Analysis of causes that led to Baby Lucas Alejandro Mullenax-Mendez' cardiac arrest and death in August-September, 2002

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Abstract

Lisa Mullenax and her husband Alejandro Mendez were accused of killing their 3½ month old Baby Lucas by blunt force trauma to the head (Shaken Baby Syndrome). The baby suffered from cardiac arrest and apnea on August 27, 2002 and his father immediately sought the assistance of a neighbor who contacted the Medical Emergency Service (MES) asking for help. The MES resuscitated the baby, treated him with epinephrine, and transported him to the Centre Community Hospital. Lucas stayed about one hour in this hospital and then was airlifted to the Geisinger Medical Center. Lucas was pronounced brain dead after six days following his arrival to the Geisinger Medical Center. In the hospitals, several physicians examined Baby Lucas and no evidence of traumatic injuries to the head was observed. In addition, Lucas' head region was examined by CT scans on August 27 and no bone fracture was found. Lisa and Alejandro were accused of killing their Baby Lucas based only on the autopsy findings of an old-healed rib fracture and bleeding in the retina of the eyes, brain, and the subdural space.

Lisa and Alejandro requested that I evaluate their case to find the factual cause(s) that led to Lucas' cardiac arrest and death. I evaluated their case by reviewing the baby's medical records, case history, the autopsy report, Lisa's medical record during her pregnancy with Lucas, and the published medical literature pertinent to Lucas' case. I used differential diagnosis to evaluate the contribution of causes and the synergistic actions among these causes that led to the baby's cardiac arrest, apnea, bleeding in the brain and other locations, and death.

I present my review and analysis of Lisa's medical records during her pregnancy with Lucas in Section I of this report. Section II contains a detailed description of Baby Lucas' treatment history and his health problems from the time of birth on May 16, 2002 to the day of his cardiac arrest on August 27, 2002 along with my analysis of those events. In Sections III and IV, I describe the clinical events that took place during Lucas' seven days in the hospitals and my analysis of those events. My analysis of the medical examiner's autopsy report is presented in Section V. Section VI contains my conclusions and recommendations.

Keywords: vaccine-related death; shaken baby syndrome (SBS), adverse vaccine reactions, homicide

Introduction

Baby Lucas was born at 41 weeks of gestation on May 16, 2002. He was in excellent health until the day of his vaccination on July 23 when he was 9 weeks of age. He was simultaneously administered seven vaccines (DTaP, Hepatitis B, Hib, IPV, and Pneumoccocal vaccine) and developed an upper respiratory tract infection within one to two days post-vaccination. He was treated with Tylenol for two to three days for fever. At seven days post-vaccination, Lucas' mother took him to his pediatrician because he was still suffering from an upper respiratory tract infection. Also, one day prior to Lucas' vaccination, Lucas' mother suffered from mastitis and she was treated with a 10-day course of Dicloxacillin. She breast-fed Lucas during her treatment with an antibiotic and he developed diarrhea. Furthermore, Lisa was also treated with an eleven-day course of an antibiotic on May 20, when Lucas was four days old and she also breast-fed him during her treatment (Table 1).

The clinical data collected during Lucas' hospitalization following his cardiac arrest on August 27 revealed that he suffered from serious health problems that were responsible for his cardiac arrest and the bleeding in the brain, subdura, retina, and other locations. These problems included diabetes mellitus; metabolic acidosis, liver damage, urinary tract bacterial infection, pneumonia, vitamin K deficiency, anemia, and brain edema. Lucas' health problems were induced as a result of his seven vaccines received on July 23 (Table 3) and the treatment of his mother's upper respiratory tract infection and mastitis with antibiotics during Lucas' breast-feeding period (Table 1).

The vaccines given to Lucas on July 23, 2002 induced an upper respiratory tract infection within 1 to 2 days post-vaccination and I believe that this infection also caused Lucas' urinary tract bacterial infection observed on August 28 and his pneumonia discovered at autopsy. Lucas' systemic infections caused hyperglycemia and metabolic acidosis that subsequently led to the reduction of the levels of potassium in the cardiac muscle and nervous tissues and that led to cardiac arrest. Serious adverse reactions to vaccines and death in children have also been reported in the medical literature. For example, reports to the Vaccine Adverse Event Reporting System (VAERS) in the US 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 (Section II-B).

The treatment of Lucas' mother with antibiotics predisposed Lucas to vitamin K deficiency by reducing the levels of vitamin K in her breast-milk, causing Lucas' diarrhea, and reducing vitamin K synthesis in Lucas' gastrointestinal tract (GIT), and

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vitamin K uptake from the GIT. Lucas also suffered from liver damage and other systemic problems that reduced the synthesis of coagulation factors in liver and reduced food intake. Moreover, Lucas was almost exclusively breast-fed during his life and human milk has low concentrations of vitamin K. Vitamin K deficiency was the primary cause of bleeding in the brain and other tissues in this case. I presented the clinical evidence that show Lucas suffered from vitamin K deficiency and vitamin K deficiency is the common and well-documented cause of bleeding in breast-fed infants in Section IV of this report.

Furthermore, the treatment of Lucas with epinephrine in the hospital on August 27 and thereafter also contributed to the subdural bleeding and bleeding in other locations as shown by several brain CT scans taken on August 27 through August 30. In addition, on August 28, the blood pH reached a critical low of 6.64 and the baby was treated with sodium bicarbonate. Unfortunately, he was treated with excessive amounts of sodium bicarbonate and the blood pH reached a critical high of 7.67 (Table 4). This treatment caused brain and pulmonary edema, hypoxia, and hypokalemia.

The radiology findings show that Lucas had an old-healed fracture of rib #11. Rib fractures have also been observed to occur during labor in babies as explained in this report (Section V). Lucas was born by vaginal delivery at 41 weeks of gestation with manual assistance and the force used caused his mother to suffer from vaginal laceration, severe bleeding, hypotension, and anemia that required a blood transfusion. It is likely that Lucas' rib fracture happened during labor.

The Medical Examiner performed an autopsy on Lucas on September 4, 2002 (case # C-02-581) and the main objective of this autopsy was to establish the factual causes of injuries and death in this case. 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." I find that the Medical Examiner's conclusions are unsupported by the clinical data related to this case, which are described in this report. I present my arguments against the Medical Examiner's methods of investigating this case and his conclusions with the supporting medical evidence in Section V of this report. The following is a list of some of the problems concerning the Medical Examiner's methods of investigation and his conclusions of the causes of injuries and death in this case:

1. The Medical Examiner stated that Lucas' cardiac arrest and bleeding were caused by blunt force trauma to the head. However, he did not provide any evidence that the baby suffered from trauma. In addition, several physicians examined Lucas on August 27 and no evidence of trauma was found in the head region or any part of his body. Also, the CT scans of the head region that were taken on August 27 did not show any evidence of trauma or bone fracture in the head region.
2. The Medical Examiner presented a list of lesions in his autopsy report without providing the gross and microscopic descriptions for these lesions (V).
3. The clinical data presented in this report showed 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, the Medical Examiner did not investigate the contribution of these illnesses to the causes of bleeding in tissues and death in this case.
4. The Medical Examiner overlooked the well-established biomarkers of vitamin K deficiency observed in this case. Lucas had a high level of PIVKA-II protein which is a sensitive marker for vitamin K deficiency. In addition, prothrombin time (PT) and activated partial thromboplastin time (APTT) were elevated on August 27 and the treatment of the baby with vitamin K reduced PT and APTT by 20% and 25%, respectively (Table 9).
5. 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. That means that bleeding started several days to several weeks prior to August 27. However, the Medical Examiner did not examine H & E stained tissue sections of the subdural hematoma and the meninges microscopically to evaluate the structure and the age of the bleeding.
6. Lucas' medical chart shows that Lucas suffered from a urinary tract bacterial 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 urinary tract infection.
7. The Medical Examiner stated that Lucas suffered from diffuse axonal injury but he did not provide the description of this injury or the method that he used to detect this injury. In addition, he claimed that diffuse axonal injury in this case was caused by blunt trauma to the head. I described several studies in this report that show axonal injuries indistinguishable from those observed in cases of head trauma that were described in cases of edema, hypoxia, hypoglycemia, cardiac arrest, and other causes (V-E). In this case the baby suffered from brain edema, hypoxia, and cardiac arrest. However, the Medical Examiner did not perform a differential diagnosis in this case.
8. The Medical Examiner did not evaluate the contribution of the adverse reactions of medications given to Lucas in the hospital 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 the bleeding in his tissues.
9. The Medical Examiner did not evaluate the contribution of the adverse reactions of vaccines given to Lucas to the causes of bleeding and death in Lucas' case. Lucas developed an upper respiratory tract infection within 1-2 days post-vaccination. Serious systemic injuries and death have been reported in babies who have received vaccines (II-B).
10. The Medical Examiner assumed that the old-healed fracture of rib #11 observed in Lucas case was resulted from child abuse without performing a review of the medical literature to find out if rib fractures had been reported to occur during labor in infants. I presented several studies that show rib fractures occurred during labor (V-G).

The Mullenax and Mendez family have suffered from two tragedies because the physicians who treated Lucas with vaccines and those who treated his mother with antibiotics during

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the breast-feeding period did not take into consideration the adverse reactions of those agents on Lucas' health. In addition, the physicians who treated Lucas during his hospitalization following his cardiac arrest and the medical examiner in charge of this case did not consider the adverse reactions of medications and vaccines given to Lucas and the adverse reaction of antibiotics given to Lucas' mother in their investigation. The first tragedy is the loss of Baby Lucas due to adverse reactions to vaccines and medications. The Mullenax and Mendez family's second tragedy is the false allegation, accusing Lucas' parents of killing their Baby Lucas, which is a horrible crime that they did not commit.

I urge the doctors who are involved in this case, health care workers, and officials in the state of Pennsylvania to review the medical evidence presented in this report. It clearly shows that Lucas died as a result of the adverse reactions to vaccines and medications and Lucas' parents are innocent. Actions should be taken to prevent similar tragedies from occurring again. The objective of the state and health care workers should be to determine the factual causes that lead to the illness and death of a child and to prevent such problems from happening to other children. Accusing innocent parents of abusing and killing their children based on unsupported theory, as occurred in this case, will not prevent the death of other children from the adverse reactions to vaccines and medications. However, it certainly imprisons innocent people and causes great suffering. It also costs the taxpayers huge sums of money in order to pay for unnecessary trials and legal fees while destroying the lives of innocent parents and caretakers.

I believe that the following recommendations will help prevent future infant deaths from occurring as a result of the adverse reactions to vaccines and medications. Furthermore, they may prevent innocent people from being wrongly incarcerated.

1. Babies who show adverse reactions to vaccines should be monitored closely. Their blood should be analyzed to check for the levels of pH, gases, glucose, potassium, vitamin K, and coagulation factors.
2. Breast-fed babies should be given 1 mg of vitamin K monthly to prevent bleeding in the brain and other locations. An ill, breast-fed baby who has feeding problem and vomiting may require higher doses of vitamin K.
3. Mothers receiving antibiotics should avoid breast-feeding their babies during the course of the treatment if possible.
4. Babies who are admitted to the hospital with bleeding should be checked for vitamin K deficiency, liver damage, and should be given vitamin K supplementation.
5. The use of sodium bicarbonate in the treatment of acidosis should be avoided if possible. Children who are treated with sodium bicarbonate should be monitored closely. In addition, the standard recommendations regarding the use of bicarbonate to treat acidosis should be followed in order to prevent the excessive use of bicarbonate and the development of brain edema.
6. In cases similar to Lucas' case, medical examiners and physicians should review the medical evidence and perform differential diagnosis prior to giving their conclusions that a child died as a result of a blunt force trauma to the head (Shaken Baby Syndrome).

Section I. Review of Lisa Mullenax's Medical Records During Her Pregnancy With Lucas

Lisa Mullenax is a white female schoolteacher. She was 27 years old (date of birth: September 25, 1974) when her son, Lucas was born on May 16, 2002. Lucas was her first child.

Her pregnancy was confirmed in September of 2001. During her pregnancy she suffered from nausea, continual painful sciatic nerve condition, and hypothyroidism. The sciatic pain prevented her from walking for at least one week. Furthermore, in December of 2001, she developed a tooth infection and she was treated with a course of penicillin [1].

She also suffered from a bacterial upper respiratory tract infection at one week prior to giving birth to Lucas and this infection continued after labor. She was treated with a course of Cephalexin at day four following giving birth (Table 1). This antibiotic inhibits the growth of bacteria that synthesize vitamin K in the intestinal tract which leads to the reduction of vitamin K levels in milk. It is also excreted in milk and inhibits bacterial growth in the intestinal tract of the baby and reduces vitamin K synthesis. It also causes diarrhea in infants and thereby reduces vitamin K uptake from the intestinal tract as described in Section IV of this report.

On May 16 at about 1400, Lisa felt the leakage of the amniotic fluid accompanied by severe pain. At about 1700, she was treated with Demerol into the spine to reduce her pain but its effects started to wear off at about 2000. Lucas was born at 2137 by vaginal delivery with manual assistance. Lisa remembered at one point that she was told to stop pushing because the baby needed to be repositioned. Her mid-wife moved the baby's head around so that he would come out right. Lisa was in a lot of pain and fainted immediately after giving birth. Her blood pressure dropped and she severely hemorrhaged as a result of vaginal laceration. At the time of delivery on first presentation she had a relatively normal hematological profile with a white count of 10,400/mL, hemoglobin 12.5 g/dL, and platelets 136,000/mL. Following delivery her hemoglobin fell to a low of 6.6 g/dL with a hematocrit of 18.9%. There was also a drop in her platelet count to as low as 108,000/mL. She was given a blood transfusion and treated with iron and vitamin B [1].

Furthermore, on May 18 she had severe pain when attempting to urinate. Her husband discovered that part of the placenta still had not been removed and a nurse and then a physician proceeded to manually remove it. However, on May 20, 2002, an ultrasound exam of the abdomen was obtained and it showed a retained placenta. Her doctor removed the rest of placenta that day. The vagina and vulva were also inspected and two small first-degree bilateral vaginal lacerations were discovered [1].

Lisa left the hospital with Lucas on May 21 and on that day she exclusively breast-fed him even though she was taking Cephalexin to treat her upper respiratory tract infection (Table 1). On July 22, Lisa came down with mastitis. Her breast was sore and her temperature was 101.2°F (38.4°). Her mid-wife gave her Dicloxacillin (Penicillin) to treat the infection without giving her instructions about the secretion of this antibiotic in her breast-milk and the impact it could have on the baby's health. Lisa continued to breast-feed Lucas as she did during her treatment with antibiotic two months earlier (Table 1). The use of these antibiotics by the mother during the breast-feeding period can lead to vitamin K deficiency in an infant. These an-

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tibiotics inhibit bacterial growth in the intestinal tracts of the mother and the breast-fed infant and thereby reduce vitamin K synthesis. These antibiotics were also excreted in milk and caused diarrhea in her breast-fed infant which led to reduction of vitamin K uptake from the intestinal tract. Furthermore, Lisa was also given Synthroid to treat her hypothyroidism which she began taking in mid-August.

Table 1. Lisa Mullenax's treatment with antibiotics during Lucas' breast-feeding period

Date	Antibiotic Type	Daily Dose (mg)	Duration (days)	Total Dose (mg)	Lucas' Age (days)
May 20-31	Cephalexin	2000	11	22,000	4-15
Aug. 22-Sept. 1	Dicloxacillin	2000	10	20,000	61-71

Section II. Review of Lucas Alejandro Mullenax-Mendez' Medical Records from the Time of Birth on May 16 to August 27, 2002, and Analysis of His Health Problems

II-A. Case history and health problems

Lucas was born on May 16, 2002 by vaginal delivery at 41 weeks of gestation and he was in perfect health. He was breast-fed and at his two-week check up, he was in the 95th percentile for his weight and height (Table 2). However, he developed an upper respiratory tract infection within 48 hours following his two-month vaccinations (Table 3). He also suffered from diarrhea, vomiting, and fatigue during the 34 days following his vaccination his mother' treatment of mastitis with an antibiotic (Table 1).

On July 23, at approximately 9 weeks of age, Lucas was administered seven vaccines simultaneously [2]. The vaccines included DTaP, Hib, Hepatitis B, IPV and pneumococcal vaccine. The compositions of these vaccines as reported in the Physicians' Desk Reference [3] are presented in Table 3. In addition, to various antigens, these vaccines contain formaldehyde and phenol as preservatives in addition to aluminum.

Lucas developed an upper respiratory tract infection at one to two days following vaccination. His parents noticed that he had a cough as well as clear mucus discharge from the nose. He was given Tylenol (Acetaminophen) at a daily dose of 320 mg for two to three days to reduce fever. Fever of 38ºC (100.4ºF) or higher has been reported in 15% to 25% of children in the first two days following pneumococcal vaccine [4] and other vaccines [3]. In addition to fever, Lucas slept a lot on the day of his vaccination and subsequent days. His parents specifically recall that he also vomited twice on July 29. Furthermore, Lucas' cough and nasal discharge continued through July 30 and his mother took him to his pediatrician. The doctor found that the baby was suffering from an upper respiratory tract infection and she recommended use of vaporizer and giving the baby an adequate amount of fluid.

Near the end of July, the baby's stool became liquid. His diarrhea continued until the day of his hospitalization on August 27. Lucas had immediate bowel movements that followed his nursing. His parents thought that this was a normal process and they did not seek medical assistance. One day prior to the baby's vaccination on July 23, his mother came down with mastitis and her mid-wife gave her Dicloxacillin to treat her bacterial infection (Table 1). This antibiotic excreted into her breast-milk and contributed to the cause of diarrhea by inhibiting the growth of normal intestinal bacteria and that enhanced the growth of chlostridium in the intestine [5]. In addition, in a study including 365 infants who were inoculated with Hib, diarrhea developed in 5.2% of the children at 48 hours post-inoculation [3, page 2318].

Furthermore, from August 17 to the time of Lucas' hospitalization on August 27, Lucas slept more than usual. He slept through the night (9:00-9:30 pm to 7:00-7:30 am). In addition, in the morning of August 26 Lucas vomited a lot after being fed. He also spit up quite a bit shortly after that. On the morning of August 27, Lisa had to wake up Lucas to breast-feed him before leaving for work. A few minutes after being breast-fed, Lucas vomited it all up again. His mother left home at 7:45 a.m. and came back at 11:15 a.m. Lucas was almost asleep when she arrived home and her husband informed her that Lucas did not want to take his milk from his sippy cup. She breast-fed him and he appeared very tired. Lisa left for her meeting at 12:50 pm. Lucas stopped breathing at about 1:30 p.m. and his father ran carrying him to the neighbor's house to get assistance.

Table 2. Baby Lucas' growth measurements

Date	Weight (Kg)	Length (cm)	Head Circum. (cm)	Age (days)
05/16/02	4.13	55.9	38	birth
05/23/02	4.15			7
05/30/02	4.48	58.4	38	14
07/11/02	6.29	61	41.2	55
07/30/02	6.89			74

Table 3. Composition of vaccines administered to Baby Lucas on 07/23 as described in the Physicians' Desk Reference

Vaccine Type	Compositions
DTaP	Each dose (0.5 mL) contains 0.625 mg aluminum; 25 Lf Diphtheria toxoid; 10 Lf tetanus toxoid; 25 mcg pertussis toxin (PT); 25 mcg filamentous hemagglutinin (FHA); 8 mcg pertacin; 2.5 mg 2-phenoxyethanol; 4.5 mg sodium chloride; and 0.1 mg formaldehyde.
Hepatitis B (Comvax)	Each dose (0.5 mL) contains 0.25 mg aluminum; 10 :g of hepatitis B antigen; 4.5 mg sodium chloride; 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 mcg of purified Haemophilus capsular polysaccharide.
Inactivated Polio Vaccine (IPV)	Each 0.5 mL dose contains 40 D antigen units of type 1, 8 D antigen units of type 2, and 32 D antigen units of type 3 poliovirus. Also present are 0.5% of 2-phenoxyethanol and 0.02% of formaldehyde (Preservatives), 5 ng neomycin, 200 ng streptomycin, and 25 ng polymyxin.
Pneumococcal vaccine (Prevnar)	Each dose (0.5 mL of vaccine) contains a mixture of purified polysaccharides of 23 most prevalent or invasive pneumococcal types of Streptococcus Pneumonia dissolved in isotonic saline solution containing 0.25% phenol as preservative.

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II-B. Adverse reactions to vaccines in children

Serious adverse reactions and death due to the vaccines given to Baby Lucas (Table 3) have been described in the medical literature. Below are brief descriptions of selective studies that describe the incidence of illnesses associated with vaccinations in children. Some of these studies are also described in the Physicians' Desk Reference [3]. However, neither Lisa nor her husband was informed by the medical staff of the possibility of the adverse reactions to vaccines prior to or after administering vaccines to Lucas.

1. 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 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 [6].
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 (38ºC) 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% [3, page 3063].
3. The whole-cell DTP vaccine has been associated with acute encephalopathy [3]. A large case-control study that included children 2 to 35 months of age who received DTP was conducted in England to study the incidence of vaccine related neurological problems. 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) was 3.3 ($p<0.001$) of a neurological illness occurring within 7-day period following receipt of DTP dose compared to children not receiving DTP vaccine in the 7-day period before onset of their illness.
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 (38.2ºC) (0.6%); irritability (12.6%); drowsiness (4.9%); diarrhea (5.2%); and vomiting (2.7%) [3, page 2318].
5. Two hundred and eleven 2 month old infants were vaccinated with IPV and DTaP and some of them developed systemic adverse reactions at 24 hours post-inoculation. These included fever > 102.2ºF (39.0ºC) (0.5%), irritability (24.6%), tiredness (31.8%), anorexia (8.1%), and vomiting (2.8%) [3, page 2335].
6. The adverse experiences that have been reported with pneumovax vaccines in clinical trials and post-marketing experience in children include: asthenia, malaise, fever >102ºF (38.9ºC), nausea, vomiting, lymphadenitis, serum sickness, arthragia, arthritis, myalgia, headache, paresthesia, rash, and urticaria [3, page 1862]
7. The Institute of Medicine (IOM) reviewed the scientific literature on the adverse reactions to vaccines in children in the early 1990s and found that the evidence favored acceptance of a causal relation between some vaccines and systemic illnesses. These causal relations included (1) diphtheria and tetanus toxoids vaccine and the development of Guillain-Barre Syndrome (GBS) and brachial neuritis, (2) oral polio vaccine and the development of GBS, and (3) unconjugated Haemophilus Influenza type b (Hib) vaccines and the susceptibility to Hib disease. The IOM also found the evidence that established causality between vaccines and certain illnesses, including (1) diphtheria and tetanus toxoids vaccine and the development of anaphylaxis, (2) oral polio vaccine and the development of poliomyelitis and death from polio vaccine-strain viral infection, and (3) hepatitis B vaccine and the development of anaphylaxis reaction [7].
8. The database from the 1994 National Health Interview Survey (NHIS) in the USA that included 6515 children less than six years of age who received 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 [8].

The above selected studies clearly show that serious health problems and even death can result from vaccinating infants and children. The parents or guardians of a child should be given the Vaccine Information Materials prior to immunization as required by the National Childhood Vaccine Injury Act of 1986. The Physicians' Desk Reference states that physicians should inform the parents or guardians about the potential for adverse reaction of pertussis-containing vaccines [3, page 3062]. However, when Lisa and Alejandro discussed the issue of vaccinating their baby with the pediatrician, they were never informed of the possibility that their baby could develop a serous adverse reaction to vaccines. In addition, the baby suffered from chronic diarrhea, vomiting, and fatigue during the 34 days following his vaccination and his mother's treatment with an antibiotic for mastitis.

On August 27, 2002, Lucas' father was taking care of Baby Lucas at home and he noticed that the baby stopped breathing at about 1:30 p.m. The father took the baby to the neighbor's house asking for help. The Medical Emergency Service (MES) was called and the baby was resuscitated and treated with epinephrine. The MES took the baby to Centre Community hospital for a short period and then airlifted him to the Geisinger Medical Center. The baby was pronounced brain dead after six days following his arrival at the Geisinger Medical Center.

Review of Lucas' medical records from both hospitals revealed that at the time of admission on August 27, Baby Lucas suffered from diabetes and complications of diabetes (metabolic acidosis, apnea, and cardiac arrest), respiratory acidosis, bacterial infections of the urinary tract, and liver damage. He also suffered from vitamin K deficiency that caused subdural bleeding and bleeding in the brain and other locations. These illnesses were induced by the vaccines given to Lucas on July 23, 2002 and by the antibiotic treatment of Lucas' mother (Table 1).

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Furthermore, the baby was treated with excessive amounts of sodium bicarbonate that caused metabolic alkalosis, hypoxia, cerebral and pulmonary edema, and hypokalemia. He was also treated with epinephrine that contributed to the bleeding in brain, spinal cord, and other locations. A detailed description of the hospital events and my analysis of these events are presented in Sections III and IV.

Section III. Review of Lucas Mullenax-Mendez' Medical Records During His Hospitalization on August 27 Through September 2, 2002

III-A. Treatment by the Emergency Medical Services and the Centre Community Hospital

On August 27, 2002, at approximately 1330, Baby Lucas was put down for a nap after being fed. His father found him unresponsive shortly thereafter. Emergency Medical Services (EMS) was called. Upon arrival the EMS found Lucas unresponsive with agonal respirations and mottled. Transport was begun. The infant was intubated with a 4.0 endotracheal tube and respiration was maintained by bag valve mask. The infant was placed on a monitor and was given 0.1 mg of epinephrine via an interosseous route. Just prior to arrival to the Centre Community Hospital, he was started on a Mannitol drip 7 gm in 50 cc of D5W (1 gm/Kg). This was infused over approximately one hour. Lucas' blood glucose level was found to be 382 mg/dL.

The child arrived at Centre Community Hospital (CCH) at about 1350 with a tachycardiac and a perfusing pulse but unresponsive [9]. Dr. Clifford J. Neal examined Baby Lucas upon arrival at CCH and found evidence of retinal hemorrhage bilaterally and the fontanel was full. However, he did not see evidence of ecchymotic lesions on skin that were observed in the Geisinger Medical Center an hour later. There was a profusable pulse and the child was in rhythm at 175 to 180 beats per minute. The abdomen was soft and there was no evidence of obvious mass. The baby had no neurologic activity at this point and there was no reaction to even painful stimulation. Baby Lucas was transferred from the Centre Community Hospital to Geisinger Medical Center by Life Flight at about 1430 on August 27, 2002.

III-B. Treatment at Geisinger Medical Center

A physical examination on admission at the Pediatric Intensive Care Unit at Geisinger Medical Center conducted by Dr. Jamian Ryan revealed a temperature of 35°C (95.0°F), heart rate of 94, blood pressure of 94/62 Hg, bulging anterior fontanel, nonreactive pupils, and pinpoint eyes. The gastrointestinal was soft, nontender, and nondistended and no bowel sounds were heard. In addition, ecchymosis on right eyelid (1-2 mm), below left eyelid (2 mm) and on the back (4 mm) and bloody endotracheal tube secretions were observed [10].

A blood analysis performed on August 27 at 1430 revealed low blood pH (7.22), low bicarbonate level (7 meq/L), high blood glucose level (382 mg/dL), low hematocrit (26%) and hemoglobin (8.9 g/dL) levels, elevated prothrombin time of 17.3 seconds and activated partial thromboplastin time of 38 seconds. In addition, on August 28, Lucas' PIVKA-11 level was 22.7 ng/mL (normal range 0.0-3.5 ng/ml). Blood glucose level increased to 415 mg/dL at 2200 on August 27 and it was reduced to normal levels on August 31 by IV infusion of N-saline (Table 4). Baby Lucas was given red blood cells (RBC) to correct his anemia and his hematocrit and hemoglobin values reached normal levels on August 30 (Table 5). On August 28th at 0315, the blood pH reached a critical low of 6.64 and the baby was treated with sodium bicarbonate. Unfortunately, Lucas was treated with an excessive amount of sodium bicarbonate and his blood pH reached a critical high of 7.67 (Table 6). This treatment caused brain and pulmonary edema, hypoxia, and hypokalemia [5].

Furthermore, Baby Lucas suffered from lactic acidosis, high Anion gap, and urinary tract bacterial infection (Tables 4 and 7). He also had liver damage as shown by elevated serum liver enzymes (Table 8). The baby was given 1 mg vitamin K per day on August 28 through August 30. This treatment reduced prothrombin time by 20% and partial thromboplastin time by 25% (Table 9). Lucas was also treated with potassium to correct his hypokalemia. In addition, the baby was treated with epinephrine, diuretic, and other agents. The list of medications given to Lucas on August 27 through September 2, 2002 is presented in Tables 10 and 11.

A computerized tomography (CT) scan of the brain taken on August 27 at 1806 showed an acute subdural hematoma along the tentorium, the posterior interhemispheric fissure and the interhemispheric fissure at the vertex. There was also a right frontotemporal parietal isodense subacute hematoma. The blood products were of various ages. The ventricles were non-dilated. No evidence of hydrocephalus or fractures was seen at this time. The CT scan of the brain taken on August 29 at 0816 showed an increase in blood in the interhemispheric fissure and extraparenchymal hemorrhage as compared with the scan of August 27. There were also multiple new focci of acute intraparenchymal and subdural hemorrhages. Cerebral edema and impending downward transtentorial herniation were also observed [10].

Furthermore, the CT scan taken on August 30 showed diffuse edema of the hemispheres bilaterally. The effacement of the sulci, basal cisterns and ventricles was increased as compared with the prior exam. There was also hyperdensity along the tentorium bilaterally consistent with blood, which is seen on the prior exam and remains unchanged.

Over the 24-48 hours from the time of admission, the CT scans revealed deterioration with increased edema. The fontanel became increasingly fuller. This led the doctors to make an incision into the baby's head and to insert a tube into the ventricles and to drain the excess fluid. On August 30, the patient's head was prepped and draped in the usual sterile fashion for surgery. A small incision was made in the right frontal region. The dura was lanced and a ventricular catheter was passed into the lateral ventricular system without difficulty. Bloody cerebrospinal fluid arose under mild pressure of approximately 15-20 cm of water [10].

The chest x-ray taken on August 27 at about 1400 showed no infiltrate in the lungs. However, an area of hazy infiltrate or edema was noted in the right perihilar region and right mid lung field in a second chest x-ray that was taken shortly after the first one. At 1620, the overall appearance of the chest was worsen-

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ing when compared to the previous films. The chest x-ray showed right upper and left lower lobe consolidation.

Furthermore, the chest x-ray taken on August 28 at 0243 showed more extensive opacification of most of the left lung since the previous examination on August 27, 2002. There was also more opacification of the right lung in this same interval. In addition, a chest x-ray taken on August 28 at 1058 showed evidence of increased opacity in the right upper lung field, which most likely represents patchy consolidation. The left lung base was hazy and may have been due to some atelectatic change. Evidence of complete atelectasis of the right upper lobe and also a right-sided pneumothorax was seen in the chest x-ray taken on August 28 at 1526. Diffuse bilateral pulmonary infiltrates were also present [10].

Chest x-rays taken on August 30 at 0750 and 1950 showed evidence that both lungs appeared diffusely opacified which is consistent with areas of consolidation or edema**.** In addition, the chest x-ray taken on September 1, 2002 at 0925 showed more diffuse abnormalities throughout the lung field than were present on the earlier study. The appearance would be compatible with pulmonary edema. Chest x-ray taken on August 27 at 1620 also showed a single left posterior non-displaced healing fracture of the 11th rib. The age of the fracture cannot be determined on this single projection. The CT scan of the abdomen and pelvis taken on August 27 at 1806 also showed a healing non-displaced rib fracture (callus formation surrounding this fracture) in the 11^{th} rib on the left posteriorly. The CT scan of cervical spine taken on August 27 at 1806 showed no evidence of fracture. In addition, an x-ray bone survey taken on August 28 at 2149 showed no evidence of a fracture or abnormalities in the skull, spine, pelvis and hips, long bones, hands and feet [10].

On day six of Lucas' hospitalization, it was determined that the baby was not breathing spontaneously. 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 and he determined that the cause of injury and death was blunt force trauma to the head [11]. However, the clinical events described above indicate that the cardiac arrest and the bleeding in the brain and other locations were caused by the adverse reactions to vaccines and medications. They caused Lucas' diabetes, metabolic acidosis, reduction of potassium levels in cardiac muscles and other tissues, vitamin K deficiency, and bacterial infections. My analysis of these events is presented in Section IV below.

Table 4. ACID-Base levels of Baby Lucas' Blood 8/27-9/02

Date & Time	Blood PH	Bicarb mEq/L	Lactic Acid mmol/L	Anion Gap mEq/L	K+ mmol/L	Glucose mg/dL
8/27/02@						
1431	**7.22 L**	**7 L**		**21 H**	**3.7**	**382 H**
1920					**4.3**	**415 CH**
2200			**6.5 CH**	**15.3 H**	4.4	**124 H**
2300	**7.25 L**	**17.7 L**				
8/28/02@						
0315	**6.64 CL**	**12.2 L**		**22.3 H**	**5.2 H**	
0355	**6.98 CL**	**13.3 L**		**23.5 H**	4.2	**182 H**
0445	**7.1 L**	17.9 L	**10.9 CH**	**17.5 H**	4.2	**182 H**
0600	**7.16 L**	18.6	**4.9 H**	**17.8 H**	4.5	**194 H**
0908	**7.03 CL**	21.2		**15.5 H**	**5.3 H**	
1100	**7.15 L**	20.4		13.1	4.8	111
1300	**7.27 L**	17.4 L	**6.4 CH**	**20.7 H**	4.5	94
1425	**7.26 L**	20.8		**19.4 H**	4.4	92
1840	**7.30 L**	22.5		**15.1**	4.3	109
8/29/02 @						
25			**5.8 CH**			
0350	7.44	**23.4 H**		15	3.5	110
0725	7.4	**25.3 H**	**5.5 CH**	14.8	3.8	117
1320	**7.50 H**	22.1		10.2	3.6	114
1600	**7.47 H**	**23.7 H**	**7.0 CH**	13.0	3.8	**121 H**
2005	7.38	21.3		12.6	4	93
8/30/02@						
0005	7.43	21.1	**6.1 CH**	19.3	3.9	86
0355	7.44	22.6		17.4	3.7	107
0830	7.44	**24.7 H**	**5.1 CH**	14.9	3.6	**124 H**
1145	**7.53 H**	**24.9 H**		14.3	**3.2 L**	**122 H**
1510	**7.50 H**	**26.5 H**	**5.2 CH**	13.2	**3.4 L**	**127 H**
2001	**7.62 CH**	**23.4 H**		14.1	**3.0 L**	**126 H**
2135	**7.61 CH**	**23.6 H**		13.7	**3.0 L**	**125 H**
2305	**7.63 CH**	22.7		14.0	**3.0 L**	116
8/31/02@						
0010	**7.57 H**	22.8	**4.0 H**	14.3	**3.0 L**	**123 H**
0410	**7.54 H**	**25.3 H**		12.6	**3.2 L**	120
0758	**7.63 CH**	21.4		12	**2.8 L**	**132 H**
0930	**7.67 CH**	**23.6 H**	**3.5 H**	9.1	**2.6 L**	104
1330	7.42	**H 25.5**		12.5	**3.2 L**	**136 H**
1600	7.406	**H 25.8**		14.5	3.6	**137 H**
2115	**H 7.58**	**H 24.3**		7.5	**3.1 L**	100
2330	**7.67 CH**	22.1		10.5	**2.7 L**	117
9/1/02@						
0020	**7.64 CH**	**24.2 H**		10.5	**2.7 L**	99
0500	**7.3 L**	**29.1 H**		6.1	**3.0 L**	81
0745	**7.26 L**	**30.6 H**		4.7	3.5	**64 L**
0935	**7.47 H**	**27.6 H**		5.7	**3.4 L**	85
1620	**7.53 H**	**25.1 H**		9.4	**3.1 L**	81
2130	**7.53 H**	**23.4 H**		11	**2.7 L**	89
9/2/02@						
0420	**7.51 H**	**23.6 H**		8.1	3.6	120
0800			1.7			
1000	7.45	22.9		8	3.9	127 H
Reference Range	**7.35-7.45**	**18-23**	**0.4-2.5**	**5-15**	**3.5-5**	**70-120**

L: Low value, H: High value; C: Critical value;

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Table 5. Lucas' hematology values on 08/27 – 09/02

Date & Time	RBC x10^6 /:L	HCT %	HGB g/dL	MCV fL	MCH pg	MCHC g/dL	RDW %	MPV fL
08/27@ 1920	3.23	**26 L**	**8.9 L**	79.3	27.2	34.4	13.3	9.9
2200		**26 L**	**9.0 L**					
08/28@ 0005		**20 L**	**6.9 L**					
0315		**19CL**	**6.3 L**					
0335		**18CL**	**6.2 L**					
0355		**23 L**	**7.7 L**					
0445		**29 L**	9.9					
0600		**29.5**	9.9	82.9	27.8	33.6	**15.4H**	9.8
0908		32	10.9					
1300		31	10.5					
1425		**29 L**	**9.8 L**					
1840		**27.1 L**	**9.3 L**	81.4	27.9	34.3	**14.8H**	9.8
08/29@ 0025		**28 L**	**9.6 L**					
0130		**26 L**	**8.9 L**					
0350		**25.6L**	**9.0 L**					
0725		**27 L**	**9.1 L**					
1600	**3.02 L**	24	**8.3 L**	80.1	28.5	35.5	**14.9H**	10
2005		34	11.5					
08/30@ 0005		36	12.2					
0355	4.31	37	12.7	83.1	29.2	35.2	**14.7H**	10
0830		39	13.3					
1145		**41 H**	**14.1**					
1510		**43 H**	**14.5**					
1553	**4.71 H**	38.9	**13.7H**	82.6	29.1	35.2	**14.7H**	10.1
2001		**42 H**	14.4					
2135		**42 H**	14.3					
2305		**41 H**	14.1					
08/31@ 0010		**45 H**	**15.4H**					
0410	**5.4 H**	**49 H**	**16.5H**	81.9	29.8	**36.4 H**	**14.7H**	10.3
0758		**39 L**	13.4					
0930		**28 L**	12.8					
1330		**44 H**	**14.8H**					
2330		35	11.8					
09/01 @ 0020		34	11.7					
0500	3.9	33.2	11.3	85.1	29	34	**15.2H**	10
0745		33	11.3					
0935		33	11.1					
1620		32	11					
2130		**28 L**	**9.6 L**					
09/02@ 0420		**28 L**	**9.5 L**					
0800	3.35	**29 L**	**9.7 L**	86.6	29	33.4	**15.4H**	11.1
1000		**29 L**	**9.9**					
Reference Range	**3.1-4.5**	**30.5-40.5**	**9.9-14.7**	**74-108**	**25-35**	**33-36**	**11.7-14.6**	**8.4-12**

L: Low value; H: High value; C: Critical value

Table 6. Blood gases of Baby Lucas from 08/27 – 09/02

Date & Time	Blood Ph	Bicarb mEq/L	CO_2 mmol/L	PCO_2 mm Hg	PO_2 mm Hg	Temp. ºC
08/27@ 1431	7.22	**7 L**	**14.5 CL**	**17**	567	34.8
1920	7.16 L	**13.3 L**		**38.3 L**		37
2200	7.19 L	**17.1 L**	**18.5 L**	45.8		37
08/28@ 0315	6.64 CL	**12.2 L**	**15.8 L**	**117 CH**	**18.6 L**	37
0355	6.98 L	**13.3 L**	**15.1 L**	**57.5 H**	**51.9 L**	37
0445	6.98 L	**17.9 L**	**19.9 L**	**57.5 H**	82.8	35.2
0600	**7.16 L**	18.6	**20.3 L**	**53.2 H**	**67.5 L**	36.2
0908	**7.03 L**	21.2	**23.7 L**	**82.3 CH**	**72.1 H**	37
1100	**7.15 L**	20.4	**22.2 L**	**60 H**	**271 H**	37
1300	**7.27 L**	**17.4 L**	**18.6 L**	38.9	**71 L**	37
1425	**7.26 L**	20.8	**22.2 L**	**47.1 H**	77.5	37
1840	**7.30 L**	22.5	**23.9 L**	46.9	**157 H**	
08/29@ 0025	**7.17 L**	21.7	**23.6 L**	**62.2 H**	**69 L**	37.7
0350	7.44	**23.4 H**	24.5	35.6	**191 H**	37
0725	7.4	**25.3 H**	26.6	41.4	**73 L**	37
1320	**7.50 H**	22.1	23 L	**28.9 L**	88	37
1600	**7.47 H**	**23.7 H**	24.7	**33.5 L**	**103 H**	37
2005	7.38	21.3	**22.4 L**	36.6	89.5	37
08/30@ 0005	7.43	21.1	**22.1 L**	**32.9 L**	98.4	37
0355	7.44	22.6	**23.6 L**	**34.0 L**	94.7	37
0830	7.44	**24.7 H**	25.8	37	97.6	37
1145	**7.53 H**	**24.9 H**	25.9	30.4 L	**104 H**	37
1510	**7.50 H**	**26.5 H**	27.5	2.3.5 L	**128 H**	37
2001	**7.62 CH**	**23.4 H**	24.2	34.4 L	89	36.6
2135	**7.61 CH**	**23.6 H**	24.3	**2.39 L**	**130 H**	37
2305	**7.63 CH**	22.7	23.4 L	**21.9 L**	90	36.2
08/31@ 0010	**7.57 H**	22.8	23.6 L	**25.8 L**	**356 H**	37
0410	**7.54 H**	**25.3 H**	26.2	**30.6 L**	**69 L**	37
0758	**7.63 CH**	21.4	22 L	**20.8 L**	**154 H**	37
0930	**7.67 CH**	**23.6 H**	24.3	**20.9 L**	**330 H**	37
1330	7.42	**H 25.5**	26.7	40.4	**14.3 L**	37
1600	7.406	**H 25.8**	27	42	78.7	37
2115	**7.58 H**	**H 24.3**	25	26 L	75.7	**35.9 L**
2330	**7.67 CH**	22.1	22.8 L	**19.1 CL**	**69 L**	**34 L**
09/01@ 0020	**7.64 CH**	**24.2 H**	25	**22.6 L**	**55 L**	**33.8 L**
0500	**7.3 L**	**29.1 H**	31	**60.8 H**	**90.7**	37
0745	**7.26 L**	**30.6 H**	32.7 H	**69.6 H**	88	37
0935	**7.47 H**	**27.6 H**	29	38.1	**60.3 L**	**34 L**
1620	**7.53 H**	**25.1 H**	26	**30.8 L**	**59.5 L**	37
2130	**7.53 H**	**23.4 H**	24.3	**28.9 L**	**67 L**	37
9/2/02@ 0420	7.51	**23.6 H**	24.6	**30.1 L**	**59.7 L**	37
1000	7.45	22.9	24	**34.0 L**	82.9	37
Reference Range	**7.35-7.45**	**18-23**	**24-32**	**35-45**	**75-100**	

L: Low value, H: High value; C: Critical value;

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Table 7. Lucas' urine analysis values

Date & Time	Measurements	Findings/Values	Ref Range
8/28/02 @ 2350	Clarity	Cloudy	Clear
	Ketone	15 mg/dL	negative
	Protein	30 mg/dL	negative
	Acetest	Moderate	negative
	Clinitest	250 mg/dL	negative
	Bacteria	Moderate	None
	WBC	5.0-9.0	
	RBC	20-29	
	Casts	1-4 Hyaline	
	Crystals	Many uric acid crystals	
9/2/02 @ 0430	Klebsiella oxytoca	100,000 colonies/mL	None

Table 8. Lucas' liver function values 08/27–09/02

Date & Time	Alkaline Phos. U/L	ALT U/L	AST U/L	Protein g/dL	Albumin g/dL	Billirubin Total mg/dL
8/28/2002						
0908	124	**75 H**	**166 H**	**4.9 L**	**3.4 L**	0.3
8/30/2002						
0908	97	**229 H**	**149 H**			
1310				6.1	3.5 L	1.4 H
2015	98	**202 H**	**112 H**	**5.8 L**	**3.3 L**	**1.7 H**
9/1/2002						
1620	57	57	**111 H**	**4.4 L**	**2.5 L**	0.4
Reference Range	**35-123**	**8.0-67**	**8.0-65**	**6.0-8.3**	**3.8-5.0**	**0.3-1.3**

Table 9. Lucas' clotting parameters 08/27 – 09/02

Date & Time	Prothrombin T Seconds	APTT Seconds
8/27/2002		
1900	**17.3 H**	**38 H**
2200		35
08/28/2002		
0205	**18.0 H**	**44 H**
0600	**19.4 H**	**45 H**
1300	**18.4 H**	**38 H**
1840	**19.7 H**	**39 H**
2205	**19.7 H**	**41 H**
08/29/2002		
0350	**18.0 H**	37
0725	**17.6 H**	**38 H**
1320	**17.5 H**	37
1600	**16.9 H**	**39 H**
2005	**15.9 H**	35
08/30/2002		
0145	**15.1 H**	33
0355	**14.6 H**	33
0830	**14.8 H**	32

Table 9. Lucas' clotting parameters 08/27 – 09/02 (Cont.)

Date & Time	Prothrombin T Seconds	APTT Seconds
8/31/2002		
0410	13.9	28
9/1/2002		
0500	**14.9 H**	32
1830	**16.4 H**	34
9/2/2002		
0015	**15.5 H**	28
0800	**15.2 H**	34
Reference Range	**11.5-14.3**	**21-37**

Table 10. List of Medications given to Lucas 08/27–08/28

Date & Time	Treatment	Dose	Actions
8/27/02@ 1400	Epinephrine intraosse-ously	0.1 mg	Stimulate cardiac muscle
1400	Saline, IV	50 cc/hr	To treat dehydration
1400	Mannitol	7 gm	Diuretic
1626	Tylenol	75 mg	To treat fever
2020	Fentanyl, IV	5 :g	To relief pain
2020	Versed	0.5 mg	For sedation
2020	DS 1/4 NS	25 cc/hr	To treat dehydration
8/28/02@ 0232	Vecuronium, IV	0.7 mg	Neuromuscular blocking agent
	Lasix, IV	7 mg	Diuretic
0300	RBC, IV	80 cc	To treat anemia
0304	Epinephrine	0.05 mg	Stimulate cardiac muscle
0311	Epinephrine	0.05 mg	Stimulate cardiac muscle
0318	Epinephrine	0.05 mg	Stimulate cardiac muscle
	Albumin 25% IV	25 cc	To teat anemia/edema
0330	Mannitol	6.25 gm	Diuretic
0330	Sodium bicarbonate, IV	5 mEq	To treat acidosis
	Epinephrine	5 :g/kg/min	Stimulate cardiac muscle
0410	Sodium bicarbonate, IV		To treat acidosis
0510	Morphine	0.7 mg	To relief pain
	Fentanyl, IV		To relief pain
	Epinephrine	0.05 mg/min	Stimulate cardiac muscle
0630	Vitamin K	1 mg/day	To stop bleeding
0844	Zantac, IV	7 mg	Reduce gastric secretion
1034	Fentanyl, IV	30 :g	To relief pain
1126	Sodium bicarbonate, IV	8 mEq	To treat acidosis
1155	Fentanyl, IV	10 ug	To relief pain
1225	Lasix, IV	7 mg	Diuretic
1340	Ativan, IV	0.8 mg/hr	Sedative
1454	Vitamin K	1 mg/day	To stop bleeding
1614	Versed	0.5 mg	Sedative
1620	RBC/IV	80 cc	For anemia
1620	DS 1/4NS	35 cc/hr	To treat dehydration

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Table 11. List of Medications given to Lucas August 29–September 9, 2002

Date & Time	Treatment	Dose	Actions
8/29/2002	Sodium bicarbonate, IV	8 mEq	To treat acidosis
0115	Vecuronium, IV	0.7 mg	Neuromuscular blocking agent
1232	Vecuronium, IV	0.8 mg	Neuromuscular blocking agent
1232	Ativan, IV	0.8 mg/hr	Sedative
1618	Albutrol	0.5 mg	Bronchodilator
1712	RBC	120 cc	To treat anemia
1712	Lasix, IV	8 mg	Diuretic
1925	Albutrol		Bronchodilator
8/30/02@ 1030	Lasix, IV	8 mg	Diuretic
1305	Morphine	0.7 mg	To relief pain
1510	Mannitol	5 g	Diuretic
8/31/02 at 0810	NS	80 cc	To treat dehydration
1425	Fluid, IV	28 cc/hr	To treat dehydration
2230	D5 1/5 Ns + 20 mEq/KCl/L	25 cc/hr	Hypokalemia
9/1/2002 at 0300	2 mEq/KCL	2 mEq/KCL/hr	Hypokalemia
1223	Vitamin K	1 mg/day	To stop bleeding
1600	D5 1/5 Ns + 20 mEq/KCl/L	25 cc/hr	Hypokalemia
1750	Albumin 25% IV	28 cc/hr	To teat anemia/edema
	Lasix, IV	7 mg	Diuretic
1930	Vasopressin, IV	1 Unit	Antidiuretic
2200	1/4 Ns + 20 mEq/KCl/L	30 cc/hr	Hypokalemia
9/2/02@ 0500	Vasopressin, IV	1 Unit	Antidiuretic
0515	70 cc NS	70 cc/hr	To treat dehydration
1315	70 cc NS	70 cc/hr	To treat dehydration

Section IV. Analysis of Clinical Events and Causes That Led to Bleeding in Lucas' Brain and other Locations

IV-A. Analysis of clinical events

On August 27, 2002, Baby Lucas suffered from cardiac arrest and apnea. The clinical data described in Section III shows that Lucas was suffering from diabetes mellitus, the 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. Baby Lucas' serum glucose levels on August 27 at 1431 and 1920 were 382 and 415 mg/dL, respectively (Table 4). Normal serum glucose range is 70-110 mg/dL. His blood pH was 7.22 at 1431 and dropped to 6.64 on August 28 at 0315 (Table 4). His serum potassium level was 5.2 mEq/L on August 28 and dropped to 2.6 mEq/L on August 31 (Table 4) 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 (Tables 10 and 11) to correct his hypokalemia.

Lucas' diabetes may have resulted from a bacterial infection of the pancreas and/or other organs. The clinical data show that Lucas was suffering from liver damage and a bacterial urinary tract infection (Tables 7 and 8). The levels of serum ALT and AST enzymes were 342% and 255% of normal, respectively. It has been stated that the metabolic decompensation of diabetes is due to a relative or absolute deficiency of insulin and a relative or absolute excess of glucagons [5]. Stress hyperglycemia, usually associated with infections and other life-threatening illnesses is due to the release of glucagons and catecholamines [5, page 2061]. Bacterial and mycotic infections complicate the life of the diabetic in whom hyperglycemia is poorly controlled. Multiple abnormalities in the host response to microbial invasion have been described in such patients. Leukocyte functions are thus compromised and immune response is blunted [5].

In metabolic acidosis resulting from diabetes, potassium usually leaves the intracellular environment because the intracellular proteins bind with hydrogen which leads to cardiac arrest and paralysis of the respiratory muscles. At this stage, serum potassium levels are usually normal or elevated, but after treatment with bicarbonate and the elevation of pH to normal or above normal, the potassium leaves the blood and goes back inside the cells. This leads to hypokalemia as we observed in Lucas' case. At the time of admission, Baby Lucas had no muscle tone and no intestinal movement as a result of low potassium levels in these tissues. *Harrison's Principles of Internal Medicine* states that in metabolic acidosis, initial serum potassium concentrations are normal to high, despite depletion of body stores, and potassium concentrations fall rapidly during therapy with sodium bicarbonate, predisposing the patient to cardiac arrhythmias and/or paralysis of the respiratory muscles [5, page 2060].

Furthermore, Lucas had metabolic acidosis, as indicated by low blood pH (6.64), low blood CO_2 level (14.5 mmol/L), low blood bicarbonate level (9.9 mEq/L), and a high anion gap (21 mEq/L). In diabetic patients, the metabolic acidosis and anion gap are almost totally accounted for by the elevated plasma levels of acetoacetate and beta-hydroxybutyrate, although other acids (e.g., lactate, free fatty acids, phosphates) contribute [5]. The levels of lactic acid in the blood were found to be critically high in Lucas' case due to diabetes and hypoxia (Table 4). Urine analysis also showed high levels of ketone bodies (Table 7).

Additionally, Baby Lucas also suffered from cerebral edema as a result of being diabetic. In diabetic children, cerebral edema is a common cause of death that occurs more frequently than in adults [5, 12, 13]. Baby Lucas had cerebral edema as shown in the CT scans and stated in the autopsy report [11]. At the time of admission, the brain edema was mild; nonetheless, his treatment with an excessive dose of sodium bicarbonate increased the severity of edema in the brain as shown by the CT scans. The CT scan of the brain taken on August 27 at 1806 showed that the ventricles were non-dilated and 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, the 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

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prior exam. The accumulation of the fluid in the brain led the doctors to operate and drain the excess cerebrospinal fluid on August 30, 2002.

The treatment with sodium bicarbonate and the excess fluid also 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 than were present on the earlier study. The appearance is compatible with pulmonary edema.

Baby Lucas was treated with sodium bicarbonate to correct acidosis. However, he was given excessive amount of bicarbonate. His blood pH was 7.22 on August 27 and it rose to 7.67 on August 31 (Table 4). *Harrison's Principles of Internal Medicine* states that bicarbonate therapy may be indicated in severely acidotic patients (pH 7.0 or below), especially if hypotension present (acidosis itself can cause vascular collapse). Bicarbonate is not used routinely in less acutely ill subjects because rapid alkalinization may have detrimental effects on oxygen therapy (5, page 2073).

Alkalinization increases the avidity of hemoglobin to bind oxygen, impairing the release of oxygen in peripheral tissues. The hemoglobin-oxygen dissociation curve is normal in diabetic ketoacidosis because of opposing effects of acidosis and deficiency of the red blood cell 2,3-bisphosphoglycerate (2,3-BPG). If acidosis is rapidly reversed, the deficiency of 2,3-BPG becomes manifest, increasing the avidity with which hemoglobin binds oxygen. 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 Baby Lucas' case and that his treatment with excessive amount of bicarbonate led to severe hypoxia and cerebral and pulmonary edema [14-16].

Furthermore, Baby Lucas suffered from hypoxia as a result of his severe anemia, as 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 a protein called a cute-phase reactant that becomes elevated with tissue inflammation or tissue destruction.

At the time of admission, Baby 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 became worse following the admission of the baby to the hospital as a result of his subsequent treatment with epinephrine. Epinephrine causes rapid rises in blood pressure and cerebral hemorrhage [3, page 675]. At the time of admission to Centre Community Hospital, Dr. Clifford J. Neal examined Baby Lucas 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 right eyelid (1-2 mm), below left eyelid (2 mm) and on the back (4 mm). In addition, bloody endotracheal tube secretions were observed. Furthermore, the CT scan of the brain taken on August 29 at 0816 showed an increase in blood in the interhemispheric fissure and extraparenchymal hemorrhage as compared with the scan of August 27. There were also multiple new focci of acute intraparenchymal, and subdural hemorrhages.

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 22.7 ng/mL (normal range 0.0-3.5 ng/ml)–this is a unique biomarker for vitamin K deficiency, (2) the prothrombin time (17.3 seconds) and the activated partial thromboplastin time (38 seconds) were elevated and the treatment of the baby with vitamin K reduced prothrombin time by 20% and PPT time by 25% (Table 9), (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 means that the bleeding had begun several days or weeks prior to August 27, 2002.

IV-B. Factors that led to vitamin K deficiency and bleeding in Lucas' case

Vitamin K is essential and has a coagulation activity. It is essential because the 1, 4 naphthoquinone nucleus cannot be synthesized by the body. However, bacteria in the intestinal tract synthesize vitamin K and can supply part of the vitamin K requirement. Vitamin K controls the formation of coagulation factors II (prothrombin), VII (proconvertin), IX (Christmas factor), and X (Stuart factor) in the liver. Other coagulation factors that depend on vitamin K are protein C, protein S, and protein Z. Furthermore, two bone matrix proteins necessary for normal bone metabolism are vitamin K-dependent. All of these vitamin K-dependent proteins contain the amino acid γ-carboxyglutamic acid and the carboxyl groups of the glutamic acid residues provide the vitamin-K-dependent proteins with characteristic calcium and phospholipid binding properties [17-18].

The clinical data described in Sections III and IV above indicate that the initial bleeding in the brain, eyes, and other locations in Lucas' case was caused by vitamin K deficiency.

In vitamin K deficiency, abnormal decarboxylated coagulation factors appear which are known as proteins induced by vitamin K absence (PIVKAs) [17, 19, 20]. Lucas' PIVKA-II protein was found to be highly elevated (22.7 ng/mL) on August 28 (normal range 0.0-3.5 ng/ml). PIVKA-II proteins usually undetectable in healthy infants received adequate amount of vitamin K. A study of vitamin K1 and (PIVKA)-II concentrations was conducted in healthy breast-fed infants at the ages of 2, 4, 8 and 12 weeks with either once 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 1 mg per week or 25 micrograms vitamin K1 per day was the only regimen found to be effective in prevention of vitamin K deficiency in breast-fed infants during the first three months of life [21].

Furthermore, both prothrombin time (PT) and partial thromboplastin time (PPT) were found to be elevated at the time of Lucas admission in the hospital. The treatment of Lucas with vitamin K on August 28 reduced PT and PPT by 20% and 25%,

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respectively (Table 9). PT and PTT are considered important indicators for vitamin K deficiency [17-19, 21-23]. PT and PTT were measured in fifteen infants at the age of 30 to 150 days who suffered from vitamin K deficiency and was 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 ± 43.0 seconds and PTT was 123.4 ± 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 [22].

The diagnosis of vitamin K deficiency should be suspected in nearly all infants with findings on screening coagulation studies. A healthy-appearing infant with hemorrhaging should be suspected of having vitamin K deficiency. The final diagnostic confirmation of vitamin K deficiency is a rapid, therapeutic response to vitamin K1 administration [24] as it so happened in Lucas' case (Table 9).

Newborns and infants who develop vitamin K deficiency usually suffer from bleeding in the brain and other locations similar to those observed in Lucas' case. 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 [25]. In a second study, fifteen infants aged 30-150 days who developed bleeding in the brain and other locations were found to be suffering from vitamin K deficiency. All infants were breast-fed 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) was taken and showed intraparenchymal, intraventricular, and subarachnoid hemorrhage. In addition, two infants had neurologic manifestations and hemorrhagic findings in cerebrospinal fluid. Skin bleeding (ecchymosis) was also observed in three patients [22].

Also, bleeding in the brain was observed in eleven infants between 30 and 119 days of age (mean: 56 ± 24 days) that developed vitamin K deficiency. None of these infants received vitamin K after birth and all of them were breast-fed. The localizations of the intracranial hemorrhage were as follows: intracerebral (91%), subarachnoid (46%), subdural (27%), and intraventricular (27%) [26].

In addition to the bleeding in the brain and other locations, Lucas also suffered from diarrhea, vomiting after meal, and lack of neurologic responses similar to those described in infants who developed vitamin K deficiency. In a study that included fifteen infants (30-150 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%). Physical examination revealed there was bulging or full fontanel in ten patients, diminished or absent neonatal reflexes in nine patients and ecchymosis in three patients [22].

In a second study, eleven breast-fed 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, tense or bulging fontanelle (73%), anisocoria (36%), weak neonatal reflexes (18%), and cyanoses (18%) were the most frequent findings [26].

There are many factors that led to vitamin K deficiency in Lucas' case that included (1) Lucas was breast-fed and human milk is very low in vitamin K, (2) Lucas' mother was treated with antibiotics while breast-feeding Lucas which reduced the synthesis of vitamin K in her intestinal tract and the level of vitamin K in her milk (Table 2), (3) Lucas was breast-fed milk containing antibiotics which reduced the synthesis of vitamin K in Lucas' intestinal tract and caused diarrhea, (4) Baby Lucas suffered from diarrhea and malabsorption and that 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 reduced the synthesis of vitamin K, and (6) Lucas suffered from diabetes and a urinary tract infection which reduced food intake. The influence of these factors on the levels of vitamin K in the body and the involvement of vitamin K in the synthesis of the clotting factors are very well established in the medical literature. Below are the reviews of some published studies that support my assertion.

IV-B1. Human milk contains low levels of vitamin K

Vitamin K deficiency remains a worldwide problem in breast-fed infants because the transfer of vitamin K from mother through placenta to infant is very poor and vitamin K concentrations in human milk are very low [27]. The daily requirement for vitamin K in an infant is about 1 :g/Kg and breast milk contains 1 to 3 :g 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 [17].

The newborn infant 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 :g/day and vitamin K1 intake in human milk-fed infants of about 0.5 :g/day [28-29]. Plasma vitamin K concentrations in the infants fed human milk remain 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) [21]. The daily intake of formula-fed infants has been found to be 50 :g/day [28].

Hemorrhagic disease of the newborn, secondary to vitamin K deficiency, remains largely a disease of breast-fed infants [27].Vitamin K deficiency causes hypoprothrombinemia and reduces the concentration of the other vitamin K-dependent coagulation factors, manifested by defective coagulation and hemorrhage [17]. Hemorrhages, for example were observed in four exclusively breast-fed 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 [30].

Furthermore, a prospective clinical trial of different methods of vitamin K prophylaxis was designed to establish recommendations for the prevention of vitamin K deficiency in healthy breastfed infants [21]. In this study, breast-fed infants at the ages of 2, 4, 8 and 12 weeks were treated with either once 1 mg vitamin K1 orally (n = 165) or intramuscularly (n = 166), or weekly 1 mg orally (n = 48), or daily 25 micrograms orally (n =

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58). The two single administrations of 1 mg were found to be insufficient to prevent the appearance of PIVKA-II and vitamin K deficiency after the age of 1 month. When vitamin K was administered at 1 mg per week or 0.025 mg per day, significantly higher concentrations of vitamin K1 were found and no PIVKA-II was detectable.

In a second study, thirteen breast-fed infants were given 1 mg vitamin K1 by intramuscular injection at birth. The levels of vitamin K in plasma reached as high as 32711 ± 25375 pg/mL shortly after birth. However, at one month of age the vitamin K1 levels of these infants were down to 698 ± 536 (n= 9) and this is the range found in breast-fed infants not receiving vitamin K prophylaxis [18]. Furthermore, 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 of 1 mg of vitamin K at birth [31]. Lucas was given only 1 mg vitamin K at birth and based on these studies; this dose is insufficient to prevent the development of vitamin K deficiency in Lucas' case. 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 and thereby reduced the synthesis and uptake of vitamin K from the intestinal tract.

IV-B2. The use of antibiotics by the mother during the breast-feeding period and/or infant predisposes the infant to vitamin K deficiency

The oral intake of therapeutic doses of antibiotics usually alter the balance of normal colonic flora and allow overgrowth of cholisteridium 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. difficle. Almost any antibiotics can lead to C. difficle infection. The occurrence of diarrhea is found to be more frequent with use of broad-spectrum antibiotic penicillins (e.g., ampicillin, amoxicillin) and cephalosporins [17]. The use of penicillins by nursing mothers can cause diarrhea in breast-fed infants [32].

Diarrhea and malabsorption in infants can predispose them to vitamin K deficiency. If the mother has ingested cephalosporins antibiotic, the risk of hemorrhage increases [5]. A study was undertaken to determine the frequency of occurrence of vitamin K deficiency in 75 infants with diarrhea when compared with 18 healthy infants used as control. Screening coagulation tests, PT and PTT along with estimation of functional activity and total antigenic levels of prothrombin were performed. 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 statistically highly significant ($p < 0.001$). Low ratio was observed in 57.3% (43/75) of infants with diarrhea [33].

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 breast-feeding Lucas (Table 1).

IV-B3. Liver and bile problems can predispose infants to vitamin K deficiency

The liver is an important organ for the synthesis of the 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 [24]. Hanawa *et al.* evaluated 57 infants from 2 weeks to 4 months of age and they discovered that they 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 [25].

Furthermore, Payne and Hasegawa evaluated a 4 week old, breast-fed female infant who appeared healthy until signs and symptoms of CNS deterioration suddenly occurred. At presentation the infant was found to have a left-sided parietal intracerebral hematoma, markedly prolonged prothrombin time, and partial thromboplastin time, 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 prothrombin time and partial thromboplastin time to normal values within 18 hours suggesting that the infant had severe vitamin K deficiency complicated by intracerebral hemorrhage [24].

The levels of serum liver enzymes were elevated at the time of Lucas' admission to the hospitals on August 27 suggesting liver damage (Table 8). The synthesis of the coagulation factors occurs in the liver and liver damage can cause bleeding problems and that should have been considered in the differential diagnosis of Lucas' case.

IV-B4. Diagnosis of vitamin K deficiency

The diagnosis of vitamin K deficiency is suspected on the basis of symptoms, signs, and a history suggesting the possibility of vitamin K deficiency. It is confirmed when the PT and PTT are prolonged [17]. 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 [24].

It is unfortunate that the medical examiner overlooked the obvious facts that Lucas suffered from diabetes, urinary tract infections, vitamin K deficiency and that these illnesses caused cardiac arrest, bleeding in the brain and other locations and edema. He also did not consider the contribution of adverse reactions to vaccines and medications given to Lucas to the causes of his injuries and death in this case. The Medical Examiner simply concluded that Lucas died as a result of blunt force trauma to the head. However, his own autopsy findings do not support his conclusions. I further elaborate upon the Medical Examiner's autopsy findings and my own analysis of them in Section V.

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Section V. Medical Examiner's Autopsy Findings in Lucas' Case and My Analysis

Lucas died on September 2, 2002 and the Medical Examiner performed the autopsy on September 4 (autopsy No. C-02-581). The Medical Examiner works with Forensic Pathology Associates, Inc. in Allentown, PA. The main objective of this autopsy was to establish the causes of injuries and death in this case. The Medical Examiner issued his report on January 3, 2003 describing his final findings in this case [11]. 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."

The Medical Examiner presented a list of lesions in his report that were supposedly caused by force blunt trauma in this case without providing gross and microscopic descriptions for these lesions or describing the connection between the blunt force trauma and the occurrence of these lesions. These lesions include: A) diffuse subdural hematoma and patchy subarachnoid hemorrhage, brain; B) subdural and subarachnoid hemorrhage, spinal cord; C) bilateral retinal and perineural optic nerve hemorrhage; D) diffuse cerebral edema; E) diffuse axonal injury; F) pneumonia of the right lower lobe of lung and contusions of left upper lobe of the lung; G) fracture of the left posterior rib #11.

The clinical data presented in Sections I through IV clearly indicate that Lucas' injuries and death resulted from adverse reactions to vaccines and medications which led to vitamin K deficiency, bacterial infections, and other metabolic changes. In addition, the fracture of Lucas' rib #11 likely happened during labor. The following are descriptions of medical evidence that support my assessment.

V-A. Diffuse subdural hematoma and patchy subarachnoid hemorrhage, brain

The Medical Examiner stated in his autopsy report that he found a diffuse subdural hematoma and patchy subarachnoid hemorrhage in Lucas' brain region without giving details concerning the magnitude and the distribution of those lesions and the method used to examine those lesions (gross examination vs. microscopic findings). Furthermore, he concluded that those lesions were caused by blunt force trauma to the head without providing medical evidence to support his claim. My review of the case history and the clinical evidence related to Lucas' case presented in this report indicates that the bleeding in the subdural space and the brain resulted from vitamin K deficiency, liver damage, and the treatment with epinephrine. The bleeding occurred at several stages prior, on, and after August 27 (the day of Lucas' hospitalization).

My conclusions are based on the following facts:

1) Physicians examined Lucas on August 27 and no evidence of trauma was found in the head region or any other part of his body. In addition, a computerized tomography (CT) scan of the head region taken on August 27 did not show any evidence of trauma or bone fracture.
2) The clinical evidence presented in Sections III and IV of this report shows that Lucas suffered from vitamin K deficiency and liver damage. These illnesses in and of themselves are known to cause bleeding in the brain as well as bleeding in other locations. For example, prothrombin time (PT) and partial thromboplastin time (PPT) were elevated on August 27 and the treatment of Baby Lucas with vitamin K reduced PT and PTT by 20% and 25%, respectively (Table 9).
3) The Medical Examiner stated in his report that the presence of chronic subdural bleeding cannot be excluded with certainty as shown by the CT scan of August 27. The blood products were of various ages. That means that bleeding in the brain started at several days to several weeks prior to August 27. It is unfortunate that these important observations were not further studied by the Medical Examiner by examining H & E stained tissue sections of the hematoma and the meninges microscopically to observe the proliferation of fibroblasts contained within the blood clot in the subdural space and in the clot attached to the dura matter.
4) The CT scan of the brain taken on August 29 at 0816 showed an increase in blood in the interhemispheric fissure and extraparenchymal hemorrhage when compared with the scan of August 27. This indicates that the increase in the bleeding resulted from the use of medications in the hospital such as the epinephrine. Epinephrine increases blood pressure and causes cerebral bleeding [3].
5) At the time of admission to Centre Community Hospital, Dr. Clifford J. Neal examined Baby Lucas and did not see evidence of ecchymotic lesion on 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. These observations indicate that Lucas suffered from a systemic problem that caused bleeding in the head region and in other several locations. They also indicate that the intensity of bleeding increased following the admission of Lucas in the hospital and this can be explained by the use of epinephrine and hypoxia caused by the excessive use of sodium bicarbonate.

V-B. Subdural and subarachnoid hemorrhage, spinal cord

The Medical Examiner reported the presence of bleeding in the subdural and subarachnoid spaces of the spinal cord in Lucas' case without giving any information concerning the magnitude and the distribution of the bleeding, and the method used to examine those lesions (gross examination vs. microscopic findings). Furthermore, he concluded that those lesions were caused by blunt force trauma to the head without providing medical evidence to support his claim. Numerous physicians examined Lucas on August 27 and no evidence of trauma was found in the vertebral column region or any other part of his body. In addition, a computerized tomography (CT) scan and x-ray of the vertebral column taken on August 27 and during Lucas' hospitalization did not show any evidence of trauma in the vertebral column region. The presence of the bleeding in the spinal cord region further supports the evidence that the bleeding in Lucas' case was caused by metabolic changes such as vitamin K deficiency and liver damage as explained in this report (Sections III and IV) rather than trauma. The use of epinephrine and hypoxia resulted from the excessive treatment

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with sodium bicarbonate that also contributed to the bleeding in the spinal cord.

V-C. Bilateral retinal and perineural optic nerve hemorrhage

My evaluation of Baby Lucas' case reveals the presence of many risk factors that usually lead to bleeding in the retina. These factors are explained in sections III and IV of this report. Briefly stated Lucas suffered from (1) vitamin K deficiency that causes systemic bleeding, (2) Lucas had a liver problem that led to reduction in the synthesis of coagulation factors and bleeding, (3) Lucas had diabetes and retinal hemorrhage (inner retina, superficial nerve fiber layer, and preretinal hemorrhage) which are commonly described in patients suffering from diabetes [5, 34]. It seems that the Medical Examiner overlooked the medical evidence described above that provides an explanation for the factual causes of the bleeding observed in Lucas' eyes.

V-D. Diffuse cerebral edema

Cerebral edema in Lucas' case mostly developed in the hospital as a result of his treatment with excessive amounts of sodium bicarbonate that also caused hypoxia and damage to the capillary endothelial cells. The fluid leaks into the extracellular space through damaged capillary endothelial cells that have lost their barrier function. A computerized tomography (CT) scan of the brain taken on the August 27 showed no evidence of hydrocephalus and the ventricles were non-dilated. Over the next 24-48 hours from the time of admission, the CT scan had revealed deterioration with increased edema. The fontanel became fuller. This led the doctors to make an incision in the baby's head and to insert a tube into the ventricles in order to drain the excess fluid.

Furthermore, Lucas' treatment with excessive amount of sodium bicarbonate also caused pulmonary edema. A chest x-ray taken on August 27 at about 1400 showed no infiltrates in the lungs. However, the chest x-rays taken on August 30 at 0750 and 1950 showed evidence that both lungs appeared diffusely opacified at this time, which is consistent with areas of consolidation or edema**.** In addition, the chest x-ray taken on September 1, 2002 at 0925 showed more diffuse abnormalities throughout the lung field than was present on the earlier examination. That appearance would be compatible with pulmonary edema.

It seems that the Medical Examiner overlooked these clinical observations and instead attributed the edema in the brain to blunt force trauma to the head based on a theory. No evidence of trauma to the head was observed by any of the physicians who examined Lucas nor was evidence of trauma observed as shown by the CT scans of the head region.

V-E. Diffuse axonal injury

The Medical Examiner stated that Lucas had diffuse axonal injury but he did not provide any description of this injury or the method that he used to detect this injury. He claimed that diffuse axonal injury in this case was caused by blunt trauma to the head.

I have two problems with the Medical Examiner's conclusion: (1) the physical examinations of the baby at the time of his hospitalization, and the x-rays and CT scans of the head region did not show any evidence of trauma, and (2) axonal injuries indistinguishable from those observed in cases of head trauma can occur as a result of edema, hypoxia, hypoglycemia, cardiac arrest, and other causes. In this case, the child was suffering from brain edema, hypoxia, and cardiac arrest. All causes that lead to an axonal injury should be considered prior to stating that this axonal injury was caused by a blunt force trauma to the head, especially in cases with no evidence of trauma. However, the Medical Examiner overlooked these medical facts and based his conclusions on a theory alone. Below is a description of the findings of six studies that show axonal injury present in the brain in cases of edema, hypoxia, and cardiac arrest, all of which were observed in Lucas' case.

Study #1

Extensive neurohistological examination was undertaken in 13 patients in whom coma was attributed to hypoglycemia. It had revealed varying degrees of widely distributed neuronal necrosis. In five of these cases there was also evidence that the intracranial pressure had been high with internal herniation. It was concluded that a significant amount of axonal injury found in these 13 cases could be attributed to hypoglycemia per se, although the amount and distribution of the axonal damage is altered in the presence of increased intracranial pressure. However, in some cases axonal damage is seen in the absence of an elevated intracranial pressure and in one case its distribution closely mimicked that seen in microscopical diffuse traumatic axonal injury. This further demonstrates that not all axonal pathology is caused by trauma [35].

Study #2

The brains of 17 individuals who died of cardio-respiratory arrest and 12 of status epilepticus were evaluated microscopically to check for axonal damage. Axonal damage was seen in 9 (53%) of 17 and 7 (58%) of 12 cases, respectively. In most of these cases there was also evidence of elevated intracranial pressure (ICP). It was concluded that the great majority of axonal damage identified in cases dying after cardiac arrest and status epilepticus can be attributed to increased ICP and the vascular complications of internal herniation. However, in some cases axonal damage was seen in the absence of an elevated ICP although the amount of damage and distribution were different from diffuse axonal injury [36].

Study #3

Sections from 28 brains showing evidence of cerebral hypoxia with no history of head injury, four with a history of head trauma but no evidence of hypoxic change, and eight with a history of head trauma and hypoxic change were evaluated by immunohistochemistry staining. Axonal damage was found in 7 (87.5%) of 8 cases of head injury and hypoxic changes and in 12 (43%) of 28 cases of hypoxia without history of head injury. The role of hypoxia, increased intracranial pressure, edema, shift effects, and ventilatory support in the formation of axonal bulbs should be considered. The presence of axonal bulbs cannot necessarily be attributed to shearing forces alone [37].

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Study #4
Brain tissue sections from 67 individuals who died due to trauma-induced focal cortical hemorrhage without dural involvement and 51 cases of non-traumatic death due to cerebral hypoxia/ischemia were evaluated by immunohistochemical staining to determine the reliability of axonal injury (AI) as a marker of traumatic insult. Investigations of the pons in these cases revealed that cases of non-traumatic death due to cerebral hypoxia/ischemia (n = 51) demonstrated AI with the same frequency as in the trauma group, although the expression tended to be less pronounced. The investigations were based primarily upon immunohistochemical demonstration of antibodies targeted to beta-amyloid precursor protein (beta-APP) in the pons as a marker of AI. The results of this study confirm that beta-APP expression in the pons is a reliable indicator of AI but does not discriminate between (a) injuries caused by traumatic strain or shearing mechanisms and (b) secondary damage due to cerebral hypoxia/ischemia or edema. Therefore, positive differentiation of the type of biomechanical event based on this criterion alone is not possible [38].

Study #5
Beta-amyloid precursor protein (beta-APP) was used to detect axonal injury (AI) in the brain of individuals who died of non-missile closed-head injury (n = 119), gunshot injury (n = 30), cerebral ischemia/hypoxia (n = 51), brain death caused by mechanical trauma (n =14), and nonmechanical injury (n = 18). AI was observed in 65% to 100% of cases of closed-head injury, fatal cerebral ischemia/hypoxia, and brain death, with a survival time of more than 3 hours. A statistically significant difference between traumatically and nontraumatically induced (nondisruptive) AI was not found [39].

Study #6
Brain tissues of 14 children who lacked skull fracture and allegedly died of Shaken Baby Syndrome (SBS) and 7 children who died of non-traumatic hypoxic ischemic encephalopathy (HIE) were evaluated using immunohistochemical stains. Swollen axons were present in 11 (79%) of 14 cases of SBS and in 6 (86%) of 7 cases of HIE [40].

V-F. Pneumonia, urinary tract infections, and liver damage

The Medical Examiner reported that pneumonia was present in the lower right lobe of Lucas' lung but he did not give information concerning the nature of this inflammation (acute vs. chronic) and the method used to identify the lesion (gross vs. microscopic). My review of Lucas' medical records revealed that Lucas developed an upper respiratory tract infection at one to two days following his vaccination on July 23. His parents later noticed that he had a cough as well as clear mucus discharge from the nose. Lucas' cough and nasal discharge continued until July 30 and his mother took him to his pediatrician. Furthermore, the urine analysis performed on August 28 showed that Lucas suffered from a bacterial urinary tract infection. A urine culture contained 100,000 colonies of Klebsiella oxytoca/mL (Table 7). Moreover, Lucas suffered from liver damage as shown by the elevated levels of liver enzymes in serum. ALT and AST enzymes were 342% and 255% of normal, respectively.

It has been reported that Klebsiella infections of the urinary tract, respiratory tract, and abdomen can result in bacteremia and each account for 15 to 30% of Klebsiella bacteremias [34, page 957]. It is possible that Klebsiella also caused Lucas' pneumonia and that was a connection between the infection in the urinary tract and the lungs. It is possible that the infection in the upper respiratory tract that developed following Lucas' vaccination extended to the lung and the urinary tracts and other locations. Lucas slept longer hours after receiving his vaccination on July 23 until the time of his hospitalization on August 27. He also vomited more than usual, ate less, and appeared especially tired especially during the two days prior to August 27. His infections made him more susceptible to vitamin K deficiency.

The Medical Examiner did not provide any information regarding the status of the urinary tract, liver, heart, and the GI tract in his autopsy report. He also overlooked the connection between Lucas' pneumonia, urinary tract infections, and liver damage and vitamin K deficiency that led to the bleeding in his brain and other locations.

V-G. Rib fractures occurred in infants during labor

A chest x-ray taken on August 27 at 1620 showed a single left posterior non-displaced healing fracture of the 11th rib. The age of this fracture is between weeks and several months old. Review of the medical literature reveals numerous cases in which rib fractures had occurred during labor but had been missed during initial examination of the baby. Lucas was born at 41 weeks of gestation by vaginal delivery with manual assistance that resulted in vaginal laceration and severe bleeding that required a blood transfusion as described in Section I of this report. His birth weight was 4.13 kg. It is very likely that Lucas' rib fracture occurred during labor similar to the four cases with rib fracture described below.

Case #1
A large weight (3912 g) for gestational age female neonate was delivered vaginally with the use of vacuum extraction. The neonate was breathing quietly with no respiratory tract distress. A chest radiograph was obtained and showed minimally displaced fractures of ribs 4 through 8 posteriorly on the right side. The lungs, heart, and other skeletal structures were normal [41].

Case #2
A large weight (4205 g) for gestational age, term male neonate was delivered vaginally with the use of vacuum extraction to assist delivery. A chest radiograph showed non-displaced fractures of ribs 6 through 8 posteriorly on the right side. The baby had normal lungs and mediastinum [41].

Case #3
A 37 year old diabetic woman spontaneously went into labor at 38 weeks of gestation. She delivered a 3300 g baby with the assistance of vacuum extraction. Physical examination did not detect any abnormality. At about 9 hours after delivery the nurse noticed the child having rapid respirations. Examination by a resident physician revealed mild respiratory distress with tachypnea and tachycardia. Crepitus was palpable over the left posterolateral chest. No skin changes suggestive of trauma were

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found. Chest x-ray examination revealed five fractured ribs over the left posterolateral chest area. There was no evidence of pneumothorax or other skeletal trauma. Over the next 36 hours, the child experienced progressively less tachypnea and gradual disappearance of the crepitus. A full skeletal survey failed to show evidence of osteogenesis imperfecta or any other abnormality of bone mineralization [42].

Case #4

A chest radiograph was taken of a 4905g female delivered by midforceps after having right shoulder dystocia. On the 11th day a prominent mass was noted in the right midclavicular region. Radiograph revealed a right midclavicular fracture with slight superior angulation and incidental fractures of left ribs #5 and #6.

Moreover, Fanaroff *et al.* explained the mechanism of rib injuries during labor as follows: Rib injury is initiated when the anterior shoulder is impacted behind the symphysis pubis with the other shoulder attempting to descend into the posterior compartment of the pelvis. This results in compression forces on the fetal arm and thorax causing spontaneous rib fractures on the same side as the posterior shoulder [43]. They also stated that the specific clinical manifestations of rib injuries are often absent, making diagnosis difficult [43].

Section VI. Conclusions and Recommendations

Lucas suffered from serious health problems that led to his cardiac arrest on August 27, 2002. These included diabetes mellitus; metabolic acidosis; liver damage; a bacterial urinary tract infection; pneumonia; vitamin K deficiency; chronic and acute bleeding in the subdural space, brain, retina, and other locations; anemia; brain edema; and diarrhea. Lucas' health problems were caused by the seven vaccines received on July 23 (Table 3) and the treatment of his mother's upper respiratory tract and breast infections with antibiotics during Lucas' breast-feeding period (Table 1). Metabolic acidosis caused the reduction in the levels of potassium in the cardiac muscles and the nervous tissues that subsequently led to cardiac arrest in Lucas' case.

The vaccines given to Lucas induced an upper respiratory tract infection within 48 hours post-vaccination and I believe that this infection also caused Lucas' pneumonia and his urinary tract bacterial infection. The treatment of Lucas' mother with antibiotics predisposed Lucas to vitamin K deficiency by reducing the levels of vitamin K in her breast-milk, causing Lucas' diarrhea, and thereby reducing vitamin K synthesis in Lucas' gastrointestinal tract (GIT) and vitamin K uptake from the GIT. Lucas also suffered from liver damage and other systemic problems that reduced the synthesis of coagulation factors in his liver and reduced food intake. Vitamin K deficiency was the primary cause of bleeding in this case.

Furthermore, epinephrine given to Lucas in the hospital on August 27 and thereafter also contributed to the subdural bleeding and bleeding in other locations as shown by several brain CT scans taken on August 27 through August 30. In addition, on August 28, the blood pH reached a critical low of 6.64 and the baby was treated with sodium bicarbonate. Unfortunately, he was treated with excessive amounts of sodium bicarbonate and the blood pH reached a critical high of 7.67 (Tables 4, 6, 10, 11). This treatment caused brain and pulmonary edema, hypoxia, and hypokalemia.

The treatment with bicarbonate for individuals with diabetes should stop at pH 7.2 because this treatment carries a high risk of causing cerebral edema, as occurred in this case. It seems that the treating physicians overlooked the adverse reactions of sodium bicarbonate as well as the recommendations presented in medical textbooks concerning the risk associated with the treatment with bicarbonate. The treating physicians additionally overlooked the biomarkers for vitamin K deficiency and assumed that the bleeding in brain and other locations were caused by shaking the baby.

The radiology findings showed that Lucas had an old-healed fracture of rib #11. Rib fractures have also been observed to occur during labor in babies as explained in this report (V). Lucas was born by vaginal delivery at 41 weeks of gestation with manual assistance and the force used caused his mother to severely hemorrhage as a result of vaginal lacerations. She further suffered from hypotension and anemia that required a blood transfusion. It is very likely that Lucas' rib fracture happened during labor.

The Medical Examiner performed an autopsy on Lucas on September 4, 2002 (case # C-02-581) with the main objective to establish the factual causes of injuries and death in this case. 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." I find that the Medical Examiner's conclusions are unsupported by the clinical data related to this case which are described in this report. I presented my arguments against the Medical Examiner's methods of investigating this case and his conclusions along with the supporting medical evidence in Section V of this report. The following is a list of some of the problems concerning the Medical Examiner's methods of investigation and his conclusions of the causes of injuries and death in this case:

1. The Medical Examiner stated that Lucas' cardiac arrest and bleeding was caused by blunt force trauma to the head. However, he did not provide any evidence that the baby suffered from trauma. In addition, physicians examined Lucas on August 27 and no evidence of trauma was found in the head region or any other part of his body. Furthermore, the 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 presented a list of lesions in his autopsy report without providing the gross and microscopic descriptions for these lesions (V).
3. The clinical data presented in this report shows 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, the Medical Examiner did not investigate the contribution of these illnesses to the causes of bleeding in the tissues and death in this case.
4. The Medical Examiner overlooked the well-established biomarkers of vitamin K deficiency observed in this case. Lucas had a high level of PIVKA-II protein which is a sensi-

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tive marker for vitamin K deficiency. In addition, prothrombin time (PT) and activated partial thromboplastin time (APTT) were elevated on August 27 and the treatment of the baby with vitamin K reduced PT and APTT by 20% and 25%, respectively (Table 9).

5. 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. That indicates that bleeding had started several days to several weeks prior to August 27. However, the Medical Examiner did not examine H & E stained tissue sections of the subdural hematoma and the meninges microscopically to evaluate the structure and the age of the bleeding.
6. Lucas' medical chart shows that Lucas suffered from urinary tract bacterial 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 urinary tract infection.
7. The Medical Examiner stated that Lucas suffered from diffuse axonal injury but he did not provide the description of this injury or the method that he used to detect this injury. In addition, he claimed that diffuse axonal injury in this case was caused by blunt force trauma to the head. I described several studies in this report that show axonal injuries indistinguishable from those observed in cases of head trauma as were described in the cases of edema, hypoxia, hypoglycemia, cardiac arrest, and other causes (V-E). In this case, the baby suffered from brain edema, hypoxia, and cardiac arrest. However, Medical Examiner neglected to perform a differential diagnosis in this case.
8. The Medical Examiner did not evaluate the contribution of the adverse reactions of medications given to Lucas in the hospital to the causes of his 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 the bleeding in his tissues.
9. The Medical Examiner did not evaluate the contribution of the adverse reactions of vaccines given to Lucas to the causes of bleeding and death in Lucas' case. Lucas developed an upper respiratory tract infection within 48 hours post-vaccination. Serious systemic injuries and death have been reported in babies also have received vaccines (II-B).
10. The Medical Examiner assumed that the old-healed fracture of rib #11 observed in Lucas' case had resulted from child abuse without performing a review of the medical literature to find out if rib fractures in infants had been reported to occur during labor. I presented several studies that show rib fractures do occur during labor (V-G).

The Mullenax and Mendez family have suffered from two tragedies because the physicians who treated Lucas with vaccines and treated his mother with antibiotics during the breast-feeding period did not take into consideration the adverse reactions of those agents on Lucas' health. In addition, the physicians who treated Lucas during his hospitalization following his cardiac arrest and the medical examiner in charge in this case failed to consider in their investigation the adverse reactions of medications and vaccines given to Lucas and the adverse reaction of antibiotics given to Lucas' mother. The first tragedy is the loss of Baby Lucas because of adverse reactions to vaccines and medications. The Mullenax and Mendez' second tragedy is the false accusation against Lucas' parents, alleging they killed their Baby Lucas–a horrible crime that they did not commit.

I urge the physicians, health care workers, and officials who are involved in this case as well as the state of Pennsylvania to review the medical evidence presented in this report. It clearly shows that Lucas died as a result of the adverse reactions to vaccines and medications and that Lucas' parents are innocent. Actions should be taken to prevent similar tragedies from occurring again. The objective of the state and health care workers should be determining the factual causes that lead to the illness and death of a child and to prevent such incidences from happening to other children. Accusing innocent parents of abusing and killing their children based on unsupported theory, as occurred in this case, will not prevent the death of other children from the adverse reactions to vaccines and medications. It certainly imprisons innocent people and causes tremendous suffering. It also costs taxpayers huge sums of money to pay for unnecessary trials and legal fees.

I believe that the following recommendations will help to prevent future infant deaths from the adverse reactions to vaccines and medications in addition to deterring innocent people from being wrongly accused, incarcerated, and convicted for a crime that never occurred:

1. Babies who show adverse reactions to vaccines should be monitored closely. Their blood should be analyzed to check for the levels of pH, gases, glucose, potassium, vitamin K, and coagulation factors.
2. Breast-fed babies should be given 1 mg of vitamin K monthly to prevent bleeding in the brain and other locations. An ill breast-fed baby who has feeding problem and vomiting may require higher doses of vitamin K.
3. Mothers receiving antibiotics should avoid breast-feeding their babies during their course of treatment if possible.
4. Babies who are admitted to the hospital with bleeding should be checked for vitamin K deficiency, liver damage, and should be given vitamin K supplementation.
5. The use of sodium bicarbonate in the treatment of acidosis should be avoided if possible. Children who are treated with sodium bicarbonate should be monitored closely. In addition, the standard recommendations regarding the use of bicarbonate to treat acidosis should be followed in order to prevent the excessive use of bicarbonate and the development of brain edema.
6. In cases similar to Lucas' case, medical examiners and physicians should thoroughly review the medical evidence and perform differential diagnosis prior to giving their conclusions that a child died as a result of a blunt trauma to the head (Shaken Baby Syndrome).

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