants who receive vaccines should be monitored. The following are descriptions of several selected studies conducted in the USA and other countries that describe adverse vaccine reactions in preterm infants.

(1) Case histories of 45 preterm babies who were vaccinated with DTP/Hib (diphtheria, tetanus toxoids, and pertussis/Haemophilus influenzae type B conjugate were studied retrospectively [14]. Apparent adverse events were noted in 17 of 45 (37.8%) babies: 9 (20%) had major events, i.e., apnea, bradycardia or oxygen desaturations, and 8 (17.8%) had minor events, i.e., increased oxygen requirements, temperature instability, poor handling and feeding intolerance. Age at vaccination of 70 days or less was significantly associated with increased risk ($p < 0.01$). Of 27 babies vaccinated at 70 days or less, 9 (33.3%) developed major events compared with none when vaccinated over day 70.

The authors concluded that vaccine-related cardiorespiratory events are relatively common in preterm babies. Problems were much more common when the vaccine is administered at or before day 70. Therefore, these babies should be monitored post-vaccination. Baby Robert was given six vaccines at 46 days of age and his vaccination with these vaccines was repeated at four months of age (Table 3). At this time the baby was suffering from severe thymic atrophy.

(2) After the occurrence of apnea (a respiratory pause of 20 seconds) in two preterm infants following immunization with DTP and Hib, Sanchez et al. conducted a prospective surveillance of 97 preterm infants (50 girls, 47 boys) younger than 37 weeks of gestation who were immunized with DTP (94 also received Hib at the same time) to assess the frequency of adverse reactions, and, in particular, the occurrence of apnea. For each infant, data were recorded for a 3-day period before and after receipt of the immunization [15]. Their study showed that apneic episodes occurred in 34 infants (34%) after immunization. Twelve infants (12% of total) experienced a recurrence of apnea, and 11 (11%) had at least a 50% increase in the number of apneic and bradycardiac episodes (heart rate less than 80 beats/min) in the 72 hours following immunization. Some of these infants required new medical interventions for the increased episodes [15].

(3) Botham et al. conducted a prospective study of 98 preterm infants (53 males, 45 females), of gestational age 24-31 weeks who were immunized at approximately 2 months postnatal age with diphtheria-tetanus-whole-cell pertussis vaccine (DTPw). Half the infants also received Haemophilus influenzae type b conjugate vaccine (Hib) simultaneously [16]. All infants were monitored for apnea and bradycardia during the 24-hour pre- and post-immunization periods.

The study showed that only one infant had apnea and/or bradycardia pre-immunization, compared with 17 post-immunization. For 12 infants these events were brief, self-limiting and not associated with desaturations (oxygen saturation < 90%). However, for five infants (30%), these events were associated with oxygen desaturation, and two of these infants required supplemental oxygen. When considering immunization for preterm infants, the benefits of early immunization must be balanced against the risk of apnea and bradycardia [16].

(4) Slack et al. reported that four premature infants developed apnea severe enough to warrant resuscitation after immunization with diphtheria, tetanus, pertussis (DTP), and Haemophilus influenzae B (Hib). One required intubation and ventilation. They also reported that although apnea after immunization are recognized they are not well documented [17].

(5) Botham et al. conducted a prospective study of 97 preterm infants who were immunized with diphtheria-tetanus-pertussis to document respiratory and cardiac events [18]. The mean gestational age at birth was 28.1 weeks (range 24-34) and the mean age at immunization was 80.6 days (range 44-257). They found that nineteen (20%) infants developed apnea or bradycardia within 24 hours of immunization. Two infants who developed concurrent upper respiratory tract infections required additional oxygen, and one of them was treated with oral theophylline.

Adverse reactions of vaccines that were administered to Baby Robert are not limited to preterm infants. They have also been reported in full term infants. Below are brief descriptions of selective studies that describe the incidence of illnesses associated with vaccinations in children. Some of these studies are described in the Physicians' Desk Reference [5].

(1) In the USA, reports submitted to the Vaccine Adverse Event Reporting System (VAERS), concerning infant immunization against pertussis between January 1, 1995 and June 30, 1998 were analyzed. During the study period, there were 285 reports involving death, 971 nonfatal serious reports (defined as events involving initial hospitalization, prolongation of hospitalization, life-threatening illness, or permanent disability), and 4,514 less serious reports after immunization with any pertussis-containing vaccine [19].

(2) Systemic adverse events occurring within 3 days following vaccination of 4,696 Italian infants with DTP at 2, 4, and 6 months of age were recorded. These included fever of more than 100.4ºF (38.0ºC) in 7% of total; irritability in 36.3%; drowsiness in 34.9%; loss of appetite in 16.5%; vomiting in 5.8%; and crying for 1 hour or more in 3.9% [5:3063].

(3) The whole-cell DTP vaccine has been associated with acute encephalopathy [5]. A large case-control study that included children 2 to 35 months of age that suffered from serious neurological problems was conducted in England. Acute neurological disorders, such as encephalopathy or complicated convulsion(s) occurred in children who were more likely to have received DTP vaccine the 7 days preceding onset than their age-matched controls. Among children presumed to be neurologically normal before entering the study, the relative risk (estimated by odds ratio) of a neurological illness occurring within a 7-day period following receipt of DTP dose, compared to

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children not receiving DTP vaccine in the 7-day period before onset of their illness, was 3.3 ($p < 0.001$).
(4) Three hundred sixty-five infants were inoculated with Hib, and some of them developed systemic adverse reactions. The following adverse reactions and their percentages occurred in two-month-old infants during the 48 hours following inoculation: fever > 100.8ºF or 38.2ºC (0.6%); irritability (12.6%); drowsiness (4.9%); diarrhea (5.2%); and vomiting (2.7%) [5:2318].

(5) The database from the 1994 National Health Interview Survey (NHIS) in the USA that included 6,515 children less than six years of age who received the hepatitis B vaccine were analyzed to evaluate the vaccine related adverse reactions. Hepatitis B vaccine was found to be associated with prevalent arthritis, incident of acute ear infections, and incident of pharyngitis/nasopharangitis [19].

The above selected studies clearly show that serious health problems and even death can result from vaccinating infants and children, especially among premature infants and infants suffering from pre-existing conditions. The authors of these studies emphasized that premature infants should be monitored following the administration of vaccines. Furthermore, the Physicians' Desk Reference states that vaccines should not be given to children treated with corticosteroid compounds [5].

Fourteen days prior to Baby Robert's respiratory arrest on August 2nd, he was given six vaccines. At the time the infant was administered these vaccines on July 19th, he was suffering from severe immune depression as indicated by his thymus weight measured on August 10, 2000. The CT scan taken on August 2nd of the head region showed that Robert had bilateral sinus and ear infections. The vaccines given to Robert on July 19th increased his risk of contracting sinus and ear infections and also predisposed him to have the seizure on August 2nd. Baby Robert additionally suffered from brain atrophy as a result of his treatment with corticosteroid [8].

4. Review of baby Robert's Medical Records During His Hospitalization on August 2-10, 2000

4.1 Case history and treatments given by the emergency teams on August 2nd

4.1.1 History given by Brian Herlihy

Robert's mother went to Brian Herlihy's apartment with her 4½-month-old Baby Robert shortly after 0900 on August 2, 2000. She fed the baby four ounces of formula milk and left the apartment at about 0920 leaving the baby with Brian. Brian is a white male and in August of 2000, he was 29 years old. He had cared for Baby Robert in the past on five occasions for a few hours each time.

Brian fed the baby approximately four ounces of formula milk. The mother laid the baby on his back between two pillows on the bed, and left the room. After about five minutes, Brian returned to find the infant on his back at the end of the bed with his nose angled towards the floor. Baby Robert's head was lower than his body and his head was wedged between the mattress and the bars of the footboard [20]. He gently tugged on the infant in order to free his head.

The infant vomited formula milk on the floor near the bed and on the bed covering an area of about 4-6 inches in diameter. The baby was not breathing [21]. Brian left the baby on the bed and called 911 at 0935 asking for help. Brian told the person who took the 911 call that the baby was draining white fluid like formula milk from his mouth and his nose. The baby was also coughing [22]. Brian was instructed by 911 personnel to place the baby on the floor and to begin CPR. Brian then proceeded to perform mouth-to-mouth resuscitation. No chest compressions were administered.

4.1.2 Treatments given by the emergency teams

The Alachua County Fire Rescue teams (EMTs) arrived on the scene at 0937 and found the baby lying on his back on the bedroom floor. Baby Robert was unconscious, unresponsive, and he was not breathing. His color was ashen gray. The baby was throwing up white milky fluid from his mouth. They bagged the baby and provided him with 100% oxygen and his blood oxygen saturation came up from 84% to 100%.

They then placed a cardiac monitor on the baby and it revealed a sinus tachycardia at the rate of 170 beats per minute. The baby had palpable pulses in all distal extremities.

In addition, the EMTs placed a line in the baby's right tibia and gave him a 100 cc of fluid. The baby's body weight was 6.8 kg. Brian was very upset and he stated that the baby had vomited and aspirated. The EMTs did not see any signs of struggle in the bedroom or elsewhere. No bruises or abrasions were noted on the baby's head, trunk, flank, back or extremities [21].

A second emergency team arrived at Brian's apartment at 0943 and also found the baby not breathing [23]. Kenneth A. Johnson, the firefighter and paramedic responsible for the EMTs stated that the baby was pale, white and unresponsive. He removed about 10 cc of red and clear mixed liquid from the baby's nose and mouth using a suction unit. The baby had a pulse rate of 190 beats/minute and sinus tachycardia [24]. Table 5 shows the baby's vital signs during the rescue on August 2nd.

Seven minutes after arrival, the EMTs managed to improve the baby's condition. The baby started to breathe on his own, but it was not sufficient to sustain his life. At 0955 the baby was placed on a backboard and was transported to the hospital. He showed some improvement on the way to the hospital. His skin color became pinkish and his respiratory efforts increased. The baby made the first audible sounds when he was wheeled into the hospital at 0958 [23, 24].

Table 5. Baby Robert's vital signs between 0938 and 0958 on August 2, 2000

Time	Pulse beats/min.	Blood Pressure	Respiration (rate/min.)	Heart Condition
0938	172	80	0	Sinus Tachycardia
0944	190	70	0	Sinus Tachycardia
0955	190	70	5	Sinus Tachycardia
0955	180	70	12	Sinus Tachycardia
0958	160	70	20	Sinus Tachycardia

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4.2 Robert's symptoms and treatments given at the hospital on August 2-10, 2000

4.2.1 Treatment at the emergency room

The baby arrived at the hospital emergency room at 1000 [8]. The baby was moving his arms and his legs and he was crying. The ET-tube was extubated because it was not properly placed. The baby's temperature was 36.2°C. His blood pressure and pulse were 70/40 mm Hg and 160 beats per minute, respectively. The baby's fontanel was very tense and pulsating. Retinal hemorrhage was noted bilaterally. No bruises or abrasions were noted on the baby's head, trunk, flank, back or extremities. Blood analysis showed that his lactic acid level was 6.2 mmol/L.

4.2.2 The results of the CT scans for the head and neck regions taken on August 2-4, 2000

After leaving the emergency room, Baby Robert was taken in for CT scans. The CT scan of the head and neck regions taken at 1029 on August 2, 2000 showed a normal skull with no evidence of fracture. A limited lateral view of the cervical spine revealed that the vertebral bodies and the disc spaces were normal down to C5. Furthermore, the cerebral CT scan taken at 1043 on August 2nd showed significant sinus and mastoid disease involving mainly the left maxillary antrum, ethmoids, and both mastoid and middle ear areas [8].

Intracranially, hygromas were observed especially on the left side. The baby was also found to have multi-generation hemorrhage (fresh and old) and bilateral subdural hemorrhage (the left greater than the right) mostly in the superior frontal parietal area. There was trace amount of subarachnoid hemorrhage at several sites most notably the right posterior parietal. Epidural hemorrhage was likely present [8]. The neuroradiologist estimated the fresh bleed that appeared white (hyperdense) on the film to be 20-25% of the total bleed detected. The old bleed (75-80% of the total) appeared dark (hypodense) on the scan [25].

The ventricles were at the upper limits of normal size and essentially non-displaced. There was no evidence of herniation, parenchymal contusion or bleeding. There was a suggestion of at least a right global retinal hemorrhage in the orbits [8].

A second CT scan of the head region was taken at 1521 on August 2nd in order to evaluate the progression of the subdural bleed and other lesions when compared to the prior CT of the head taken at 1028. There was interval worsening in the appearance of the subdural bleed at the vertex of the skull with a new subdural bleed in the right frontal and right parietal-occipital area [8]. The radioneurologist also observed fresh and old bleeds and estimated the fresh bleed to be 35% of the total bleed present. The CT scan taken in the afternoon showed a little more fresh blood along the flax which is the dura that divides the two hemispheres in half [25].

A third cerebral CT scan was taken on August 3rd at 1046 and was compared with the scans taken on August 2nd. The blood had shifted down over the tentorium and it accumulated in the right parietal lobe of the brain and it looked like it was causing a brain laceration as described by the radiologist [25]. A fourth cerebral CT scan was taken on August 4th and did not show a significant change in the bleed from the August 3rd scan.

4.2.3 Robert's symptoms and treatment given at the PICU on the morning of August 2nd

The baby arrived at the PICU at about 1100 on August 2nd at which time he was having a severe seizure. The treating physician stated that Baby Robert was seizing and he was very stiff. She said, "He was so stiff you could pick him up by a leg and he would stay stiff." When the baby was brought to the PICU, the endotracheal tube was not in place. He was trying to cry. His breathing was not normal and his color was gray and ashen. The treating physician also stated that the biggest problem was that the baby was seizing and his tongue was stiff. Therefore, he was not passing air in and out of his mouth or his nose very effectively [26].

The treating physician performed a very thorough physical examination on Baby Robert looking specifically for signs of trauma all over his body (front, back, head, and genitals) and she did not see any bruise or sign of trauma. His neck and mouth were free from any marks or signs of strangulation. His head was normal and nicely shaped. The fontanel (the soft spot on the top of the head) was slightly rounded, but it was still fairly soft and depressible. The baby did not have a significant increase in intracranial pressure at that time [26]. A pediatric neurologist also examined Baby Robert and saw no evidence of red marks or bruising on his body [4].

Following examination, the treating physician treated Baby Robert with muscle relaxant and anti-seizure medications. He was given vecuronium (muscle relaxant) which paralyzes the muscles for a temporary period of time (about a half-hour). The baby was so stiff and his tongue was so swollen that the physician needed him to relax so that the breathing tube could be placed without traumatizing him. The baby was also treated with pentothal which is an anesthetic and anti-convulsant agent [26].

Furthermore, the pediatric neurologist performed an electroencephalograph (EEG) and found that the amount of electrical activity in the brain was deeply suppressed throughout Robert's brain. The waves themselves were very slow, indicating that the process that led to his brain dysfunction was one that involved the whole brain [4]. The clinical tests also revealed that Baby Robert suffered from cardiac damage, hyperglycemia, glycosurea, lactic acidosis, and bilateral retinal hemorrhage. Furthermore, his serum creatinine and protein values were low. Below are descriptions of these health problems and the clinical data.

4.2.4 Robert's heart problems

The emergency teams monitored the baby's heart at 0943 on August 2nd and found that he had sinus tachycardia with a pulse rate of 172-190 per minute (Table 5). Blood analysis showed that the baby had high levels of troponin I, CKMB, and CK total, which indicated that the baby was suffering from myocardiac damage (Table 6). The EKG exam at 1830 on August 2nd also demonstrated that the baby's heart had suffered from

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ischemic changes and arrhythmia. Some elevation in ST waves was noted indicating acute myocardial injury [8].

After admission to the Pediatric Intensive Care Unit, Baby Robert was started on dopamine 10 mcg/kg/minute and received multiple fluid boluses in addition to the normal saline bolus with improvement in blood pressure. The baby was weaned to 3 mcg/kg/minute of dopamine by August 3rd. The infant remained hemodynamically stable post discontinuation of dopamine until August 8th. However, he suffered from cardiac arrest in relation to his respiratory arrest on August 10th.

Table 6. Indicators of myocardial infarctions in Baby Robert's blood

Date (mm/dd/yyyy)	Time	Troponin I (ng/mL)	CK Total (IU/L)	CK MB (ng/mL)
08/02/2000	1225	4.7 H	172 H	7.7 H
08/03/2000	1516	< 0.4	289 H	4.9 H
Normal Range		0.0-0.04	55-170	< 3.1

4.2.5 Baby Robert's metabolic and hematology values during his hospitalization

The blood analysis performed following Baby Robert's arrival at the emergency room on the morning of August 2nd showed that the baby suffered from hyperglycemia and lactic acidosis. The baby also suffered from glycosurea as shown by a urine analysis performed on August 2nd at 1204. His glucose urine value was > 1000 mg/dL. He was treated with sodium bicarbonate to correct his metabolic acidosis [8].

Furthermore, the baby had low blood potassium levels and was treated with potassium. The serum creatinine values on August 2nd and 3rd were very low. They indicated that Baby Robert was suffering from a muscle-wasting problem. His serum levels of albumin and total protein were also low. The baby's metabolic values are presented in Tables 7 and 8.

Table 7. Baby Robert's serum chemistry values on August 2 and 3, 2000

Date (mm/dd) and Time	Albumin	Protein	Creatinine (mg/dL)	Urea Nitrogen (mg/dL)
08/02				
1225	2.5 L	4.6 L		
2340			0.3 L	7 L
08/03				
0400			0.2 L	7 L
1213			0.2 L	6 L
1913	2.1 L	4.3 L	0.2 L	7 L
Normal Range			0.8-1.5	9-20

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Table 8. Baby Robert's metabolic parameters measured following his respiratory arrest

Date & Time	pH Arterial	Glucose mg/dL	HCO_3 mmol//L	Lactic Acid mmol/L	Potassium mmol/L
08/02/2000					
1012	7.35	317 H	19.1 L	6.2 H	3.7
1136	7.26 L	235 H	22.0	4.4 H	3.8
1243	7.32 L		19.4		
1420	7.37		21.2	2.1 H	
1705	7.38		18.6 L	1.9 H	3.8
2001	7.41	111	19.1	1.8 H	
2340	7.43	109	16.2	1.3	3.4 L
08/03/2000					
0147	7.44		17.7 L		3.3 L
0355	7.40		17.1 L		
0553	7.41		17.5 L		
1026	7.38		22.0		
1613	7.37		23.3		
2150	7.38		23.2		
08/04/2000					
0408	7.39		23.8		
0811	7.47 H		24.1		
08/05/2000					
0051	7.52 H		24.0		
0846	7.46 H		25.1		
1627	7.46 H		26.1		
1935	7.46 H		24.5		
Normal Range	7.35-7.45	75-115	21-28	0.3-1.3	3.5-5.0

Robert's hematology values are presented in Table 9. From the values measured following the baby's admission at Shands Hospital at 1012 on August 2nd, the platelet count was reduced by 50% on August 3rd. These values indicate that the baby suffered from bleeding in tissues following his admission to the hospital. His prothrombin time (10.6 seconds) and partial prothrombin time (25 seconds) measured at 1454 on August 2nd were within normal rages. These values indicate that Robert's bleed observed in the retina and subdura were not caused as a result of vitamin K deficiency or liver problems [8]. Baby Robert received a packed red cell transfusion that improved his hemoglobin and hematocrit levels, which continued to remain stable throughout his hospital stay.

Table 9. Baby Robert's hematology parameters measured following his respiratory arrest

Date & Time	RBC x 10^6/;L	Hemoglobin g/dL	Hematocrit %	Platelets x x 10^3/;L
08/02/2000				
1006	4.00	10.7	32.1	392
1147	3.19	8.7	25.2	390
1706	3.78	10.6	31.0	328
2000	4.11	12.0	33.7	238
2340	4.68	13.4	38.3	217
08/03/2000				
0400	4.49	13.2	37.0	186
1213	4.14	11.8	34.5	180
1913	4.07	11.5	33.7	180
08/04/2000				
0400	3.85	11.3	32.3	177
1133	3.90	11.1	32.6	194
08/05/2000				
0051	4.50	13.4	38.7	173
08/06/2000				
0050	4.81	13.8	40.6	159
08/07/2000				
0400	4.90	14.3	41.9	218
Normal Range	2.7-4.9	9-14	28-42	150-450

4.2.6 Retinal bleed and other lesions observed in Robert's eyes.

The pediatric ophthalmologist at the hospital examined Robert's eyes on August 2, 2000 and found no external injury. The eyelids, front of the eyes, cornea, and lens, were normal. The pupils were reactive which indicated that the intracranial pressure was not extremely elevated. The pediatric ophthalmologist dilated the baby's pupils and looked inside Robert's eyes. He observed a massive amount of blood in both eyes [27].

The pediatric ophthalmologist found blood in the center of the eye (vitreous body), the area in front of the retina and in the retina. The baby had two other retinal lesions, which were documented by photography. There were big white spots in the back of his eye, which are referred to as Purtscher's retinopathy. Those big white spots are due to what is believed to be damaged arteries. There was also a crack in the back of the eye, which is known as a choroidal rupture [27].

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4.2.7 Treatment given to Baby Robert on August 2-10, 2000

Baby Robert was treated with fosphenytoin, versed, and ranitidine at high therapeutic levels to control his severe seizure. The baby was given a loading dose of fosphenytoin and then kept on a maintenance dose of fosphenytoin. The baby was also started on a fentanyl drip. The baby had additional seizures on this dose and was given ativan. On the EEG performed on August 4th, Robert continued to show evidence of clinical and subclinical seizures and was maintained on a versed and a fentanyl drip. Blood analysis on August 4th showed the baby's blood level of phenytoin was very high at 29.1 μg/mL. The normal therapeutic levels are between 10 and 20 μg/mL [8].

The baby was also treated with dopamine, Tylenol®, potassium chloride, and sodium bicarbonate to increase his blood pressure and to treat his hypokalemia and metabolic acidosis. He was also given normal saline and red blood cells to treat dehydration and anemia. Presented in Table 10 is a partial list of medications given to the baby during his stay in the hospital.

On August 6th the baby was taken off fentanyl and the versed drip. Seizure activity resumed after approximately six hours. Throughout the hospital stay the baby's head circumference remained stable at about 45 cm and his anterior fontanel remained full and pulsating. His pupils were equal, round, and reactive to light. Baby Robert had decreased tone in the lower extremities bilaterally, positive doll's eyes, a sluggish corneal reflex, and decreased tone in the upper extremities bilaterally. He did not exhibit any response to pain.

On August 7th the baby was started on phenobarbital and once the phenobarbital was at a therapeutic level, the fosphenytoin was tapered. The neurologist was consulted on the case and felt that the examination was consistent with irreversible brain damage. There was no mass effect or potential herniation. The baby was extubated on August 9th. Initially the baby had labored breathing and he was given doses of dexamethasone and racemic epinephrine with mild improvement. The baby was given additional doses of racemic epinephrine and then started on heliox and showed some improvement.

On the morning of August 10th, the baby was noted to have decreased air movement and decreased oxygen saturations into the 90s. The parents had initiated a DNR order and according to this order the baby was not to be reintubated. The baby's heart rate and blood pressure continued to drop and then proceeded quickly into an agonal rhythm of about 20 beats per minute and then into asystole at 0052. This event lasted no longer than ten minutes. It was felt that the baby suffered from respiratory arrest secondary to cardiac arrest due to his brain injury. An autopsy was performed on August 10, 2000. Section 5 contains a detailed description of the autopsy findings.

Table 10. Partial list of medications given to Baby Robert while at the hospital August 2-9, 2000

Date (mm/dd) & Time	Treatment	Actions
08/02		
1115	Fosphenytoin 150 mg IV	Anti-epileptic (Anti-convulsant)
1140	Normal saline, 30 cc/hr	Treat dehydration
	Fosphenytoin 20 mg IV	Anti-epileptic (Anti-convulsant)
1400	Versed 0.4-0.6 mg	Sedative
	Dopamine (10:g/KG/min.)	Increase blood pressure
1450	Vecuronium	Muscle relaxant
	NS + 20 meq KCl/L	Treat dehydaration & hypokalemia
1630	Fentanyl drip	Analgesic
1658	NS + 20 meq KCl/L	
1800	Tylenol® 100 mg	Analgesic and antipyretic
1815	Ranitidine 10 mg IV	Histamine H_2-receptor antagonist
	Fentanyl IV	Analgesic
1830	80 cc Red Blood Cells IV	Treat anemia
2020	Fosphenytoin 40 mg IV	Anti-epileptic (Anti-convulsant)
	Dopamine	Increase blood pressure
2230	NS + 20 meq KCl/L at 20 cc/hr	Treat dehydration and hypokalemia
08/03		
0015	NS + 20 meq KCl/L	
0025	Sodium bicarbonate	Treat metabolic acidosis
0225	Dopamine	Increase blood pressure
1100	Fosphytoin IV	Anti-epileptic (Anti-convulsant)
1300	Fosphytoin IV	
08/04	Fosphentoin	
08/06	Fentanyl IV	Analgesic
	Versed	Sedative
08/07	Phenobarbital	Anti-convulsant
08/09	Dexamethasone	Anti-inflammatory
	Epinephrine	Increase cardiac output

5. Review of the medical examiner's autopsy findings and pathology reports

Baby Robert died on August 10th at 0052 and the Medical Examiner performed the autopsy at 1500 (Case # ME00-297). His autopsy was limited to gross examination of the body and selected organs [12]. He sent the brain and eyes to two consultants for gross and microscopic examinations. The neuropathologist examined the brain and Dr. Michael D. Bell examined the tissues from the eyes [28, 29].

These physicians concluded that baby Robert's injuries and death were caused by violent shaking. However, the autopsy and pathology findings do not support their conclusions that Baby Robert's injuries resulted from violent shaking or trauma. Below are descriptions of their findings and my analysis of them.

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5.1 External examination of Robert's body did not reveal bruises

The medical examiner/forensic pathologist examined Robert's body and he did not see any evidence of injury or trauma. He stated that no significant injuries were seen on the front or the back of the body [12]. The scalp was free of traumatic injuries including lacerations, contusions and bruises. The oral cavity was free of trauma or obstruction. The underlying calvarium and skull base were intact. The neck organ block was free of trauma or obstruction.

5.2 The subdural hemorrhage indicates that the baby had pre-existing condition

The CT scan of Robert's brain taken on August 2nd at 1029 showed that the subdural space contained multi-generation hemorrhage (fresh and old), which indicates that the baby had a pre-existing condition. The medical examiner/forensic pathologist examined the dural membranes grossly. He observed lightly adherent clots in the subdural spaces over cerebral convexities and a clot attached to the undersurface of the dura overlying the cerebral hemispheres. He concluded that the baby did not have chronic subdural hemorrhage. I believe that the medical examiner/forensic pathologist's conclusion with regard to the time of the bleed is not scientifically valid because he failed to microscopically examine the dura to date the bleed.

5.3 The brain lesions indicate that the baby suffered from anoxia and ischemia

The gross and the microscopic examinations of Robert's brain did not indicate that Baby Robert died as a result of trauma but, instead they showed that the baby had pre-existing chronic brain atrophy and the brain suffered from severe anoxia and ischemia. However, the medical examiner and other physicians who evaluated this case all overlooked these facts.

The neuropathologist examined Robert's brain microscopically and found that the brain was an immature brain and inconsistent with that of a child of four and a half months [28, 30]. The treating physician examined the CT scans of Robert's brain and also determined that he suffered from brain atrophy. She stated that Baby Robert had a smaller brain than normal. That means that there was probably some atrophy or wasting of the surface of the brain or that the brain was not growing as rapidly as it should have been [31, page 754].

The other lesions observed in the brain were edema and cell necrosis, which were caused by severe anoxia and ischemia of the brain. The medical examiner/forensic pathologist examined Robert's brain grossly and found evidence of edema. The brain substance was quite soft. The gyri were flattened and sulci were narrowed. The brain weight was 826 g, which was about 150% of normal (normal weight about 600 g) [13]. The medical examiner did not see evidence of trauma [12].

The neuropathologist examined eight glass microscopic slides of the brain that were stained with hemotoxylin and eosin (H & E). He found that the lesions consisted of cell necrosis that resulted from global anoxia and ischemia of the brain. He also stated that Baby Robert's brain was immature and that was due to improper development. The neuropathologist never stated that these lesions were caused by trauma. He stated that this was an immature human brain with subarachnoid hemorrhage and hematoma, global anoxic-ischemic encephalopathy, and generalized cerebral edema [28].

The slides that were examined by the neuropathologist included tissue sections from left and right frontal gyri, bilateral hippocampal formations, midbrain, basal ganglia, and cerebellum. He found that the neurons displayed the typical anoxic-ischemic histologic changes. He observed laminar necrosis in the cerebral cortex and he said that these anoxic-ischemic microscopic changes were most prominent in the grayl surfaces, but were also present at the depths of the sulci.

Furthermore, the neuropathologist stated that both the Sommer's sector and endplate of the hippocampal formation displayed bilateral symmetry, which consisted of acute neuronal necrosis consisting of cytoplasmic hyperesosinophilia, karyorrhexis and nuclear pyknosis. There was no inflammation other than foamy macrophages. Except for neuronal swelling and parenchymal and leptomeningeal vascular congestion, the midbrain was histologically within normal limits. He also observed acute neuronal necrosis in the basal ganglia.

The cerebellum was remarkable for relative preservation of the external granular neuronal lamina. The internal granular cell layer was also prominent. There was widespread neuronal necrosis with cytoplasmic hypereosinophilia, karyorrhexis and nuclear pyknosis. There was no inflammation, other than foamy macrophages.

The neuropathologist's findings described above clearly indicate that Baby Robert suffered from brain atrophy, severe anoxia and ischemia and not from physical trauma. The neuropathologist stated that these gross and microscopic findings are consistent with a multi-day survival after hospitalization. However, the neuropathologist found a very small cerebral cortical lesion microscopically (8 x 8 x 9 millimeters), that he believed was a contusion caused by trauma [30]. He observed diffuse endothelial cell swelling with extravasated erythrocytes, vacuolation of the neutrophil and foamy macrophages throughout.

It is my opinion that the neuropathologist's claim that Robert's brain had a contusion resulting from trauma is not supported by medical facts based on the following reasons:

(1) Baby Robert was examined by many physicians and no evidence of injuries caused by trauma was observed in the head or neck regions. These facts were confirmed by the medical examiner on August 10th.
(2) No laceration or contusion of Robert's brain was observed on the cerebral CT scans taken on 1029 and 1521 on August 2nd. However, a minor cortical lesion in the brain was observed in the third cerebral CT scan taken on August 3rd at 1046, which resulted from the accumulation of blood in the brain. The neuroradiologist read the CT scan and stated that the blood had shifted down over the tentorium and that it had accumulated in the right parietal lobe of the brain. He referred to this lesion as a brain laceration [25].
(3) Anoxia and ischemia can cause diffuse endothelial swelling and extravasation of red blood cells and Robert's brain suffered from severe anoxia and ischemia.

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5.4 Bleeding in the eyes

The medical examiner harvested Robert's eyes at autopsy and sent them to Dr. Michael D. Bell for examination. Dr. Bell examined Robert's eyes grossly and microscopically and he observed retinal hemorrhages [29]. Multiple diffuse retinal hemorrhages were observed in the right eye, while the retina of left eye had only multiple focal retinal hemorrhages. The retinal hemorrhages were positive for iron stain in both eyes, which means that the bleed was older than 24 hours. Baby Robert suffered from diabetes and adverse reactions to treatment with corticosteroid. Both of these conditions cause vascular abnormalities and severe bleeding in the retina as described in Section 6 of this report.

5.5 Thymus atrophy

The medical examiner stated that Robert's thymus weight was 4 g and its external and sectioned surfaces were unremarkable [12]. Baby Robert was 4½-months old at the time of autopsy and his thymus weight should have been about 22.5 g. The average thymus weight in a white, infant male at three months and six months of age was found to be 20 and 25 g, respectively [13]. These data indicate that Robert's thymus weight was approximately 20% of normal size.

The baby was treated with corticosteroid because he was born four-weeks premature. The treatment with corticosteroid causes immune depression as measured by the reduction in size and the function of the lymphoid tissues. The medical examiner did not review the baby's medical record; therefore he overlooked the facts that both the baby and his mother developed diabetes as a result of their treatment with corticosteroid.

Furthermore, the use of corticosteroid at high therapeutic doses causes atrophy in the adrenal gland. The medical examiner stated that the combined weight of the right and left adrenal glands was 4 grams, but he did not evaluate their structure microscopically to check for abnormalities. He also did not take tissue samples from other endocrine glands to be examined microscopically and to check for abnormalities. A study conducted in the United States of America showed that dexamethasone therapy in newborns for a period of a week or longer was associated with suppression of the hypothalamic-pituitary-adrenal axis (HPAA) in a substantial number of premature infants [32].

5.6 Robert's spleen weight appeared less than normal

Robert's spleen weight at autopsy was 16 g [12]. The average spleen weights for infants at 3 and 6 months of age were found to be 16.3 g and 22 g respectively [13]. Based on these data, the expected weight of the spleen in a 4½-month-old white baby is 19 g. Furthermore, it is possible that the actual spleen weight in Robert's case was significantly less than 16 g because his organs were congested with blood.

Robert's liver weight was 324 g and the expected liver weight for a 4½-month-old baby is about 220 g [12, 13]. His lungs were also heavier than normal (160 g) because of congestion. The normal lungs weight for a baby at Robert's age is about 94 g. The liver and lungs weight were increased by 45% and 70%, respectively because of congestion.

These data indicate that the actual spleen weight in Robert's case may have been much less than 16 g; Robert had severe atrophy of the spleen as it happened with the thymus. The medical examiner did not examine the spleen microscopically and gross examination alone is not adequate to detect microscopic abnormalities.

5.7 Inadequate examination of the heart

The clinical data indicate that Robert suffered from heart problems (Tables 5 and 6). However, the Medical Examiner did not take tissue samples to microscopically evaluate the structure of Robert's heart. He examined the heart grossly and declared that Robert's heart was normal. I believe that the Medical Examiner's conclusion is not valid based on the following facts: (1) The emergency teams monitored the baby's heart at 0943 and found that he had sinus tachycardia with a pulse rate of 172-190 per minute (Table 5); (2) blood analysis showed that he had high levels of troponin I, CKMB, and CK total which indicated that the baby was suffering from myocardiac damage (Table 6); (3) the EKG exam taken at 1830 on August 2nd showed the baby's heart suffered from ischemic changes and arrhythmia; (4) some elevation in ST waves was noted indicating acute myocardial injury.

6. Analysis of clinical events and causes that led to Baby Robert's respiratory arrest and death

The clinical data described in Sections 2 through 5 of this report show that Baby Robert suffered from several chronic illnesses and some of these illnesses led to his respiratory arrest and death in August of 2000. These illnesses include: brain atrophy and other neurological problems, subdural bleed, retinal bleed and other abnormalities, diabetes, heart problems, sinus and ear infections, muscle weakness, atrophy of the lymphoid tissue, and excessive weight gain.

Robert's illnesses resulted from his exposure to high therapeutic doses of corticosteroid (betamethasone). His thymic atrophy was severe and it indicates that the baby was treated with high doses of corticosteroid. His thymus weight at autopsy was about 20% of normal. The thymus is a very sensitive biomarker for the effect of corticosteroid.

Adverse reactions to corticosteroid have been reported in children. These include: (1) fluid and electrolyte disturbance (sodium retention, fluid retention, potassium loss, and hypokalemic alkalosis); (2) increased capillary fragility; (3) musculoskeletal problems (muscle weakness, steroid myopathy, and loss of muscle mass); (4) cardiovascular problems (congestive heart failure in susceptible patients, cardiac hypertrophy, and hypertension); (5) gastrointestinal problems (peptic ulcer with possible subsequent perforation and hemorrhage, pancreatitis, abdominal distention, ulcerative esophagitis); (6) neurological problems (convulsions and increased intracranial pressure, brain atrophy, and demylination); (7) manifestations of latent diabetes mellitus and glycosuria; (8) ophthalmic problems (posterior subcapsular cataracts, increased intraocular pressure

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and glaucoma, exophthalmos, and retinopathy); (9) metabolic problems (negative nitrogen balance due to protein catabolism); (10) abnormal weight gain; (11) decreased functions of the immune systems and increased susceptibility to infections [33, 5].

Baby Robert was also exposed to micronase (anti-diabetic drug) via milk and this medication should not be given to a nursing mother. It causes hypoglycemia in breast-fed infants. Robert's mother did not suffer from diabetes during her pregnancy. However, her medical record indicates that she was treated with micronase after giving birth and that she stopped taking this medication on June 8th. This indicates that her diabetes was caused by her treatment with corticosteroid during her last week of pregnancy. The baby also suffered from glycosuria and diabetes as a result of his exposure to corticosteroid [8].

Furthermore, the baby was given vaccines (Tables 3 and 4) when he was suffering from immune depression as a result of his treatment with corticosteroid which increased his risk of developing ear and sinus infections. Glucocorticoids cause profound and varied metabolic effects. They also modify the body's immune response to diverse stimuli. Immunization procedures should not be undertaken in patients who are on corticosteroids, especially in high doses, because of the possible hazards of neurological complications and lack of antibody response [5:2824]. The cerebral CT scan taken at 1043 on August 2nd showed that Baby Robert suffered from significant sinus and mastoid disease [8].

6.1 Events that led to Robert's respiratory arrest on August 2, 2000

Baby Robert suffered from respiratory arrest on August 2nd, 2000 between 0920 and 0935 and the medical evidence indicates that the following events led to his respiratory arrest:

(1) Baby Robert suffered from a seizure prior to 0935 and his seizure resulted from neurological problems and brain atrophy caused by his treatment with corticosteroids.

Dr. Anne Elizabeth Dickison who treated Baby Robert on the morning of August 2nd stated that the baby was seizing and he was very stiff. She said, "He was so stiff you could pick him up by a leg and he would stay stiff." His tongue was so stiff that the physician needed to cause him to relax in order to correctly place the breathing tube without traumatizing him. Dr. Dickison treated Baby Robert with muscle relaxant and anti-seizure medications [26].

(2) The severe seizure caused Baby Robert to vomit and led to the blockage of his airways with fluids which prompted his respiratory arrest. The baby vomited on the bed and the floor near the bed. Brian told the person who took the 911 call that the baby was draining white fluid-like formula milk from his mouth and nose. The baby was also coughing [22:445]. The emergency team who arrived on the scene to treat the baby had to clear fluid from his mouth and nose in order to get oxygen into him. The paramedic used a vacuum to suck about 10 mL of formula from his airway. The baby had been fed about 8 ounces of formula milk within 30 minutes prior to his seizure.

(3) The movement of the baby from his place on the bed to the end of the bed can be explained by his seizure and his struggle to breath. Robert's mother stated that her baby was able to roll halfway [11].

6.2 The biomechanisms of Robert's injuries

Baby Robert suffered from respiratory arrest for a significant time and that led to brain anoxia and cardiac damage. In addition, the baby suffered from a chronic subdural bleed and retinal bleed as a result of his treatment with corticosteroid. Corticosteroid induces diabetes, hypertension, brain atrophy, and it increases capillary fragility and abnormal vascular growth in the retina. Glucocorticoid causes hypertension and cardiovascular disease due to its capacity to promote sodium retention and increased blood pressure [33].

Baby Robert suffered from polyurea as early as April 19, 2000 when he was four weeks old. On April 17, 19, and 26, 2000 his grandmother stated that she was changing wet diapers 8-10 times per day. This indicates that the baby was suffering from polyurea. The urine and blood analyses performed on August 2nd showed that the baby was suffering from hyperglycemia and glycosurea. His blood glucose level was 317 mg/dl and he had glycosurea (>1000 mg/dL) [8]. These conditions usually cause polyurea.

The cerebral CT scan taken August 2nd at 1028 showed that Robert had a multi-generation subdural bleed. The neuroradiologist estimated the fresh bleed to be 20-25% of the total bleed observed [25]. The fresh bleed increased to 35% of the total at 1521 on August 2nd. The cerebral CT sans taken on August 3rd and 4th showed no change in the status of the bleed observed in the last CT scan taken August 2nd.

The occurrence of the fresh bleed in the subdural space observed August 2nd can be explained by the synergistic actions of several factors that include the following: (1) the presence of previous vascular injury in the subdura which led to re-bleeding; (2) severe seizure that led to an increase in the intracranial pressure; (3) elevation in heart rate that led to an increase in the blood pressure; and (4) the baby's pulse rate was 172 at 0938 on August 2nd and injection of relatively large volumes of therapeutic fluid administered intravenously can cause an increase in the blood pressure.

The retinal bleed observed by the Pediatric Ophthalmologist on August 2nd can be explained by Robert's treatment with corticosteroid and diabetes. These conditions have been known to cause retinopathy and retinal bleeds. In addition to the retinal bleed, the Pediatric Ophthalmologist observed blood in the vitreous body, big white spots in the back of the eye, (which he referred to as Purtscher's retinopathy), and a crack in the back of the eye, (which he called a choroidal rupture) [27]. These lesions have also been reported in patients suffering from diabetes and/or treatment with high therapeutic doses of corticosteroid [5, 33].

Robert's symptoms and lesions described in this report resemble those that have presented in other infants who have also been treated with corticosteroids. Adverse reactions to corticosteroids in infants have been widely reported in the medical lite-

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rature. However, none of the physicians or the medical examiner that evaluated this case paid any attention to these facts. Baby Robert was born four weeks premature and he and his mother were treated with corticosteroid. The following are descriptions of selected studies that explain the adverse reactions to corticosteroids in preterm infants and older children.

6.3 Corticosteroids cause neurological problems in children

6.3.1 Treatment with corticosteroids and neurological problems

Corticosteroid compounds are given to pregnant women who are at risk of delivering premature infants and to preterm infants in order to help in maturation of the infant's lungs. Robert's mother was treated with corticosteroid during her last week of pregnancy. Baby Robert suffered from severe thymic atrophy and other adverse reactions to corticosteroid. His symptoms indicate that he was treated with high therapeutic doses of corticosteroid. Prenatal and postnatal treatments with therapeutic doses of corticosteroid compounds have shown to cause early and delayed neurological problems in infants as described below.

Eighteen premature (23 to 31 weeks) infants (7 treated with dexamethasone and 11 not treated) were studied at term, i.e., 38 to 41 post-conceptional weeks. Advanced quantitative volumetric 3-dimensional magnetic resonance imaging (MRI) technique was used to quantify cerebral tissue volumes in these infants. Fourteen healthy term infants were also studied for comparison. Cerebral cortical gray matter volume in premature infants treated with dexamethasone was reduced by 35% when compared with gray matter volume in premature infants not treated with dexamethasone (mean $\pm$ standard deviation, 130.3 $\pm$ 54.0 vs. 200.6 $\pm$ 35.1 mL, respectively). Premature infants treated with dexamethasone exhibited a reduction (30%) in total cerebral tissue volume compared with total cerebral tissue volume in both the control term infants and premature infants not treated with dexamethasone (312.7 $\pm$ 43.7 vs. 448.2 $\pm$ 50.2 and 471.6 $\pm$ 36.4 mL respectively) [34].

Furthermore, dexamethasone given postnatally to treat or prevent bronchopulmonary dysplasia (BPD) has also shown to increase the risk of neurologic impairment, neurodevelopmental disability, and the rate of cerebral palsy in preterm infants and later in survivors [35-37]. Nineteen randomized controlled trials of postnatal corticosteroid treatment within 96 hours of birth in high-risk preterm infants were reviewed. In the two trials which have reported late outcomes, several adverse neurological effects were found at follow-up examinations of survivors treated with early steroids. These include abnormal neurological examination, cerebral palsy, and developmental delay [38, 39].

In addition, twenty-one randomized controlled trials of postnatal corticosteroid treatment within 96 hours of birth (early) enrolling a total of 3072 preterm infants were reviewed. In the nine trials that have reported late outcomes, several adverse neurological effects were found at follow-up examinations of survivors treated with early steroids. These include cerebral palsy and other abnormal neurological findings [40]. Also, randomized controlled trials of postnatal corticosteroid treatment initiated at > 3 weeks of age in preterm infants with CLD were reviewed. There were increases in long-term neurologic sequelae including abnormal neurologic examination and cerebral palsy [41].

Yeh et al. evaluated a total of 133 preterm infants (70 in the control group, 63 in the dexamethasone-treated group) who survived the initial study period and lived to 2 years of age. For infants in the treatment group, dexamethasone was started at a mean age of 8.1 hours and given 0.25 mg/kg every 12 hours for one week and then tapered off gradually over a 3-week period. The dexamethasone-treated group had a significantly higher incidence of neuromotor dysfunction (25/63 vs. 12/70) than the control group. Significant handicap was seen in 22 children (31.4%) in the control group and 26 (41.2%) in the dexamethasone-treated group [42].

Furthermore, a study compared a three-day course of dexamethasone (n =132) with a saline placebo (n = 116) administered from before 12 hours of age in preterm infants. Dexamethasone treatment was associated with increased incidence of hypertension, hyperglycemia, and gastrointestinal hemorrhage and no reduction in either the incidence or severity of chronic lung disease or mortality. A total of 195 infants survived to discharge and five died later [43].

Follow up data were obtained on 159 of 190 survivors at a mean age of 18 months. Dexamethasone treated children had a significantly higher incidence of cerebral palsy than those receiving placebo 49% vs. 15%, respectively. The most common form of cerebral palsy was spastic diplegia and the incidence rates were 28% vs. 6% in dexamethasone and placebo treated infants, respectively. Developmental delay was also significantly more common in the dexamethasone treated group (55%) than in the placebo treated group (29%) [43].

Also, a study evaluated the long-term adverse effects of corticosteroid given to infants on the nervous system. Of the 120 children who received corticosteroids, 98 (81.7%) survived to 5 years of age, compared with 200 (88.5%) of the 226 children who did not receive corticosteroids. At 5 years of age, survivors treated with corticosteroids postnatally had significantly higher rates of cerebral palsy (23%) compared with children not treated (4%).

In addition, the rate of sensorineural disabilities was significantly higher in children treated with postnatal corticosteroids, and the association between adverse sensorineural outcome and postnatal corticosteroids remained the same after adjustments for potentially confounding variables. In a separate case-control analysis of 60 children in each group, the rate of cerebral palsy remained significantly elevated (corticosteroids 22%, no corticosteroids 5%) [44].

Bos et al. studied 37 preterm infants with Prechtl's method for the qualitative assessment of general movements before, during and after dexamethasone therapy and found that the quality of general movements was impaired in 9 of 13 initially normal infants. The quality of fidgety movements at 3 months was abnormal in the majority of the infants and strongly correlated with neurological abnormalities at 2 years of age [45].

6.3.2 The release of high levels of endogenous corticosteroid also causes neurological problems

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The adrenocorticotrophic hormone (ACTH) is released from the pituitary gland to stimulate the cortex of the adrenal glands. This causes an increase in the synthesis and the release of cortisol. It has been shown that the treatment of children with ACTH also caused neurological problems as a result of the release of endogenous cortisol. For example, eight children with different petit mal epilepsies were systematically treated with ACTH and dexamethasone. Cranial computed tomography (CCT) examinations were performed before, during and after treatment. Severe cerebral changes were observed in all children. Enlargement of ventricles and subarachnoid space was developed during the initial phase of treatment with Depot-ACTH. Similar changes, but to a lesser degree were observed during the phase of dexamethasone therapy thereafter [46].

Furthermore, Riikonen and Donner evaluated 162 children with infantile spasms who were treated with ACTH. In a large proportion (37%) of the children, the treatment caused pronounced side effects, and the mortality rate was 4.9%. At autopsy, fresh intracerebral hemorrhages were observed [47].

6.3.3 Abnormal changes in the nervous system caused by corticosteroid can also be reproduced in experimental animals

Experimental studies in animals have shown that multiple courses of antenatal corticosteroid cause deleterious effects on lung growth, brain myelination, hypothalamic-pituitary-adrenal function, and retina development [48]. Animal studies have also shown that maternal corticosteroid delays myelination and reduces the growth of all fetal brain areas, particularly the hippocampus [49].

Furthermore, Aghajafari et al. evaluated the results of eighteen studies dealing with the adverse effects of antenatal corticosteroids on the nervous system and fetal growth in experimental animals. Seven studies investigated the effects of repeated doses of antenatal corticosteroids on the brain and nervous system function or growth. All seven studies found adverse effects with repeated doses of antenatal corticosteroids. Eleven studies looked at the effect of repeated doses of antenatal corticosteroids on fetal growth. Nine studies found evidence of fetal growth restriction with repeated doses of antenatal corticosteroids [50].

In addition, pregnant ewes were given saline or betamethasone (0.5 mg/kg) at 104, 111, 118, and 124 days gestation, stages equivalent to the third trimester in humans. Lambs were delivered at 145 days (term), perfused and the corpus callosum examined with light and electron microscopically. Total axon numbers were unaffected ($p > 0.05$). However, myelination was significantly delayed. Myelinated axons were 5.7% in the experimental group and 9.2% in controls ($p < 0.05$). Myelinated axon diameter and myelin sheath thickness were also reduced (0.68 vs. 0.94 and 0.11 vs. 0.14 microm, $p < 0.05$) [51].

Furthermore, Quinlivan examined the effect of single or repeated injections of maternally administered corticosteroids on fetal growth in sheep. Forty-six date-mated singleton gestation ewes were allocated at random to one of three groups: a single or repeated injection of betamethasone, or a control group which received saline. On days 125 (preterm) or 145 (term) caesarean section delivery was performed. After the lambs were killed, measures of size and weight were recorded. Significant betamethasone dose dependent reduction in body and organ weights and biometry were found at preterm and term gestational ages ($p < 0.05$). There was little catch up growth in those in whom delivery was delayed until term. Thymus, spleen and liver were particularly targeted [52].

6.4 Corticosteroid causes cardiomyopathy in infants

Yunis et al. reported three cases of newborns whose mothers were treated with betamethasone prenatally at different doses and duration of time. They developed various degrees of hypertrophic cardiomyopathy (HCM), which was diagnosed by echocardiography. They suggested that repeated antenatal maternal steroid intake might cause changes of HCM in the newborn. These changes appear to be dose- and duration-related [53]. In addition, Miranda-Mallea et al. reported two cases of hypertrophic cardiomyopathy in two preterm newborns secondary to dexamethasone treatment [54].

Furthermore, Israel et al. conducted a retrospective review of one preterm infant who received a 26-day course, and 13 preterm infants who received at least one 42-day course of dexamethasone, and who had serial echocardiographic data available. Left ventricular hypertrophy was noted in 8 (57%) of 14 infants; hypertrophy usually was noted near the end of the treatment course. Five of these eight affected infants died; the hypertrophic cardiomyopathy was considered to have contributed to mortality in three of these five infants. They speculate that prolonged dexamethasone treatment for chronic lung disease (CLD) is associated with hypertrophic cardiomyopathy in a significant portion of preterm infants [55].

Also, twenty preterm infants were studied serially with Doppler echocardiography to document changes in myocardial thickness associated with dexamethasone treatment for chronic lung disease. Ventricular septa and left ventricular posterior wall thickness was increased in all 11 infants in whom it was measured. The median increase was 0.9 and 0.8 mm, respectively. In most infants this increase was small, less than 1 mm, however two infants developed marked septal hypertrophy with Doppler evidence of left ventricular outflow tract obstruction. In addition, myocardial hypertrophy occurs in most infants and in some of them it was severe [56].

Furthermore, eighteen children treated for infantile spasms with high-dose of adenocorticotropin were evaluated for adverse effects of corticosteroid on the heart. Abnormal ventricular hypertrophy occurs in the majority of these patients. Many of these patients developed hypertrophic cardiomyopathy with dramatic asymmetric septal hypertrophy. Abnormal cardiac hypertrophy was seen in 13 (72%) of 18 patients. Five of 18 patients developed hypertrophic cardiomyopathy with asymmetric septal hypertrophy and concentric left ventricular hypertrophy was seen in eight patients [57].

6.5 Corticosteroid causes hypertension, diabetes, and other systemic problems

Dexamethasone treatment of preterm infants started within the first 4 days of life has been shown to cause metabolic disturbances, cardiac hypertrophy, reduced growth and gastroin-

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testinal perforation. Nineteen randomized controlled trials of postnatal corticosteroid treatment within 96 hours of birth in high-risk preterm infants were reviewed. Gastrointestinal bleeding and intestinal perforation were important adverse effects and the risks of hyperglycemia and hypertension were also increased [38, 39].

In addition, a multi-center, randomized, placebo controlled, blinded study was carried out in 18 neonatal intensive care units in Israel to study the effect of early postnatal dexamethasone (days 1-3). The study consisted of 248 infants (dexamethasone n = 132; placebo n = 116). Gastrointestinal hemorrhage, hypertension, and hyperglycemia were more common in dexamethasone treated infants [59]. Furthermore, 21 randomized controlled trials of postnatal corticosteroid treatment within 96 hours of birth (early) enrolling a total of 3072 preterm infants were reviewed. Gastrointestinal bleeding and intestinal perforation were noted adverse effects and the risks of hyperglycemia and hypertension also increased [40].

The magnitude and duration of the effect of dexamethasone on systolic blood pressure has also been examined in 13 preterm infants (median gestational age 25 weeks). All had chronic lung disease (CLD). To exclude any effect of CLD on blood pressure each infant acted as his or her own control. Systolic blood pressure increased in all infants ($p < 0.01$) and remained elevated for at least 48 hours following cessation of therapy. The median maximum increase in blood pressure was 24 mmHg (range 13-49 mmHg) and occurred on day 4 (median, range 2-10) of treatment. One infant also developed hypertensive encephalopathy. These results demonstrate the need to monitor infants with CLD throughout steroid therapy and preferably for some days after treatment has ceased [60].

Furthermore, randomized controlled trials of postnatal corticosteroid treatment from 7-14 days of birth in high-risk preterm infants were reviewed. Adverse effects included: hypertension, hyperglycemia, gastrointestinal bleeding, hypertrophic cardiomyopathy and infection [61]. In addition, nine trials enrolling a total of 562 infants were reviewed to determine the adverse reactions of late (usually > 3 weeks) postnatal corticosteroid treatment vs. control (placebo or nothing). Short-term adverse affects included glycosuria and hypertension. There was also an increase in severe retinopathy of prematurity [62].

Also, randomized controlled trials of postnatal corticosteroid treatment initiated at > 3 weeks of age in preterm infants with CLD were evaluated. Short-term adverse affects included hyperglycemia, glycosuria and hypertension. There was also an increase in severe retinopathy of prematurity [41].

The influence of dexamethasone (0.25 mg/kg I.V. twice daily) on diuresis in preterm infants was also studied in 15 preterm infants at risk for chronic lung disease. Urine output, blood glucose, serum urea, serum creatinine, serum sodium and serum potassium, as well as systolic, diastolic and mean arterial pressure were measured on the day before, and on 4 consecutive days after starting treatment with dexamethasone (0.25 mg/kg I.V. twice daily). The authors found an increase of diuresis of 30 mL/kg per day, 48-96 hours after starting dexamethasone treatment. This coincided with a gradual but significant increase of serum urea levels and arterial pressure [63].

These findings suggest that the increased urine output following dexamethasone treatment might be caused by two factors: (1) pressure diuresis induced by the increase of arterial pressure and (2) an increase of the osmolar load to the kidney due to an increase of serum urea. This study demonstrates that a significant increase of diuresis occurs in preterm infants, 48-96 hours after starting dexamethasone [63].

Furthermore, a study conducted in the U.S. showed that dexamethasone therapy in newborns for a period of a week or longer was associated with suppression of the hypothalamic-pituitary-adrenal axis (HPAA) in a substantial number of premature infants [32].

Also, Riikonen and Donner evaluated 162 children with infantile spasms who were treated with ACTH. In a large proportion (37%) of the children, the treatment caused pronounced side effects with a mortality rate of 4.9%. The most common complications were infections: septic infections, pneumonias, and urinary and gastrointestinal infections. Other side effects were arterial hypertension (11), osteoporosis (2), hypokalemic alkalosis (2), and other marked electrolyte disturbances (10) [47].

6.6 Corticosteroid increases the risk for infections in infants

Vermillion et al. performed a non-concurrent prospective analysis of singleton pregnancies delivered between 24 and 34 weeks of gestation after antenatal betamethasone exposure. Patients were categorized into two groups according to betamethasone exposure: (1) two 12-mg doses in a 24-hour interval on admission (single-course group) and (2) repeated dosing after the initial single course (multiple-course group). A total of 453 patients were included, with 267 in the single-course group and 186 in the multiple-course group. Multiple courses of antenatal betamethasone are associated with increased risks of perinatal infectious morbidity and neonatal death [64].

Also, a total of 371 low birth weight infants were enrolled in the trial. For the first 14 days of study, 182 infants received dexamethasone (group I) and 189 infants were given placebo (group II). Infants who received a 14-day course of dexamethasone initiated at 2 weeks of age were more likely to develop a bloodstream or cerebrospinal fluid infection while on dexamethasone therapy than were those who received placebo [65]. In a second study**,** the medical records of 158 premature infants were evaluated. Seventy-five infants (58%) received antenatal steroids and 88 infants (68%) received postnatal steroids. Sepsis developed in 44 (34%) infants and fungal sepsis in 14 (11%) [66].

Furthermore, potential side effects of antenatal administration of corticosteroids to prevent neonatal respiratory distress syndrome were evaluated in 10 to 12-year-old children whose mothers had participated in a randomized, double-blind, placebo-controlled trial of betamethasone. In the corticosteroid group, more hospital admissions were reported due to infectious diseases during the first years of life [67].

6.7 Corticosteroid causes retinopathy and other vision problems in patients

Medical records of 158 premature infants were studied. Seventy-five infants (58%) received antenatal steroids and 88 infants (68%) received postnatal steroids. Incidence of retino-

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pathy of prematurity (ROP) was 77% (100/130) and severe ROP (stage ≥ 3) was reported in 52% (68). Postnatal steroid use is an independent risk factor for development of severe ROP [66].

Development of central chorioretinopathy (CSC) following the administration of corticosteroids by diverse routes is a well-known fact**.** Schalenbourg et al. reported acute visual loss after the use of systemic corticosteroids in three patients to treat long-standing ocular inflammatory disorders [68]. Furthermore, Spraul et al. evaluated five patients who developed maculopathy during treatment with systemic corticosteroids. Three patients displayed focal and two patients showed diffuse retinal pigment epithelial changes comparable to the acute and chronic form of central chorioretinopathy, respectively. Four of five patients had a complete visual recovery after decrease or cessation of treatment with corticosteroids. The authors concluded that systemic treatment with corticosteroids may damage the posterior blood-retinal barrier, leading to central chorioretinopathy [69].

In addition, Chaine et al. described fourteen cases of detachment of the macula due to central chorioretinopathy in patients given long-term steroid therapy. None of the patients had hypertension. Detachment occurred between 6 days and 10 years after the start of steroid treatment. The higher the doses, the earlier the onset of ocular disease appeared. All patients were symptomatic, with rapid onset of blurred vision. Detachment was bilateral in two cases [70]. Also, Song et al. reported five patients who were diagnosed as having CSC during systemic corticosteroid treatment based on medical records and fluorescein angiography [71].

7. Brian Herlihy's jury trial and analysis of the evidence presented

Brian Herlihy's jury trial was held in the Eighth Judicial Circuit in Alachua County, Florida on September 10, 2002. Trial lasted sixteen days (Case No. 01-2000-CF-2753-A) and the Honorable Judge Martha Ann Lott presided over this trial. Attorneys Jeanne Singer and Stephen Pennypacker represented the State, and attorneys Gordon Groland and John Tedder represented the defendant [2; 4; 7; 11; 22; 26; 27; 30; 31; 72:311-435, 1369-1503, 1504-1673, 1674-1787, 1788-1961, 1962-2088, 2089-2176, 2177-2267, 2268-2432, 2433-2522, 2523-2534].

The State claimed that Baby Robert was perfectly fine and that there was absolutely nothing wrong with him when his mother brought him to Brian's apartment shortly after 0900 on August 2, 2000. In addition, the State alleged that Baby Robert was never lethargic or anxious from the time of his birth until the morning of August 2, 2000. The State asserted that while Baby Robert was alone with the Brian Herlihy, the baby suffered from violent shaking which ultimately resulted in fatal neurological damage and his death. The State furthermore claimed that Brian punished Baby Robert because the baby was crying and had annoyed, maddened, and frustrated him [72:2226].

Several physicians and the medical examiner examined the baby on August 2nd through 10th and they did not find any sign of injuries on the baby's head or body that was caused by trauma or abuse. Brian has stated to the police numerous times that he is innocent and that he took very good care of the baby. Brian also had cared for Baby Robert about five times in the past, for several hours per day and the baby had been fine [11]. However, Brian Herlihy was convicted of manslaughter in the death of baby Robert and sentenced to 15 years in prison.

I reviewed the medical evidence and trial documents related to this case. I found that the State did not present any medical evidence that showed Brian Herlihy harmed Baby Robert nor that Robert's injuries were caused by violent shaking and trauma. My investigation furthermore revealed that the baby was suffering from serious health problems prior to August 2, 2000 that led to his respiratory arrest, neurological damage, and his death in August of 2000.

The medical examiner and seven physicians testified as expert witnesses for the State. Two physicians testified as experts for the defense. The physicians that testified for the State in this case included: (1) a forensic pathologist, (2) an emergency medicine specialist, (3) a pediatrician/intensive care specialist; (4) a neuropathologist, (5) a pediatric neurologist, (6) a pediatric ophthalmologist, (7) a pediatrician, and (8) an obstetriction/gynecologist. The physicians who testified for the defense are Dr. John Plunkett, forensic pathologist and Dr. Ronald Henry Uscinski, neurosurgeon.

7.1 The testimonies given by the State's expert witnesses are based upon theory

The medical examiner and the State's expert witnesses alleged that Baby Robert's respiratory arrest, neurological damage, and death were caused by violent shaking while he was with Brian Herlihy prior to 0937 on August 2, 2000. My review of the medical evidence described in the previous five sections of this report clearly shows that Baby Robert suffered from chronic health problems (chronic subdural bleed, retinal hemorrhage, brain atrophy, atrophy of the thymus, diabetes, muscle weakness, and sinus and ear infections) that led to his seizure and his respiratory arrest on August 2, 2000.

Robert's serious health problems were caused by his treatment with corticosteroid. In addition, the vaccines given to Baby Robert contributed to his health problems. I described the adverse reactions to vaccines and corticosteroids in Sections 3 and 6 of this report. None of these physicians considered adverse reactions to corticosteroids and vaccines in their evaluation of this case. In addition, none of these physicians reviewed the baby's prenatal and postnatal medical records to learn about Robert's pre-existing chronic health problems.

My review of the medical evidence furthermore revealed that several of these physicians were aware that Baby Robert suffered from chronic health conditions such as a chronic subdural bleed, brain atrophy, and sinus and ear infections. However, they did not make any attempt to investigate the links between the baby's chronic illnesses and his respiratory arrest on the morning of August 2nd, 2000. The following is a list of medical evidence that shows the State's expert witnesses conducted an incomplete medical investigation in this case. They blindly rushed to judgment and their conclusions are invalid and unsupported by the medical facts pertaining to this case.

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(1) The emergency team, several physicians, and the medical examiner examined the baby on August 2nd through 10th and they did not find any sign of injuries on the baby's head or body that was caused by trauma or abuse.

(2) The four cerebral CT scans taken on August 2nd through 4th showed that Baby Robert suffered from a chronic subdural bleed. However, none of the physicians who testified for the State ever investigated the causes of the bleed. Furthermore, the medical examiner did not take a sample from the dura to examine microscopically in order to date the bleed. The data described in Section 6 of this report show that prenatal and postnatal treatments of infants with corticosteroid caused hypertension, hyperatrophic cardiomyopathy, encephalopathy, and increased capillary fragility, which can lead to subdural bleeding.

(3) The treating physician and the neuropathologist were both aware that Baby Robert suffered from brain atrophy but they did not investigate the cause(s) of the atrophy or the link between the atrophy and the baby's seizure and his respiratory arrest that occurred on August 2nd. The treating physician stated, "The baby had a smaller brain than the size of the skull, meaning that there was probably some atrophy or wasting of the surface of the brain or that the brain was not growing as rapidly as it should have been [31]." The neuropathologist said that Robert's brain was an immature brain and it was inconsistent with a brain of a child of four and a half months of age [30].

Baby Robert suffered from severe thymic atrophy which indicates that he had been treated with high doses of corticosteroid. It has been reported that premature infants treated with dexamethasone exhibited a 30% reduction in total cerebral tissue volume compared with total cerebral tissue volume in both premature infants not treated with dexamethasone and the control term infants [34]. Furthermore, dexamethasone given postnatally to infants has been shown to increase the risk of neurologic impairment, neurodevelopmental disability, and the rate of cerebral palsy in preterm infants and later in survivors [35-37].

(4) The lesions observed in the brain were edema and cell necrosis. They were caused by severe global anoxia and ischemia and not by trauma. Brian found the baby was not breathing at 0937 on August 2nd. The treating physician also found the baby was not breathing well and the endotracheal tube was not in place at about 1100 because the baby was suffering from a severe seizure and his tongue was very stiff. His color was gray and ashen [26]. The baby had been suffering from anoxia for at least 60 minutes. This lack of oxygen also caused heart injury as explained in Section 4.

(5) Baby Robert's pediatrician stated in the trial that the baby was normal. However, his examinations showed that the baby suffered from excessive weight gain, polyurea, muscle weakness in the neck region, neurological problems, and possible vision problems. For example, on April 19, 2000, the pediatrician reported that the baby gained eight ounces (227 g) in two days and he would have gained two ounces (57 g) under normal circumstances. The baby's weight at 4½ months was more than 300% his body weight at birth (Table 2).

In addition, the baby's tracking with his eyes was not consistent following an object more than one hundred degrees. The baby also had poor head and neck control, decreased muscle tone in the shoulders and neck, and tight hip flexors [11]. Robert's serum creatinine levels measured on August 2nd and 3rd showed that the baby was suffering from a muscle-wasting problem. His serum creatinine levels were less than 25% of normal (Table 7). It is clear that Robert was suffering from adverse reactions to corticosteroid as described in Section 6. However, the pediatrician overlooked the connection between the baby's treatment with corticosteroid and his symptoms.

(6) The medical examiner found that the weight of Robert's thymus was 4 grams, which is about 20% of the normal weight. However, he stated that Robert's thymus was normal. The average thymus weight in a white, infant male at three and six months of age was found to be 20 and 25 g, respectively [13]. Baby Robert was 4½ months old and his thymus weight should have been about 22.5 g. Treatment with corticosteroid causes immune depression as measured by the reduction in the size and the function of the lymphoid tissues. It is obvious that the medical examiner overlooked an important biological indicator that showed the baby was suffering from a severe adverse reaction to corticosteroid.

(7) The Pediatric Ophthalmologist examined the baby's eyes and found retinal hemorrhage, white spots in the back of the eye, which he called "Purtscher's retinopathy," and a crack in the back of the eye which he called a choroidal rupture. He claimed that these lesions were caused by trauma, however his examination of the eyes and eyelids did not reveal any sign of external injuries caused by trauma.

I presented the findings of several studies in Section 6 of this report that show the treatment of children and adults with corticosteroid caused retinopathy, increased capillary fragility, hypertension, and diabetes. Hypertension and diabetes have also been known to cause retinopathy. The baby suffered from severe thymic atrophy that indicates he was treated with high doses of corticosteroids. In addition, the clinical data presented in Section 4 show that Baby Robert suffered from diabetes. It is very clear that the Pediatric Ophthalmologist overlooked crucial medical evidence that showed the link between the baby's treatment with corticosteroids and the lesions observed in the retina.

8. Conclusions and Recommendations

Baby Robert suffered from gastrointestinal disturbance, reduction in food intake, polyurea, excessive weight gain, myopathy, neurological problems, brain atrophy, chronic subdural and retinal hemorrhage, vision problems, atrophy of the thymus, diabetes, and sinus and ear infections. These symptoms and lesions have been described in preterm infants treated with high therapeutic doses of corticosteroid (Section 6).

Robert was born four weeks premature and his mother was treated with betamethasone (corticosteroid) during the last

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week of her pregnancy. She developed diabetes as a result of this treatment. Robert's thymus weight at autopsy was less than 20% of normal which indicates that he was also treated with high doses of corticosteroids. Thymus weight is a very sensitive biomarker for the exposure to corticosteroids.

In addition, Baby Robert was exposed to micronase during his first three months of life. Robert's mother developed diabetes as a result of her treatment with corticosteroid and she was treated with micronase. Micronase is not recommended as a treatment in nursing mothers due to the risk of causing hypoglycemia in infants. However, she breast-fed Robert during her treatment with micronase.

Furthermore, the baby was given six vaccines at two months of age and he was re-vaccinated again with these six vaccines at four months of age. At that time he was suffering from severe immune depression as indicated by his thymus weight. These vaccines increased his susceptibility to develop infections. The baby suffered from sinus and ear infections as shown by his cerebral CT scans taken on August 2nd. Also, DTP vaccines have been known to increase the risk of developing neurological disorders in children, such as encephalopathy or complicated convulsion(s).

Baby Robert suffered from respiratory arrest on August 2, 2000 between 0920 and 0935. The events that led to his respiratory arrest can be explained as follows: (1) Baby Robert suffered from a seizure prior to 0935 and his seizure resulted from a neurological problem and brain atrophy caused by his prenatal and postnatal treatment with corticosteroids. In addition, the vaccines received on July 19, 2000 might have played a role in triggering Robert's seizure. (2) The severe seizure caused the baby to vomit and that blocked his airways with fluids which subsequently led to his respiratory arrest. The baby vomited a significant amount of formula like fluid and the paramedic used a vacuum to suck about 10 mL of formula from his airway. The baby had been fed about 8 ounces of formula milk within 30 minutes prior to his seizure. (3) Baby Robert suffered from respiratory arrest for at least 60 minutes and that led to severe anoxia, which caused brain and cardiac damage.

The cerebral CT scan taken on August 2nd at 1028 showed that Robert had a multi-generation subdural bleed. The fresh bleed was estimated to be 20-25% of the total bleed. The occurrence of the fresh bleed in the subdura on August 2nd can be explained by the synergistic actions of several factors that included the following: (1) the presence of previous vascular injury in the subdura which led to re-bleeding; (2) Robert suffered from a severe seizure that caused an increase in intracranial pressure; (3) Robert had an elevated heart rate and that led to an increase in the blood pressure; and (4) Robert's pulse rate was 172 at 0938 on August 2nd and injection of relatively large volumes of fluid intravenously led to an increase in the blood volume and an increase in the blood pressure.

The retinal bleed and other retinal vascular changes that were observed by the Pediatric Ophthalmologist on August 2nd can be explained by Robert's treatment with corticosteroid and diabetes. These conditions have been known to cause retinopathy and retinal bleeding as described in Section 6.

The medical examiner and the State's expert witnesses alleged that Baby Robert's respiratory arrest, neurological damage, and death were caused by violent shaking during his stay with Brian Herlihy prior to 0937 on August 2, 2000. However, the emergency teams, several physicians, and the medical examiner examined the baby on August 2nd through August 10th and did not find any sign of injuries on the baby's head or body caused by trauma or abuse.

In addition, none of these physicians reviewed the baby's prenatal and postnatal medical records to learn about his preexisting health problems, his treatment with corticosteroid, or his adverse reactions to vaccines. Also, my review of the evidence in this case has revealed that some of these physicians were aware that Baby Robert suffered from chronic health conditions such as a chronic subdural bleed, brain atrophy, and sinus and ear infections. However, they made no attempt to investigate the links between the baby's chronic illnesses and his respiratory arrest on the morning of August 2, 2000. Presented in Section 7 of this report is a comprehensive list of medical evidence that shows the State's expert witnesses conducted an incomplete medical investigation in this case.

The extensive medical evidence presented in this report clearly shows that Baby Robert died as a result of adverse reactions to corticosteroid and vaccines and that Brian Herlihy is innocent. The evidence also shows that Brian was convicted and imprisoned due to sloppy and incomplete medical investigations. I believe that the State of Florida has the responsibility to review the evidence presented in this report. Furthermore, it is my opinion that the State has the obligation to take immediate action to free Brian from prison. Brian should be reimbursed for all legal expenses incurred in addition to being compensated for his pain and suffering.

The objective of the State and that of physicians should be to focus on determining the factual causes that lead to the illness and death of a child so that they can prevent such problems from happening to other children. Accusing innocent people of abusing and killing children based upon unsupported theory, as it happened in this case, will not prevent the death of other children by vaccines and adverse reactions to medications. However, it certainly places innocent people in prison and causes great suffering. It also costs taxpayers huge sums of money to pay for unnecessary trials and legal fees.

I spent approximately 280 hours evaluating the medical evidence in order to find the factual causes of injuries and death in this case and to write this detailed report. I have also evaluated three other cases from the USA within a 12-month period involving children who died as a result of adverse reactions to medications and vaccines. In these cases, either their parents or their caretakers were falsely accused of killing the children and imprisoned due to false allegations of Shaken Baby Syndrome.

It is my hope that the State of Florida, our federal government, physicians, and our society will take the time to review the evidence presented in the cases that I have evaluated. It is imperative that prompt action is taken to re-evaluate the Shaken Baby Syndrome theory. This theory is not supported by science. Differential diagnosis should and must be used in order to solve complicated medical problems as I have used in these cases to find the factual causes of the injuries and death.

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