# Shaken baby syndrome or medical malpractice?

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**Abstract**

Babies Alan, Robert, Lucas, and toddler Alexa were born at different places and times in the United States of America by different parents. However, they have many things in common: (1) vaccines and medications caused their deaths, (2) without conducting thorough medical and legal investigations, their treating physicians, medical examiners, police, and states accused their parents or caretakers of killing them, and (3) based upon an erroneous theory, their innocent parents or caretakers were imprisoned for killing their children by violent shaking and blunt trauma.

The primary objective of health care providers and the State should be to find and then focus on the facts. It is the duty of the medical establishment to properly investigate the causes of injuries and death of children in cases such as these in order to prevent such tragedies from occurring again. Accusing innocent parents and caretakers of abusing and killing their children based upon unsupported theory, such as SBS, will not prevent the death of another child by vaccines and inappropriate medications. However, it certainly will lead to wrongful incarceration and unimaginable suffering—psychologically to parents/caretakers and physically to those fragile, highly sensitive children it is our duty to protect.


*Keywords:* Shaken Baby Syndrome, medical malpractice, homicide

## Introduction

The falsely accused and their families requested that I evaluate the medical evidence in order to find the factual causes that led to the fatal injuries. I investigated these cases by reviewing prenatal and postnatal medical records, autopsy reports, vaccines and medications given to the children, trial documents and testimonies of expert witnesses, and the medical literature pertinent to these cases. In each case, I used differential diagnosis to evaluate the contributions of agents relevant to the case and the possible synergistic actions among agents in causing injuries and death [1-4].

My findings clearly show that the "shaken baby syndrome" (SBS) theory is not supported by science. The SBS theory has been applied since the early 1970's in cases of babies and toddlers who suffer from subdural and/or retinal bleeding when they do not exhibit signs of external injuries. My investigation of the four alleged SBS cases noted above revealed that the treating physicians and medical examiners were negligent in carrying out proper medical investigations in order to find the factual causes of the bleeding in tissues. The stories of the four children described below provide the medical evidence that supports my conclusions.

## I. Baby Alan: vaccines and heparin-induced injuries

While his 2½ month old son, Alan Ream Yurko, was still alive in the hospital, his father Alan R. Yurko was arrested on November 26, 1997 for having abused the baby by vigorous shaking of the head. When the baby died in hospital, Alan was charged with murder. He entered a plea of not guilty; however, he was convicted by a jury in 1999 in the State of Florida and given a life sentence plus ten years [1].

Investigating this case, I found that baby Alan was born five weeks premature and suffered from chronic health problems. At two months of age, he was given six vaccines (DTaP, Hib, OPV and Hepatitis B) and I conclude that these vaccines caused illness and cardiac arrest. The megadoses of heparin given to the baby in the hospital following his cardiac arrest and prior to autopsy caused bleeding in the subdural space and other tissues. The treating physicians and the medical examiner did not do proper medical investigations in this case. Their testimonies in court were based upon a theory and were not supported by medical facts. The following is a description of my findings.

### I-A. The factual causes of baby Alan's cardiac arrest and bleeding in tissues

Baby Alan was born on September 16, 1997 in the State of Florida. Labor was induced because his mother suffered from oligohydramnios. She also suffered from multiple chronic illnesses during her pregnancy including gestational diabetes, anemia, loss of appetite, spastic colon, urinary and vaginal bacterial infections, and hemorrhoids. She gained only two pounds (907 g) during her entire pregnancy.

Alan spent the first week of his life in the hospital because he suffered from respiratory distress syndrome, jaundice, hypoxia, hypoglycemia, and bacterial infections. Three days following his birth, Alan's serum bilirubin level was l7.4 mg/dL, which is capable of causing encephalopathy [1]. Review of the medical literature reveals that gestational diabetes, oligohydramnios, and jaundice have tremendous negative impacts on prenatal and postnatal developments. These conditions cause congenital anomalies, growth retardation, skeletal deformities, respiratory distress syndrome, hypoxia, hypoglycemia, encephalopathy, and increases in: mortality rate, rate of premature labor, and rate of infections in the newborn [1, 5-12].

doi: 10.1588/medver.2004.01.00017

As noted, baby Alan was given six vaccines (DTaP, Hib, OPV and Hepatitis B) at two months of age (11 November 1997) and sent home without monitoring or medical supervision. Serious adverse reactions, such as apnea, cardiorespiratory problems, and oxygen desaturations that require medical intervention, are commonly associated with vaccination of preterm infants. Preterm babies who were vaccinated at 70 days of age or less, similar to baby Alan, developed the most serious adverse reactions to vaccines. The authors of many well-documented studies concluded that the risk and benefit of vaccination in preterm infants should be evaluated prior to administering vaccines. They also emphasized that preterm infants who received vaccines should be monitored [1, 13-18].

Adverse reactions to vaccines that were administered to baby Alan are not limited to preterm infants. They have also been reported in full-term infants. For example, in the USA, reports to the Vaccine Adverse Event Reporting System (VAERS) concerning infant immunization against pertussis between January 1, 1995 and June 30, 1998 revealed 285 cases of death and 971 cases of non-fatal serious illnesses [1].

At l0 or 11 days following the vaccine injections, baby Alan developed a high-pitched cry, his skin became warm to the touch, and he was increasingly lethargic. On November 24, 13 days post-vaccination, the baby had a cardiac arrest and apnea episode. Alan Yurko drove his baby to the Emergency Room at Princeton Hospital around noon where the baby was admitted with cardiac arrest and apnea; he was then resuscitated.

According to the records, the first examination revealed that the baby was flaccid, his corneas were somewhat cloudy, and he had a gastric ulcer. There was no injury caused by trauma found on his head or the rest of his body, except for a small reddish linear bruise under the right eye. His four-year-old sister had accidentally caused this minor injury when she was handing a baby bottle to her father.

The baby's first blood test revealed that he suffered from metabolic and respiratory acidosis (pH of 7.18), diabetes (blood glucose level of 337 mg/dL and anion gap level of 22 mEq/L), anemia, elevated serum liver and heart enzymes. He also had an elevated white blood cell count (20,900/μL) and platelet count (571,000/μL). The baby was treated with high therapeutic doses of three antibiotics: rocephin, gentamicin, and Claforan® (cefotaxine sodium) to fight the bacterial infections. He was given IV fluids and dopamine and was then transferred to Florida Hospital at about 1400 on November 24 [1].

At Florida Hospital the baby's temperature rose to 105.8ºF (41.0ºC), and his blood glucose reached 397 mg/dL. The treatment with three types of antibiotics reduced his temperature, blood glucose level, and serum enzymes. On November 26, his serum glucose level dropped to a normal level of 95 mg/dL (76% reduction); lactic dehydrogenase (LDH), alkaline phosphotase, and serum glutamic pyruvic transaminase (SGPT) levels dropped by 70%, 47%, and 19%, respectively; and the white blood cell count by 35%. These data clearly indicate that the baby had a systemic bacterial infection and that it resolved because of the treatment with antibiotics. The baby also suffered from hypotension, dysrhythmia, dehydration and weight loss (lost 1.05 lb or 476.3 g in five days). The baby was given IV fluids, plasmanate, red blood cells, heparin, potassium, dopamine, and anti-diuretic hormones.

Furthermore, the baby was treated with excessive amounts of sodium bicarbonate on November 24, and this treatment caused metabolic alkalosis, hypoxia, hypokalemia, and cerebral edema. His blood pH increased from 7.10 to 7.67. When a patient exhibits high blood pH, the release of oxygen from hemoglobin to the tissues is reduced significantly. In addition, the baby was also given heparin at a high dose level of 219 IU/kg per hour beginning at 1445 o'clock. At 1515, blood analysis indicated elevated prothrombin time and fibrinogen split product levels [1].

Heparin has been known to cause serious hemorrhagic events when given to patients suffering from anemia, hypotension, and unexplained symptoms similar to those of baby Alan. A computerized tomography (CT) scan of the brain taken at 1950 showed intraparenchymal hemorrhage and a subdural hematoma on the right side of the brain. Based on the hourly heparin dose (219 IU/kg per hour), the estimated total dose infused in five hours was 1095 IU/kg, which is about 8.8 times the recommended maintenance dose of 125 IU/kg per five hours for infants [1].

Unfortunately, the baby was again treated with excessive doses of sodium bicarbonate and heparin (219 IU/kg per hour) on November 25, despite his problems with metabolic alkalosis (pH 7.61) and bleeding in the brain. This treatment resulted in metabolic alkalosis, hypoxia, hypokalemia, cerebral edema, and bleeding. His serum potassium level dropped from 4.9 mEq/L to 2.3 mEq/L. Baby Alan also suffered from disseminated intravascular coagulation (DIC) as a result of his treatment with heparin.

The platelet count prior to the administration of heparin on November 24, 1997 was 571,000/μL of blood, and dropped to 397,000/μL (30% reduction) on November 25. Heparin increases the tendency of the platelets to aggregate and form a clot. Also, blood analysis performed at about 30 minutes post-heparin infusion showed increased fibrinogen split product level (160 μg/mL) and prothrombin time (11.6 seconds). These values are 1600% and 115% of normal, respectively. These values returned to normal on November 26 following the cessation of the treatment with heparin.

Approximately 75 hours after initial hospital admission, on November 27, 1997, baby Alan was pronounced brain dead. On November 29, the Chief Medical Examiner of District Nine in Orlando, Florida performed an autopsy whose main objective was to establish the cause(s) of death [19]. Prior to autopsy, Translife, a company specializing in donor organ removal and transport, took the baby's heart, liver, pancreas, and a portion of the intestine for organ transplant. Prior to and during the harvesting of these organs, baby Alan was given 22,950 IU of intravenous heparin. That is more than 100 times the recommended therapeutic dose of heparin (25 IU/kg).

### I-B. The allegations against Alan Yurko are false

Alan R.Yurko's jury trial took place from February 22 to 24, 1999 in the state of Florida [1, 20]. The prosecutors sought the opinions of the medical examiner and three other physicians to serve as expert witnesses testifying for the state. These physicians stated that baby Alan died as a result of shaking and blunt trauma to the head. However, none of them provided medical evidence to prove their claims. Their testimonies were based

doi: 10.1588/medver.2004.01.00017

only upon a theory. The following is a list of inadequacies contained within the State's expert witnesses' testimonies that invalidate their own conclusions [1].

1. None of the State's witnesses reviewed the baby's prenatal or birth records, his doctor's charts during his two months of weekly visits, or the adverse reactions to vaccines and medications given to the baby. In addition, they never interviewed his parents to get a complete case history.

2. The treating physician did not reveal to the court the following important clinical events that show the baby died of natural causes: (1) the baby had high blood glucose levels resulting from diabetes and complications of diabetes, such as dehydration, gastric ulcer, infections, cerebral edema, hypokalemia, loss of weight, and cardiac dysrhythmia; (2) he treated the baby with three types of antibiotics to fight bacterial infections and the baby responded very well to this treatment; (3) he treated the baby with excessive doses of sodium bicarbonate and heparin that caused bleeding, metabolic alkalosis, hypoxia, and edema.

3. The medical examiner stated in court that the bleeding in the subdura happened within seconds or minutes on November 24. However, his descriptions of the bleeding in the subdural spaces of the brain and spinal cord presented in his autopsy report indicate that the bleeding occurred in at least three stages during a two to five day period.

4. The presence of hemorrhaging in the lungs, brain, and spinal cord does not support the Medical Examiner's claim that the bleeding was caused by vigorous shaking of the head, but shows that it was induced by the megadoses of heparin and by metabolic and cardiovascular problems. The baby was given large doses of heparin on November 24 and 25 and a megadose of heparin (more than 100 times the therapeutic dose) prior to autopsy. Heparin causes bleeding even when it is given at the therapeutic level.

5. The Medical Examiner described the histology of the heart in his autopsy report, but the heart was donated several hours prior to autopsy and therefore was not available for examination.

6. The Medical Examiner stated that the baby's head circumference was 22 cm, whereas the head circumference had been reported to be 37.5 cm eighteen days prior to the autopsy.

7. The Medical Examiner stated in court that he did not take a sample of cerebrospinal fluid (CSF) at the time of autopsy to check for meningitis, because it was mixed with blood. However, he stated in his autopsy report that the CSF fluid was clear which clearly contradicted his testimony.

8. The Medical Examiner claimed that the baby did not suffer from meningitis, although his autopsy report and the clinical evidence described in the baby's chart, as well as the pathology evidence indicate that the baby suffered from acute meningitis. The meningial blood vessels were swollen and congested and the meningial tissue was infiltrated with inflammatory cells. The baby had an elevated white blood cell count and body temperature of 105.8ºF (41.0ºC). Gore also overlooked evaluating whether the antibiotic treatment had any influence on the lesions in the meninges.

9. The State's witnesses claimed that vigorous shaking of the head causes axonal injury in the brain. However, none of them provided any evidence in court that axonal injuries in the brain actually existed. Additionally, no description of axonal injury was mentioned in the autopsy report.

10. The Medical Examiner presented the minor retinal bleeding in the right eye as evidence that baby Alan died as a result of "shaken baby syndrome." However, he neglected to investigate the factual causes that led to the retinal bleeding such as the megadoses of heparin, the diabetes, infections, and hypoxia.

During Alan Yurko's jury trial, the prosecutors presented only one theory—that baby Alan died of "shaken baby syndrome" (SBS), and that Alan Yurko, his father, did it. They did not question the list of the discrepancies in the testimonies of the medical examiners and other physicians described above. On the contrary, the prosecutors allowed the medical examiner to present as evidence two photographs of minor contusions in the temporal areas of the head, which occurred in the hospital about one day prior to autopsy.

The evidence presented in this case clearly shows that Alan Yurko is innocent and that the physicians and the State conducted grossly unscientific and incomplete investigations. It seems incredible that Florida Hospital and the treating physician contacted the Orange County Sheriff's Office and "The Child Protection Team" on November 24, 1997. They filed a child abuse report based on the assumption that baby Alan had been injured as a result of abuse. Alan Yurko was arrested two days later while his baby was still alive. It is interesting to note that the treating physician was the very same physician responsible for treating the baby with excessive doses of sodium bicarbonate and heparin which, in turn, caused hypoxia and bleeding.

The Yurko case is not by any means an isolated incident in the state of Florida wherein a baby died as a result of adverse reactions to vaccines and medications, and an innocent caretaker was accused of causing the death.

## II. Baby Robert: corticosteroid and vaccine induced health problems

Robert Benjamin Quirello was 4½ months old when he suffered from respiratory arrest at Brian Herlihy's apartment on the morning of August 2, 2000. Brian is a white male; he was 29-years old at the time. Robert's mother came to 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 previous occasions for a few hours per day. Brian was arrested on August 3, 2000 based on verbal communications between the treating physicians and police. At this point, the baby was still alive in the hospital. The doctors told the police that the baby was suffering from injuries caused by shaking. The baby died on August 10, 2000 [2].

Brian Herlihy's jury trial was held in the Eighth Judicial Circuit in Alachua County, Florida on September 10, 2002, and the trial lasted sixteen days (Case No. 01-2000-CF-2753-A). The State claimed that Robert Quirello 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 Robert was alone with Brian he suffered from violent shaking, which ultimately resulted in fatal neurological damage and death. The State claimed that Brian had punished baby Robert because the baby was crying and had

doi: 10.1588/medver.2004.01.00017

annoyed, maddened, and frustrated him [2, 21]. In addition, the State alleged that 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 never harmed him. However, in September of 2002, Brian was convicted of involuntary manslaughter in the death of baby Robert and sentenced to 15 years in prison [21].

My investigation revealed that the State's allegations are false and that Robert died as a result of adverse reactions to corticosteroid and vaccines. The medical examiner, physicians, and police did not conduct thorough medical and legal investigations to determine the factual causes of injuries and death in this case. They rushed to judgment and accused Brian of killing Robert, even though some of the physicians were aware that Robert was suffering from chronic health conditions. Below is a description of the medical facts that support my conclusions.

**II-A. The factual causes of baby Robert's respiratory arrest and bleeding**

Robert's mother was involved in a serious car accident on January 10, 2000, at 26 weeks of gestation. Her abdominal region was hit 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 for about one week and released. Robert's mother went into premature labor at 34 weeks of gestation. She spent eight days in labor, and Robert was born on March 22, 2000, four weeks premature and with a broken collarbone.

Robert's mother was treated with betamethasone (corticosteroid) during the last week of her pregnancy, thus Robert was exposed to corticosteroid in utero. It seems that she developed diabetes as a result of her treatment with corticosteroid, as 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, his mother breastfed Robert during her treatment with Micronase [2].

Baby Robert suffered from serious health problems resulting from corticosteroid exposure in utero and postnatal treatment with it. These problems included gastrointestinal disturbance and reduction in food intake, polyurea, excessive weight gain, myopathy, neurological problems, brain atrophy, chronic subdural and retinal hemorrhaging, vision problems, atrophy of the thymus, diabetes, and sinus and ear infections [2]. These symptoms and lesions have been reported in infants treated with corticosteroids [2, 22-31]. However**,** none of the physicians who evaluated this case ever addressed this issue.

Furthermore, Robert was given six vaccines on May 9, 2000, and this procedure was repeated on July 19. Premature babies are usually more susceptible to adverse reactions to vaccines than full-term babies. Furthermore, vaccines should not be given to children treated with corticosteroid or other immunosuppressants. 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 his age. Thymus weight is also an extremely sensitive biomarker of corticosteroid exposure [2, 13-18, 32, 33].

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

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) He experienced a seizure prior to 0935 which resulted from a neurological problem and brain atrophy caused by the prenatal exposure to and postnatal treatment with corticosteroids. In addition, the vaccines received on July 19, 2000 may have played a role in triggering the **s**eizure. (2) The severe seizure caused the baby to vomit, blocking his airways with fluids and leading to respiratory arrest. The baby expelled significant amounts of vomitus. In addition, the paramedic used a vacuum to extract about 10 mL of fluids including formula from his mouth and nose. He had been fed approximately 8 ounces (227 g) of formula within 30 minutes prior to his seizure.

Robert's period of intermittent respiratory arrest lasted for at least 60 minutes which led to severe anoxia with resultant brain and cardiac damage. As noted, the baby also suffered from chronic subdural and retinal bleeding as a result of treatment with corticosteroid. Administered at high doses, corticosteroid induces diabetes, hypertension, brain atrophy, and increases capillary fragility and abnormal vascular growth in the retina. Corticosteroid causes hypertension and cardiovascular disease due to its capacity to promote sodium retention and increased 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 fresh blood in the subdural space on August 2 can be explained by the synergistic actions of several factors: (1) the presence of previous vascular injury in the subdura, which led to re-bleeding, (2) severe seizure that led to an increase in intracranial pressure, (3) elevated heart rate leading to increased blood pressure (Robert's pulse rate was 172 per minute at 0938 on August 2), and (4) intravenous injection of relatively large volumes of fluid by the medical staff led to an increase blood volume which led to an increase in blood pressure.

The retinal bleed and other retinal vascular changes observed by Dr. Lawrence Levine on August 2 can be explained by Robert's treatment with corticosteroid and by his diabetes. These factors are known to cause retinopathy and retinal bleeding [2].

**II-B. Allegations against Brian Herlihy are false**

The medical examiner and the State's expert witnesses alleged that Robert's respiratory arrest, neurological damage, and his death were caused by violent shaking that occurred while he was at Brian Herlihy's apartment prior to 0937 on August 2, 2000. However, not one 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 [2, 21].

Furthermore, my review of the evidence has revealed that some of these physicians were aware that Robert was suffering from chronic health conditions such as chronic subdural bleeding, brain atrophy, and sinus and ear infections. Nonetheless,

doi: 10.1588/medver.2004.01.00017

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. Following is a list of medical evidence that shows the State's expert witnesses conducted incomplete medical investigations, and that they rushed to judgment by accusing Brian. Their conclusions that the baby died as a result of shaking were based on a theory and not on medical facts or sound scientific principles [2, 21].

1. The emergency teams, several physicians, and the medical examiner examined the baby from August 2 through August 10. During these examinations 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 2 through August 4 showed that Robert was suffering from chronic subdural bleeding. However, none of the physicians who testified for the State investigated the causes of the chronic bleeding. Prenatal exposures 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. Furthermore, the medical examiner failed to take a sample from the dura to be examined microscopically in order to date the bleed.

3. The treating physician and the neuropathologist were aware that 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 of August 2. 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." The neuropathologist also said, "Robert's brain was an immature brain and it is inconsistent with a brain of a child of 4½ months of age."

It has been reported that premature infants treated with dexamethasone exhibited a 30% reduction in total cerebral tissue volume when compared with total cerebral tissue volume in both control term infants and premature infants not treated with dexamethasone. Furthermore, dexamethasone given postnatally to infants increased the risk of neurologic impairment, neurodevelopmental disability, and the rate of cerebral palsy in preterm infants and later in survivors. Robert was treated with high therapeutic doses of corticosteroid as indicated by the severity of his thymic atrophy. His thymus weight was less than 20% of normal.

4. At autopsy, the lesions observed in Robert's brain consisted of cell necrosis and edema, which were caused by severe global anoxia and ischemia, not by trauma. The baby was not breathing properly for at least 60 minutes. Brian found the baby was not breathing at 0937 on August 2. In addition, the treating physician found at approximately 1100 that the baby was not breathing well because of the severe seizure in which his tongue was very stiff, blocking the airways.

5. Robert's pediatrician stated in court 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. The baby demonstrated poor head and neck control, decreased muscle tone in the shoulders and neck, and tight hip flexors. In addition, the baby's visual tracking was not consistent in following an object for more than a hundred degrees. These symptoms have been reported in infants treated with corticosteroid.

6) The medical examiner found that Robert's thymus weight was 4 grams which is about 20% of normal. However, he stated in court that the thymus was normal. The average thymus weight in a white infant male of Robert's age (4½ months old) is expected to be about 22.5 grams. Treatment with corticosteroid causes immune depression and is measured by the reduction in thymus size and decreased functions of the lymphoid tissues. It is clear that the medical examiner overlooked important biological indicators showing that Robert was suffering from severe adverse reactions to corticosteroid.

7) The 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 referred to as a choroidal rupture. He claimed that these lesions were caused by trauma although his examination of the eyes and eyelids did not reveal any external lesions that indicated trauma.

Again, it has been shown that treatment of children and adults with corticosteroid causes retinopathy, hypertension, diabetes, and increased capillary fragility. Hypertension and diabetes have also been known to cause retinopathy. This baby was treated with high doses of corticosteroid. In addition, he suffered from diabetes. It is very clear that the ophthalmologist overlooked crucial medical evidence that revealed the link between the baby's treatment with corticosteroids and the lesions found in the retina.

Medical evidence presented in this report clearly shows that Robert died as a result of adverse reactions to corticosteroid and vaccines; Brian Herlihy is innocent. The evidence also reveals that Brian was convicted and imprisoned as a result of sloppy and incomplete medical investigation. I believe that the state of Florida has the responsibility to review the evidence presented in this report and is obligated to take immediate action to free Brian from prison.

The tragedy of infant death from adverse reactions to vaccines and medications in which parents or caretakers are falsely accused of shaking and killing them is not limited to premature infants or to the state of Florida. Baby Lucas' case described below exemplifies that it also happens to full-term babies in other states.

## III. Baby Lucas: antibiotics and vaccine induced health problems

Alejandro Mendez was accused and arrested for killing his 3½ month old son Lucas by blunt-force trauma to the head. The baby suffered from cardiac arrest and apnea on August 27, 2002. Emergency Medical Services (EMS) resuscitated the baby, treated him with epinephrine, and transported him to the hospital. My investigation revealed that baby Lucas suffered from vitamin K deficiency, chronic bleeding in the subdural space, and systemic infections. As described below, his health problems were induced by his exposure to antibiotics in milk and by the vaccines administered to him.

doi: 10.1588/medver.2004.01.00017

**III-A. The factual causes of baby Lucas' cardiac arrest and bleeding**

Lucas was born at 41 weeks of gestation on May 16, 2002. He was in excellent health until the day of his vaccinations on July 23 when he was 9 weeks of age. He was simultaneously administered seven vaccines (DTaP, Hepatitis B, Hib, IPV, and Pneumoccocal vaccine). He developed an upper respiratory tract infection within 1 to 2 days post-vaccination and was treated with Tylenol® for two to three days as per the nurse's instructions. At seven days post-vaccination Lucas' mother took him to his pediatrician because he was still suffering from an upper respiratory tract infection [3].

Serious adverse reactions to vaccines (including death) in children have been reported in the medical literature [3,13,18,34,35]. For example, in the USA, reports to the Vaccine Adverse Event Reporting System (VAERS) concerning infant immunization against pertussis between January 1, 1995 and June 30, 1998 revealed 285 cases of death and 971 cases of non-fatal serious illness. The vaccines given to Lucas on July 23, 2002 induced an upper respiratory tract infection within 1 to 2 days post-vaccination. He also developed pneumonia and a bacterial urinary tract infection. Lucas' systemic infections caused hyperglycemia and metabolic acidosis that subsequently led to the reduction of potassium levels in the cardiac muscles and nervous tissues which, in turn, led to cardiac arrest.

In addition, one day prior to Lucas' vaccination, his mother suffered from mastitis. She was treated with a ten-day course of dicloxacillin. She breastfed Lucas during her treatment with this antibiotic and he developed diarrhea. Furthermore, Lucas' mother had also been treated with an eleven-day course of an antibiotic on May 20 when Lucas was just four days old. Once again she breastfed him during her treatment with antibiotics as she had been assured by both her midwife and doctor that it was safe. His mother's treatment with antibiotics predisposed Lucas to vitamin K deficiency by reducing the levels of vitamin K in her breast milk. This treatment also caused Lucas' diarrhea, and reduced both vitamin K synthesis in Lucas' gastrointestinal tract (GIT) and vitamin K uptake from the GIT [3].

On August 27, 2002, at approximately 1330, Lucas was put down for a nap after being fed. His father found him unresponsive shortly afterwards. Emergency Medical Services (EMS) was called. Upon arrival, EMS found Lucas unresponsive with agonal respirations and mottled skin. The infant was placed on a monitor and was given 0.1 mg of epinephrine via an interosseous route. Lucas' blood glucose level was found to be 382 mg/dL.

The unresponsive baby arrived at Centre Community Hospital (CCH) at about 1350 with tachycardia and a perfusing pulse. The child was in rhythm at 175 to 180 beats per minute. He had bilateral retinal hemorrhage and the fontanel was full. He was transferred from the CCH to Geisinger Medical Center by Life Flight at about 1430 on August 27, 2002.

A physical examination on admission to the Pediatric Intensive Care Unit at Geisinger Medical Center revealed a temperature of 35ºC (95ºF), heart rate of 94, blood pressure of 94/62 mmHg, bulging anterior fontanel, non-reactive pupils, and pinpoint eyes. The gastrointestinal area was soft, non-tender, non-distended and no bowel sounds were heard. In addition, ecchymosis on the right eyelid (1-2 mm), below left eyelid (2 mm) and on the back (4 mm), and bloody endotracheal tube secretions were observed [10].

The clinical tests performed at the hospital showed that Lucas was suffering from diabetes mellitus and complications of diabetes (metabolic acidosis and reduction of potassium levels in cardiac muscles and nervous tissues which led to his subsequent cardiac arrest and apnea) and respiratory acidosis. He also suffered from a bacterial urinary tract infection, liver damage, vitamin K deficiency, and bleeding in the brain and other locations. Lucas' serum glucose levels on August 27 at 1431 and 1920 were 382 and 415 mg/dL, respectively. Normal serum glucose range is 70-110 mg/dL. The levels of serum ALT and AST enzymes were 342% and 255% of normal, respectively, which indicate liver damage.

In addition, Lucas' blood pH was 7.22 at 1431 and dropped to 6.64 on August 28 at 0315. Lucas had metabolic acidosis, as indicated by the blood pH of 6.64, low blood $CO_2$ level (14.5 mmol/L), low blood bicarbonate level (9.9 mEq/L), and high anion gap (21 mEq/L). The levels of lactic acid in the blood were found to be critically high due to diabetes and hypoxia. Urinalysis also demonstrated high levels of ketone bodies.

In metabolic acidosis resulting from diabetes, potassium usually leaves the intracellular environment because the intracellular proteins bind with hydrogen, leading to cardiac arrest and respiratory paralysis. At this stage, serum potassium levels are usually normal or elevated; but after treatment with bicarbonate, elevating pH to normal or above, potassium leaves the blood and goes back inside the cells. This leads to hypokalemia, as we observed in Lucas' case [5, page 2060].

Lucas' serum potassium level was 5.2 mEq/L on August 28 and dropped to 2.6 mEq/L on August 31 following his treatment with excessive amounts of sodium bicarbonate (blood pH was 7.67). He was also treated with potassium solutions by IV infusion several times between August 31 and September 1 to correct his hypokalemia.

Lucas also suffered from cerebral edema as a result of being diabetic. Cerebral edema is a common cause of death in diabetic children and it occurs much more frequently in children than in adults [5, 6, 9]. Lucas had cerebral edema as shown in the CT scans and stated in the autopsy report [3, 36]. At the time of admission to Geisinger Medical Center, the brain edema was mild; nonetheless, treatment with excessive doses of sodium bicarbonate increased the severity of the edema as evidenced by the CT scans. The scan of the brain taken on August 27 at 1806 showed that the ventricles were non-dilated and that no evidence of hydrocephalus was seen at this time. However, the CT scan of the brain taken on August 29 at 0816 showed cerebral edema and impending downward transtentorial herniation.

Furthermore, a CT scan taken on August 30 showed diffuse edema of the hemispheres bilaterally. The effacement of the sulci, basal cisterns and ventricles were increased as compared with the prior exam. The accumulation of fluid in the brain led the doctors to operate and drain the excess cerebrospinal fluid on August 30, 2002.

Treatment with sodium bicarbonate and excessive fluids caused edema in the lungs. The chest x-ray taken on August 27 at about 1400 showed no fluid accumulation in the lungs. However, the chest x-ray taken on September 1, 2002 at 0925 showed more diffuse abnormalities throughout the lung field

doi: 10.1588/medver.2004.01.00017

than were present on the earlier study. The appearance is compatible with pulmonary edema [3].

Lucas was treated with sodium bicarbonate to correct acidosis. However, he was given excessive amounts. His blood pH was 7.22 on August 27 and it rose to 7.67 on August 31. Bicarbonate therapy may be indicated in severely acidotic patients (pH 7.0 or below), especially if hypotension is present (acidosis itself can cause vascular collapse). Bicarbonate is not routinely used in less acutely ill subjects because rapid alkalization may have detrimental effects on oxygen uptake in tissues [5, p 2073].

Alkalization increases the avidity of hemoglobin to bind oxygen, impairing the release of oxygen in peripheral tissues. If bicarbonate is given, the infusion should be stopped when the pH reaches 7.2 in order to minimize possible detrimental side effects and to prevent metabolic alkalosis as circulating ketones are metabolized to bicarbonate with reversal of ketoacidosis. The key parameters to follow are the pH and the calculated anion gap. It is very obvious that these vital treatment recommendations were not followed in Lucas' case as his treatment with excessive amounts of bicarbonate led to severe hypoxia as well as cerebral and pulmonary edema [3, 37-39].

Furthermore, Lucas suffered from hypoxia as a result of his severe anemia which is shown by very low hemoglobin of 6.2 g/dL and hematocrit of 18%. His apnea, cardiac arrest, and hypotension also resulted in hypoxia and general ischemia of the brain. During Lucas' hospitalization, the level of fibrinogen increased from 150 mg/dL (normal level in newborn**:** 125 mg/dL-300 mg/dL) on August 27 at 1900 to 388 mg/dL on August 29**.** Fibrinogen is an acute-phase reactant that becomes elevated with tissue inflammation or tissue destruction.

At the time of Lucas' admission to CCH, Dr. Clifford J. Neal examined him and did not see any evidence of ecchymotic lesions on the skin. However, the examination of the baby at Geisinger Medical Center showed that Lucas had ecchymosis on the right eyelid (1-2 mm), below the left eyelid (2 mm) and on the back (4 mm). In addition, bloody endotracheal tube secretions were observed. The CT scan of the brain taken on August 29 at 0816 showed an increase of blood in the interhemispheric fissure and extraparenchymal hemorrhage as compared with the scan of August 27. There were also multiple new foci of acute intraparenchymal and subdural hemorrhages.

At the time of hospital admission to Geisinger Medical Center, Lucas had bleeding in the brain resulting from vitamin K deficiency. The CT scan of the brain taken on August 27 at 1806 showed a subdural hematoma and bleeding in the brain. Furthermore, the blood products were of various ages. The bleeding worsened as a result of his treatment with epinephrine following admission. Epinephrine causes rapid rise in blood pressure, cerebral hemorrhage and bleeding in other locations [3].

The bleeding in the brain, eyes, and other locations in Lucas' case was caused by vitamin K deficiency as indicated by several biomarkers that included (1) the PIVKA-II protein was found to be grossly elevated (22.7 ng/mL) on August 28 (normal range 0.0-3.5 ng/ml), (2) Lucas' prothrombin time and the partial thromboplastin time were elevated (17.3 seconds and 38 seconds, respectively), and treatment with vitamin K on August 28 reduced prothrombin and partial thromboplastin time by 20% and 25%, respectively; (3) bleeding was also observed in several locations of the body in addition to the brain; (4) the bleeding in the brain represented several different stages (acute, subacute, and chronic). That finding clearly shows that the bleeding had begun several days or weeks prior to August 27, 2002.

Vitamin K is essential and it has coagulation activity. In vitamin K deficiency, abnormal decarboxylated coagulation factors appear, which are known as "proteins induced by vitamin K absence" (PIVKAs) [40-43]. This is a unique biomarker for vitamin K deficiency. PIVKA-II proteins are usually undetectable in healthy infants who receive adequate amounts of vitamin K. A study of vitamin K1 and (PIVKA)-II concentrations was conducted in healthy, breastfed infants at the ages of 2, 4, 8 and 12 weeks, once with 1 mg vitamin K1 orally (n = 165) or intramuscularly (n = 166); or weekly 1 mg orally (n = 48); or daily 25 micrograms orally (n = 58). The 25 micrograms per day was the only regimen found to be effective in the prevention of vitamin K deficiency in breastfed infants during the first three months of life [44].

In a second study, thirteen breastfed infants were given 1 mg vitamin K1 by intramuscular injection at birth. The levels of vitamin K in plasma reached as high as 32711 $\pm$ 25375 pg/mL shortly after birth. However, at one month of age the vitamin K1 levels of these infants were down to 698 $\pm$ 536 (n = 9) and this is the range found in breastfed infants not receiving vitamin K prophylaxis [41]. Also, Verity et al. presented three infants with the late-onset form of hemorrhagic disease of the newborn (4, 6, and 7 weeks after birth) who had received 1 mg of vitamin K at birth [54]. Lucas was given only 1 mg vitamin K at birth; based on these studies, this dose is insufficient to have prevented vitamin K deficiency in his case.

Furthermore, both prothrombin time (PT) and partial thromboplastin time (PTT) were elevated at the time of Lucas' hospital admission. PT and PTT are considered important indicators for vitamin K deficiency [40-42, 44-46]. PT and PTT were measured in fifteen infants at the age of 30 to 150 days who suffered from vitamin K deficiency and were found to be highly elevated. They were reduced sharply in a few hours following the administration of vitamin K1. Before administration of vitamin K, PT was 76.1 $\pm$ 43.0 seconds and PTT was 123.4 $\pm$ 68.8 seconds. Six to 12 hours after administration of vitamin K, PT and PTT were reduced to 15.6 seconds and 33.4 seconds, respectively [45].

Vitamin K deficiency should be suspected in nearly all infants with findings in screening coagulation studies. An otherwise healthy-appearing infant with hemorrhaging should be suspected of having vitamin K deficiency. Final diagnostic confirmation of vitamin K deficiency is a rapid, therapeutic response to vitamin K1 administration [47] as happened in Lucas' case.

Newborns and infants who develop vitamin K deficiency usually suffer from bleeding in the brain and other locations similar to those observed in Lucas. In a study conducted in Japan, intracranial hemorrhage was observed in 353 (75%) cases out of 473 infants aged 2 weeks to 4 months who suffered from vitamin K deficiency [48].

In a second study, fifteen infants aged 30 to 150 days who developed bleeding in the brain and other locations were found

doi: 10.1588/medver.2004.01.00017

to be suffering from vitamin K deficiency. All infants were breastfed and were born at term from healthy mothers. The delivery histories were uneventful and there was no history of vitamin K administration at birth. In nine infants, cranial tomography (CT scan) was taken and showed intraparenchymal, intraventricular, and subarachnoid hemorrhage. In addition, two infants had neurologic manifestations and hemorrhagic findings in the cerebrospinal fluid. Skin bleeding (ecchymosis) was also observed in three patients [45].

Bleeding in the brain was observed in eleven infants between 30 and 119 days of age (mean: 56 $\pm$ 24 days) who developed vitamin K deficiency. None of these infants received vitamin K after birth and all of them were breastfed. The localization of the intracranial hemorrhage was as follows: intracerebral (91%), subarachnoid (46%), subdural (27%), and intraventricular (27%) [49].

In addition to the bleeding in the brain and other locations, Lucas also suffered from diarrhea and vomiting after meals. He also exhibited a lack of neurologic responses similar to those described in infants who developed vitamin K deficiency. In a study that included fifteen infants (30 to 150 days of age) who suffered from vitamin K deficiency, signs and symptoms of the patients were convulsions (47%), feeding intolerance and poor sucking (47%), irritability (33%) and pallor (20%). On physical examination there was found bulging or full fontanel in ten patients, diminished or absent neonatal reflexes in nine patients, and ecchymosis in three patients [45].

In a second study, eleven breastfed infants between 30 and 119 days of age who developed vitamin K deficiency were examined. The presenting complaints were seizures (91%), drowsiness (82%), poor sucking (64%), vomiting (46%), fever (46%), pallor (46%), acute diarrhea (27%), irritability and high-pitched cry (18%). On examination the most frequent findings were tense or bulging fontanel (73%), anisocoria (36%), weak neonatal reflexes (18%), and cyanosis (18%) [49].

There are many factors that led to vitamin K deficiency in Lucas' case, including the following: (1) Lucas was breastfed, and human milk is very low in vitamin K, (2) Lucas' mother was treated with antibiotics while breastfeeding him, which reduced the synthesis of vitamin K in her intestinal tract and the level of vitamin K in her milk, (3) Lucas was breastfed milk containing antibiotics, which reduced the synthesis of vitamin K in Lucas' intestinal tract and caused diarrhea, (4) Lucas suffered from diarrhea and malabsorption which reduced vitamin K absorption from the intestinal tract, (5) Lucas suffered from liver damage as shown by the elevation of serum liver enzymes, and that consequently reduced the synthesis of vitamin K, and (6) Lucas suffered from diabetes and a urinary tract infection which reduced food intake.

The current worldwide problem of Vitamin K deficiency in breastfed infants persists also because the prior transfer of vitamin K from mother to infant through the placenta is very poor. Vitamin K concentrations in human milk are very low [50]. The daily requirement for vitamin K in an infant is about 1 μg/Kg and breast milk contains 1 to 3 μg vitamin K/L. In addition, the neonatal liver is immature with respect to prothrombin synthesis and the neonatal gut is sterile during the first few days of life [40].

The newborn usually has undetectable vitamin K levels in serum with abnormal amounts of the coagulation proteins and undercarboxylated prothrombin. The recommended dietary intake (RDI) for infants up to 6 months is 5 μg/day and vitamin K1 intake in human milk-fed infants of about 0.5 μg/day [51,52]. Plasma vitamin K concentrations in the infants fed human milk remained extremely low (mean <0.25 ng/mL) throughout the first six months of life compared with the formula-fed infants (4.39 to 5.99 ng/mL) [44]. The daily intake of formula-fed infants was found to be 50 μg/day [51].

Hemorrhagic disease of the newborn, secondary to vitamin K deficiency, remains largely a disease of breastfed infants [50]. Vitamin K deficiency causes hypoprothrombinemia and reduces the concentration of the other vitamin K-dependent coagulation factors, manifested by defective coagulation and hemorrhage [40]. Hemorrhages were observed in four exclusively breastfed infants within a period of 8 weeks. The onset of bleeding was unexpected and without prior indication. The bleeding was of a serious nature and involved the Central Nervous System (CNS) in two children. There was a prompt improvement after administration of vitamin K. These four cases confirm the necessity to consider vitamin K deficiency in hemorrhages found in infants during the post-neonatal period [53].

In addition, Lucas suffered from chronic diarrhea, vomiting, and liver damage, which in effect reduced the synthesis of vitamin K and coagulation factors in the liver, thereby reducing the synthesis and uptake of vitamin K from the intestinal tract. In general, the oral intake of therapeutic doses of antibiotics usually alters the balance of normal colonic flora and allows overgrowth of Clostridium difficile, an anaerobic gram-positive bacillus. Colonization occurs by the fecal-oral route through the ingestion of heat-resistant spores that persist in the environment for long periods.

Diarrhea and colitis are caused by toxins produced by pathogenic strains of C. difficile. Almost any antibiotic can lead to C. difficile infection. The occurrence of diarrhea is found to be more frequent with use of broad-spectrum antibiotic penicillins (e.g., ampicillin, amoxicillin) and cephalosporins [40]. The use of penicillin by nursing mothers can cause diarrhea in breastfed infants [55]. Diarrhea and malabsorption can predispose to vitamin K deficiency in infants. If the mother has ingested a cephalosporin antibiotic, the risk of hemorrhage increases [5]. Lucas suffered from diarrhea following the use of dicloxacillin (penicillin) by his mother in July of 2002. In addition, his mother was also treated with cephalexin (cephalosporin) antibiotic in May of 2002 while she was breastfeeding Lucas.

A study was undertaken to determine the frequency of vitamin K deficiency in seventy-five infants with diarrhea when compared with eighteen healthy infants used as a control. Screening coagulation tests PT and PTT were performed, along with estimation of functional activity and total antigenic levels of prothrombin. PT was prolonged in 30% (24/75) of all infants with diarrhea as compared to controls, where the abnormality was observed in 11.1% of infants (2/18). The ratio of functional to total prothrombin was significantly lower in infants with diarrhea, the mean $\pm$ SD values being 0.65 $\pm$ 0.41 vs. 1.1 $\pm$ 0.26. This difference was highly statistically significant ($p < 0.001$). Low ratio was observed in 57.3% (43/75) of infants with diarrhea [56].

doi: 10.1588/medver.2004.01.00017

The liver is important for the synthesis of coagulation factors, and bile is required for the absorption of lipid soluble vitamins such as vitamin K. Liver damage and cholestatic liver disease have been found to cause vitamin K deficiency in infants and adults [47]. Hanawa et al. evaluated fifty-seven infants from 2 weeks to 4 months of age and discovered that the infants had experienced bleeding episodes due to vitamin K deficiency. The main causes of vitamin K deficiency were hepatobiliary lesions, chronic diarrhea, and long-term antibiotic therapy [48].

The serum liver enzyme levels were elevated at the time of Lucas' admission to both hospitals on August 27, suggesting liver damage. Synthesis of coagulation factors occurs in the liver, and liver damage can cause bleeding problems; this should have been considered in Lucas' differential diagnosis.

Furthermore, Payne and Hasegawa evaluated a 4-week-old, breastfed female infant who appeared healthy until signs and symptoms of CNS deterioration suddenly occurred. At presentation the infant was found to have a left-side parietal intracerebral hematoma, markedly prolonged PT and PTT, normal platelet count, and jaundice with a total and direct serum bilirubin level of 5.4 mg/dL and 2.6 mg/dL, respectively. Vitamin K1 and fresh frozen plasma returned the PT and PTT to normal within 18 hours, suggesting that the infant had severe vitamin K deficiency complicated by intracerebral hemorrhage [47].

To summarize, the diagnosis of vitamin K deficiency is suspected on the basis of its symptoms and signs, and a history suggesting the possibility. It is confirmed when the PT and PTT are prolonged [40]. Vitamin K deficiency may occur in both acutely ill and healthy-appearing infants. The physician must remain alert to the possibility of vitamin K deficiency in a wide variety of clinical situations. Material drug ingestion, failure to administer vitamin K1 at birth, the use of broad-spectrum antibiotic therapy by mother and/or infants, birth asphyxia, feedings limited to breast milk, and cholestatic liver disease are some of the causes that lead to vitamin K deficiency in infants [47].

**III-B. The State's allegations against Lucas' father are false**

On day six of Lucas' hospitalization it was determined that the baby was not having any spontaneous breaths. Brain death protocol was initiated and followed. Lucas was pronounced dead at 1200 on September 2, 2002. An autopsy was performed on September 4, 2002 by the medical examiner (case # C-02-581). He stated, "After review of the clinical history and a complete autopsy, it is determined that the cause of death of this 3-month-old male is blunt force trauma to the head and the manner of death is homicide [3, 36]."

The Medical Examiner's conclusions are unsupported by the clinical data related to this case as well as by scientific facts. Furthermore, his assertions are contradicted by his own findings. The following is a list of some inadequacies concerning the Medical Examiner's methods of investigation and his conclusions regarding the causes of injuries and death:

1. The Medical Examiner stated that Lucas' cardiac arrest and bleeding were caused by blunt force trauma to the head. However, he failed to provide any evidence that the baby suffered from trauma. Several physicians examined Lucas on August 27 and no evidence of trauma was found in the head region or on any other part of his body. Additionally, CT scans of the head region taken on August 27 did not show any evidence of trauma or bone fracture in the head region.

2. The Medical Examiner overlooked the well-established biomarkers of vitamin K deficiency observed in this case. Lucas exhibited high levels of the PIVKA-II protein which is a sensitive marker for vitamin K deficiency. In addition, prothrombin time (PT) and partial thromboplastin time (PTT) were elevated on August 27 and the treatment of the baby with vitamin K reduced PT and PTT by 20% and 25%, respectively.

3. The clinical data presented in this report show that Lucas suffered from diabetes, metabolic acidosis, hypokalemia, liver damage, urinary tract bacterial infection, pneumonia, and vitamin K deficiency, which are known to cause bleeding and death in children. However, it should be noted that the Medical Examiner did not investigate the contribution of these illnesses to the causes of bleeding in the tissues or to the death in this case.

4. The Medical Examiner stated in his autopsy report that the occurrence of chronic bleeding in the subdural space cannot be excluded with certainty as shown by the CT scan of the head on August 27. The blood products were of various ages, meaning that bleeding started several days to several weeks prior to August 27. However, the Medical Examiner failed to examine H & E stained tissue sections of the subdural hematoma and the meninges microscopically to evaluate the structure and determine the age of the bleeding.

5. Lucas' medical chart shows that he suffered from a bacterial urinary tract infection on August 28. However, the Medical Examiner did not present any description for the urinary tract in his autopsy report, nor did he mention that the baby suffered from a bacterial urinary tract infection.

6. The Medical Examiner stated that Lucas suffered from diffuse axonal injury, but neither provided the description of this injury nor the method used to detect it. In addition, he claimed that diffuse axonal injury was caused in this case by blunt trauma to the head. Axonal injuries indistinguishable from those observed in cases of head trauma have been described in cases of edema, hypoxia, hypoglycemia, cardiac arrest, and other causes. Lucas suffered from brain edema, hypoxia, and cardiac arrest. However, the Medical Examiner did not perform a differential diagnosis.

7. The Medical Examiner did not evaluate adverse reactions to hospital medications for their contributions to the causes of bleeding and death. Lucas was treated with excessive doses of sodium bicarbonate that caused severe edema in the brain and lungs, hypoxia, and hypokalemia. Lucas was also treated with epinephrine, which contributed to and exacerbated the bleeding in his tissues.

8. The Medical Examiner neglected to evaluate adverse reactions to vaccines for contributing to the causes of bleeding and death in Lucas' case. Lucas developed an upper respiratory tract infection within 1 to 2 days post-vaccination. Serious systemic injuries and death have been reported in babies who have received vaccines.

The travesty of falsely accusing and arresting people for killing children based on the erroneous SBS theory is not limited to people caring for babies. It also happens to people taking care of older children as described in Section IV below.

doi: 10.1588/medver.2004.01.00017

## IV. Alexa; vaccine and heparin induced pancreatitis and bleeding

Kathleen Butcher is a 40-year-old white woman, the mother of five children. She was arrested for killing Alexa Marie Shearer by vigorous shaking of the head and by blunt trauma to the head and abdomen. Alexa was a 15-month-old toddler who suffered from cardiac arrest and apnea on November 16, 1999 while at Kathleen's home in Howard County, Maryland. Kathleen had been Alexa's daycare provider since Alexa was two months old.

Kathleen was arrested in December of 1999 based upon a verbal communication between the Chief Medical Examiner for the District of Columbia and the Howard County Police. The Chief Medical Examiner performed Alexa's autopsy on November 19, 1999. He told the police officer present at the autopsy that Alexa's injuries and death were caused by blunt trauma to the head and that the manner of death was homicide. In February of 2001, Kathleen was convicted of involuntary manslaughter and child abuse in Alexa's death. She was sentenced to 10 years and 5 years, respectively, to be served concurrently (Criminal Case No. 13-K-99-38775). Kathleen has repeatedly stated that she cared for Alexa as she would her own child and that she never harmed her [4, 57].

My investigation revealed that Alexa died as a result of adverse reactions to vaccines that were given to her when she was sick. She developed pancreatitis and vitamin K deficiency. The medical examiner and other State's expert witnesses conducted an incomplete investigation in this case. They overlooked important clinical evidence that showed Alexa had been sick prior to her cardiac arrest on November 16, 1999. Below is the description of events that led to Alexa's cardiac arrest.

### IV-A. The factual causes of Alexa's cardiac arrest and bleeding

Alexa was born near term on August 11, 1998 and was delivered by cesarean section. She suffered from jaundice and a bacterial upper respiratory tract infection during the first week of her life. Her blood bilirubin level was 16.5 mg/dL at five days following birth – about 8 times the expected normal level of 2 mg/dL. Neurological damage has been observed in infants who have blood bilirubin levels > 12 mg/dL [4].

Alexa's appetite became poor at approximately 10 months of age and her appetite worsened gradually up until the time of her death at 15 months. For example, on July 20, Alexa's mother told her pediatrician that Alexa had a poor appetite for the last 2-3 weeks. She developed white thrush on her tongue and was treated with three consecutive courses of nystatin (anti-fungal) orally which caused vomiting and diarrhea. However, Alexa's physician overlooked her chronic health problems and proceeded to vaccinate her with the polio (IPV) and hepatitis B (Hep B) vaccines on July 20, 1999 at 11 months of age [4].

Alexa was again vaccinated on August 13, 1999 with four attenuated live-virus vaccines (measles, mumps and rubella (MMR) and varicella) when she was suffering from chronic immune depression, fungal infection, poor appetite, and poor weight gain. She also had frequent bowel movements and vomited on many occasions. In addition, she received the MMR vaccines three months earlier than the recommended age in a healthy child (15 months of age).

After receiving these vaccines, Alexa developed an upper respiratory tract infection and low-grade fever, and her poor weight-gain became worse. At two months of age Alexa was in the 50th percentile for weight on the growth chart. At 15 months of age her weight dropped to below the 1st percentile. Her length also dropped from the 25th percentile at 7.4 months to the 10th percentile at 12 months of age.

Alexa suffered from cardiac arrest and apnea between 1230 and 1245 on November 16, 1999 at Kathleen Butcher's home. The clinical data described in this report clearly show that Alexa's cardiac arrest was triggered by acute pancreatitis and diabetes mellitus. It was not caused by violent shaking and blunt trauma as the State alleged. Alexa did not breathe properly for approximately 30 minutes following her cardiac arrest. Therefore, her brain suffered from severe ischemia and hypoxia which caused severe diffuse edema and nerve damage.

Alexa also suffered from vitamin K deficiency, anemia, acute bacterial infections, oesteomyelitis, otitis media, and mastoiditis. In addition, the complications of acute pancreatitis and diabetes caused hypovolemia, metabolic acidosis, reduction of potassium levels in cardiac muscles and nervous tissues, edema, bleeding, and disseminated intravascular coagulation (DIC). Vitamin K deficiency caused bleeding, and affected calcium metabolism in the bone [4].

Furthermore, Alexa's treatment with high therapeutic doses of epinephrine during resuscitation along with the administration of epinephrine and heparin during her hospitalization caused bleeding in the subdural space, retina, skin, and other locations. She was also treated with excessive amounts of sodium bicarbonate that resulted in brain edema, hypoxia, and hypokalemia. Alexa's treatment with high therapeutic doses of epinephrine, dopamine, fresh frozen plasma, albumin, and fluid influenced the intravascular osmotic and hydrostatic pressure that caused the leakage of the fluid outside the blood vessels, thereby contributing to the formation of edema.

As noted, Alexa was vaccinated with four attenuated live virus vaccines (measles, mumps and rubella [MMR] and varicella) on August 13, 1999 when she was suffering from serious, chronic health problems. These vaccines caused the following serious illnesses that led to Alexa's cardiac arrest and apnea on November 16, 1999 [4].

1. MMR and varicella vaccines induced an upper respiratory tract infection that increased Alexa's susceptibility to develop a bacterial ear infection and osteomyelitis. Viral respiratory tract infections caused edema of the eustachian tube mucosa and blocked the tube, which led to the accumulation of the fluid in the middle ear and mastoid cavities, providing a culture medium for the bacteria present. Streptococcus pneumonia and Haemophilus influenza are the primary causes of bacterial ear infection in children and these bacteria also cause osteomyelitis in children. It is very likely that these bacteria caused Alexa's otitis, mastoiditis, and osteomyelitis of the T-10 vertebrae.

2. Alexa's viral and bacterial infections, poor appetite, weight loss, anemia and vitamin K deficiency led to significant immune depression, especially in T-cell counts and functions. This made Alexa's response to the MMR and varicella vaccines inadequate and increased her risk for developing infections and

doi: 10.1588/medver.2004.01.00017

having a serious adverse reaction to the vaccines. The mumps virus from the vaccine probably overcame Alexa's weakened immune function and infected the pancreatic tissues. The clinical tests and the pathological findings in the abdominal cavity indicated that Alexa suffered from acute pancreatitis which subsequently led to her cardiac arrest and apnea on November 16, 1999.

**IV-B. The allegations against Kathleen Butcher are false**

I reviewed the Chief Medical Examiner's autopsy report and his court testimony concerning this case and found that his autopsy and investigation of this case were incomplete [4, 58]. He misinterpreted vital clinical data including the results of his own tests. He then proceeded to present flawed conclusions to the police and the court with regard to the causes of injuries and death. Below is a list of observations that delineates numerous flaws contained within Dr. Arden's investigation:

1. The Chief Medical Examiner neglected to review Alexa's medical records prior to her cardiac arrest to find out if she had pre-existing health problem(s) that may have contributed to her cardiac arrest on November 16, 1999. My investigation revealed that Alexa suffered from chronic health problems and that her illnesses increased her risk to develop an adverse reaction to vaccines.

2. The Chief Medical Examiner did not assess the adverse reactions of medications given to Alexa prior to her cardiac arrest on November 16. She was treated with Nystatin (antifungal) for six weeks which caused diarrhea and vomiting and subsequently contributed to her poor weight gain, immune depression, and vitamin K deficiency.

3. The Chief Medical Examiner failed to evaluate adverse reactions to the vaccines given to Alexa; he did not consider their contributions to her injuries and death. My investigation revealed a direct link.

4. The Chief Medical Examiner did not review Alexa's medical records during her hospitalization from November 16 through 18. He overlooked the biomarkers of acute pancreatitis, bacterial infections, diabetes, vitamin K deficiency, and anemia described in this report. He missed the opportunity to see the progression of Alexa's symptoms and lesions. By not reviewing this critical information, he neglected to consider the fact that Alexa did not have any sign of trauma when the rescue team picked her up from Kathleen's house around 1300 on November 16.

5. The Chief Medical Examiner failed to address the biomarkers, lesions, and symptoms of acute pancreatitis in Alexa's case which included elevation of serum amylase and lipase, hyperglycemia, bloody intraperitoneal fluid, induration of root of mesentery with inflammatory process and fibrin exudates, severe inflammation in the area of the infrahepatic vena cava and the upper portion of the right kidney, hematoma of the right upper omentum, coagulopathy, hypotension, and edema.

6. The Chief Medical Examiner overlooked the fact that Alexa's prothrombin time (PT) and partial thromboplastin time (PTT) levels were elevated on November 16 because she was suffering from vitamin K deficiency. Her PT and PTT were 33.3 seconds and >100 seconds, respectively. Alexa's PT and PTT levels were reduced by more than two fold by November 18 because she was treated with fresh frozen plasma (FFP). FFP is efficacious for treatment of factors II, V, VII, IX, X, and XI deficiency.

7. The Chief Medical Examiner did not evaluate the adverse reactions of medications given to Alexa during her hospitalization from November 16 through November 18. She was treated with high doses of epinephrine and heparin, which caused bleeding. She was also given excessive amounts of sodium bicarbonate which caused hypoxia, brain edema, and hypokalemia.

8. The Chief Medical Examiner examined sections of the dural membranes and the skin from Alexa's back microscopically and found that the bleeding was fresh and less than 24 hours old. His finding indicated that the bleeding in these tissues occurred between November 18 and 19. Alexa had been given 5000 IU of heparin (11.2 times the therapeutic dose) approximately 7 hours prior to autopsy and this treatment caused serious bleeding, yet the Chief Medical Examiner did not consider Alexa's treatment with heparin in his investigation.

9. Alexa suffered from otitis media and mastoiditis bilaterally. The physicians who treated Alexa in the hospital and the radiologist who read the CT scan mentioned the possibility that Alexa suffered from a chronic ear infection. Yet, the Chief Medical Examiner did not examine her ear at autopsy. Her blood test results taken on November 16 indicated that Alexa was suffering from an acute bacterial infection. Streptococcus pneumonia and Haemophilus influenza are the primary causes of bacterial ear infection in children. In addition to otitis media, S. pneumonia and H. influenza also cause osteomyelitis in children. Alexa had osteomyelitis of the T-10 vertebrae.

10. The Chief Medical Examiner did not examine the lytic lesion in Alexa's T-10 vertebrae microscopically nor was a bone scan ever performed to rule out osteomyelitis. He claimed that Kathleen broke this vertebrae by hyperextension. The Chief Medical Examiner's claim is not supported by medical facts, much less by his own findings. He found fresh bleeding in the tissues associated with T-10 and T-8 but not in T-9. If Kathleen had exerted tremendous pressure on the toddler's back to break her T-10 vertebrae, and if it caused bleeding in T-10 and T-8, then we would expect to see bleeding in the T-9 vertebrae as well. We would also expect to see a three-day old bleed and not a fresh bleed.

11. The Chief Medical Examiner did not investigate the cause(s) of left 8th rib fracture that had healed. Investigating this matter in a scientific manner may reveal the factual cause(s) of the rib fracture and may help to explain the causes of Alexa's cardiac arrest. Alexa suffered from vitamin K deficiency, and vitamin K is important for calcium metabolism in bone. Thus, vitamin K deficiency may also cause bone problems. The lesion in the rib observed in Alexa's case may represent a local bone defect caused by vitamin K deficiency and subsequent healing.

**Conclusions and Recommendations**

After evaluating the medical evidence presented in these cases, one can conclude that the theory behind "shaken baby syndrome" is false. The federal government has the responsibility to take immediate action by launching an investigation into the SBS diagnosis and theory, and I am hereby requesting that

doi: 10.1588/medver.2004.01.00017

they do so. The validity of this erroneous theory must urgently be questioned and re-evaluated in order to prevent other wrongful convictions.

Not only will this save innocent people from being falsely accused and incarcerated, but it will also save millions of dollars from being spent on unnecessary trials and legal fees. I further believe that the states of Florida, Maryland, and Pennsylvania have the obligation to review the medical evidence presented, in addition to immediately freeing Alan Yurko, Brian Herlihy, Alejandro Mendez, and Kathleen Butcher from prison.

The primary objective of health care providers and the State should be to find and then focus on the facts. It is the duty of the medical establishment to properly investigate the causes of injuries and death of children in cases such as these in order to prevent such tragedies from occurring again. Accusing innocent parents and caretakers of abusing and killing their children based upon unsupported theory, such as SBS, will not prevent the death of another child by vaccines and inappropriate medications. However, it certainly will lead to wrongful incarceration and unimaginable suffering—psychologically to parents/caretakers, and physically to those fragile, highly sensitive children it is our duty to protect.

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