Analysis of causes that led to Toddler Alexa Marie Shearer's cardiac arrest and death in November, 1999

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Summary

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

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

Kathleen Butcher and her husband, Ducman Butcher, requested that I evaluate the medical evidence in Alexa's case to find the factual cause(s) that led to Alexa's cardiac arrest and death in November of 1999. I evaluated Alexa's case by reviewing: her medical records, autopsy report, adverse reactions to vaccines and medications given to Alexa, trial documents and testimonies of expert witnesses, and the medical literature pertinent to this case. I used differential diagnosis to evaluate the contribution of agents relevant to this case and the possible synergistic actions among agents in causing Alexa's cardiac arrest, apnea, bleeding, pathologic changes in tissues, and death in this case.

I present my review and analysis of Alexa's medical records from birth on August 11, 1998 to the time of her cardiac arrest on November 16, 1999 in Section I. I also explain the adverse reactions of vaccines given to Alexa in this section. In Section II, I illustrate the clinical events that occurred during Alexa's three days in the hospitals following her cardiac arrest, and my analysis of these events. Section III contains a description of the abnormal findings observed in Alexa's abdominal cavity during the organ harvesting procedure and the adverse reactions to heparin given to Alexa. In Section IV, I describe the pathogenesis of Alexa's illnesses and their contributions to her cardiac arrest and the pathology of her lesions. My detailed review and analysis of the medical examiner's autopsy report and his court testimony are presented in Section V. My review of the State's Expert Witness' testimony is described in Section VI. She was the second key expert witness for the State. Section VII contains my conclusions and recommendations.

Alexa was born on August 11, 1998; she was near term and was delivered by Caesarean section. She suffered from jaundice and an upper respiratory tract bacterial infection during the first week of her life. Her blood bilirubin level was 16.5 mg/dL at five days following birth, which is about 8 times the expected normal level of 2 mg/dL. Neurological damages have been observed in some infants who had blood bilirubin level > 12 mg/dL.

Alexa's appetite became poor at about 10 months of age and her appetite got worse gradually toward the time of her death at 15 months. For example, on July 20, Alexa's mother told Alexa's pediatrician that Alexa had a poor appetite for the last 2-3 weeks. She developed white thrush on her tongue and was treated with three consecutive courses of Nystatin (anti-fungal) orally that caused vomiting and diarrhea. Alexa's physician overlooked her chronic health problems and vaccinated her with the polio (IPV) and hepatitis B (Hep B) vaccines on July 20, 1999 at 11 months of age.

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

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

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

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

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

Alexa was vaccinated with four attenuated live viruses vaccines (measles, mumps, rubella, and varicella) on August 13, 1999 when she was suffering from serious chronic health problems. Alexa's poor weight gain and her low food intake caused a significant depression in the functions of her immune system, especially the T-cell count and functions. It made Alexa's re-

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sponse to the vaccines given inadequate and increased her risk for having serious adverse reactions to vaccines and developing infections. The MMR and varicella vaccines caused the following serious illnesses that led to Alexa's cardiac arrest and apnea on November 16, 1999.

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

2) The MMR and varicella vaccines along with her viral and bacterial infections caused Alexa to eat less, lose weight, develop anemia, vitamin K deficiency, and led to significant immune depression, especially T-cell counts and functions. The mumps virus from the vaccine probably overcame Alexa's weakened immune system and infected the pancreatic tissues. The clinical tests and the pathological findings in the abdominal cavity indicated that Alexa suffered from acute pancreatitis, which led to her cardiac arrest and apnea on November 16, 1999.

I reviewed the Medical Examiner's autopsy report and his court testimony in this case and found that his autopsy and his investigation of this case were incomplete. He also misinterpreted the clinical data including the results of his own tests. He presented the wrong conclusions to the police and the court about the causes of injuries and death in this case. His work led to the false accusation, arrest, and conviction of Kathleen Butcher for a horrible crime that she did not commit. The Medical Examiner's work should be reviewed by the medical board and the State in order to save innocent people from the false accusations of having killed children by the so called "Shaken Baby Syndrome."

I present my detailed analysis of the Medical Examiner's autopsy report and his court testimony in Section V of this report. Below is a list of observations that show the problems with his investigation of this case:

1) The Medical Examiner did not review Alexa's medical records prior to her cardiac arrest to find out if she had preexisting health problem(s) that contributed to her cardiac arrest on November 16, 1999. My investigation showed that Alexa suffered from chronic health problems and her illnesses increased her risk to develop an adverse reaction to vaccines.

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

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

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

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

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

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

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

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

10) The Medical Examiner did not examine the lytic lesion in Alexa's T-10 vertebrae microscopically nor was a bone scan performed to rule out osteomyelitis. He claimed that Kathleen broke Alexa's vertebrae by hyperextension. The Medical Examiner's claim was not supported by medical facts and even by his own findings. He found fresh bleeding in the tissues associated with T-10 and T-8 but not in T-9. If Kathleen put tremendous pressure on Alexa's back to break her T-10 vertebrae and caused bleeding in T-10 and T-8, then we expect to see bleed-

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ing in T-9, too. We would also expect to see a three-day old bleed and not a fresh bleed.

11) The Medical Examiner did not investigate the cause(s) of the left 8th rib fracture in this case.

Investigating this matter in a scientific manner may reveal the factual cause(s) of the rib fracture in this case, and may help in explaining the causes of Alexa's cardiac arrest.

Alexa suffered from vitamin K deficiency and vitamin K is important for calcium metabolism in bone. People who suffer from vitamin K deficiency also suffer from bone problems. The lesion in the rib observed in Alexa's case may represent a local bone defect caused by vitamin K deficiency and followed by healing as I describe in Section V of this report.

I also reviewed the State's Expert Witness' report and her testimony in this case (Section VI). She is a physician, and was asked by the prosecutors to review Alexa's case and to provide her opinion concerning the causes of injuries and death in this case. She declared that Alexa's injuries were caused by violent and repeated shaking and blunt trauma to the head and abdomen after she ate lunch at 1215 on November 16.

The medical evidence presented in this report does not support the State's Expert Witness' theory and her conclusions. I discussed her findings and testimony in Section VI. She based her conclusions mainly on the Medical Examiner's incomplete autopsy report. She also added a new false allegation to this case that Alexa was struck by blunt trauma to the abdominal region. Her allegation was not supported by the medical examiner who did the autopsy, the surgeon who harvested Alexa's organ and examined the abdominal cavity and the results of the CT scans taken for Alexa's abdominal region on November 16 and 17.

I believe that the State's Expert Witness' involvement in this case misled the court and Alexa's family into believing that Kathleen killed Alexa. She caused great harm to Kathleen and her family. Furthermore, the criteria used by the State's Expert Witness to diagnose cases of SBS are not supported by medical facts. The use of her criteria will likely put many innocent parents and daycare providers at great risk of being falsely accused and imprisoned for harming their children by violent shaking. The State's Medical Witness' work in this case and her involvement with other cases of alleged SBS should be reviewed by a medical board in order to save innocent people from being accused of horrible crimes that they did not commit. I described her criteria in Section VI of this report.

The extensive medical evidence presented in this report clearly shows that Alexa and her family as well as Kathleen and her family were the victims of a broken medical system that needs to be urgently fixed. Alexa died as a result of adverse reactions to vaccines and medications and Kathleen was convicted and imprisoned because of sloppy and incomplete medical investigations. Alexa was given four live vaccines without any consideration for her chronic health problems and her immune depression. The nurse and the physician who did this should bear the responsibility for injuring Alexa.

I believe that Howard County and the State of Maryland have the responsibility to review the evidence presented in this report. It shows that Kathleen is innocent, and that they should take immediate action to free her from prison so that she may be reunited with her five children (ages 2-11 years) and her husband. Additionally, the State should investigate the Medical Examiner and the States Expert Witness' involvement with similar cases resulting in the conviction of a parent or caretaker accused of having killed a child by SBS.

Furthermore, I believe that the doctors who caused Alexa's death and her family's suffering should compensate this family for the loss of their child, their suffering, in addition to the reimbursement of expenses paid. These same physicians should be held responsible for compensating Kathleen and her family for the pain and suffering they have endured as a result of this grave injustice which has transpired as a result of the physicians' poor and inadequate medical investigations.

The objective of the State and the medical system should be determining 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 and daycare providers of abusing and killing their children based on unsupported theory as happened in this case will not prevent the death of other children by vaccines and adverse reactions to medications. However, it certainly puts innocent people in prison and causes great suffering. It also costs taxpayers huge sums of money in order to pay for trials and legal fees.

I spent approximately 300 hours evaluating the medical evidence in this case to find the factual causes of death and to write this detailed report. I hope that the State of Maryland, our Federal Government, physicians, and our society will take the time to review the evidence and then act to rectify the problems. The entire "Shaken Baby Syndrome" theory should be evaluated. This is the third case that I have evaluated within a 10-month period involving children who died as a result of adverse reactions to vaccines and their parents or their caretaker were falsely accused of killing them and imprisoned. Differential diagnosis should be used to solve complicated medical problems as I have used in this case to find the factual causes of the problem.

Section I. Review of Alexa M. Shearer's Medical Records from time of Birth on August 11, 1998 to November 16, 1999 and Analysis of Her Health Problems

I-A. Review of Alexa's birth record

Alexa Marie Shearer was born on August 11, 1998 via a Caesarean section at Howard County General Hospital in Maryland. She was a white female, born near term and her birth weight was 6 pounds and 9 ounces (2977g). Her length and her head circumference were 19¾ inches and 33 cm, respectively [1, 2]. Her prenatal medical record was not available for review to know the medical reason(s) that led to her delivery by Caesarean section. She stayed in the hospital for five days and was discharged on August 15.

Her medical record indicates that she suffered from jaundice and her blood bilirubin level was 16.5 mg/dL on August 16. It was about 8 times the expected normal level of 2 mg/dL (Table 1). In addition, she suffered from an upper respiratory tract bacterial infection (streptococcus) on August 16 and the standard treatment for this infection is to administer antibiotics. Antibiotics usually compete with the unconjucated bilirubin in binding with plasma protein and lead to increase in the levels of unbound unconjucated bilirubin in plasma. The free unconjucated

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bilirubin can cross the blood brain barrier and cause neurological damage as explained below (Section I-B).

Table 1. Alexa's bilirubin levels in blood at 3 through 6 days after birth

Date	Bilirubin level (mg/dL)*	Age
08/14/98	15.1	3
08/15/98	16.1	4
08/16/98	16.5	5
08/17/98	15.9	6

*Expected normal value = 2 mg/dL

I-B. The impact of jaundice on Alexa's health

Alexa suffered from neonatal jaundice and her blood bilirubin level was 16.5 mg/dL at five days after birth. It was about 8 times the expected normal level (Table 1). In the full-term newborn, physiologic jaundice is characterized by a progressive rise in unconjugated bilirubin concentration from approximately 2 mg/dL in cord blood to a mean peak of 5 to 6 mg/dL between 60 and 72 hours of age in white infants. This is followed by a rapid decline to approximately 2 mg/dL by the fifth day of life [3].

During the period from the fifth to tenth day of life in white infants, blood bilirubin concentrations usually decline slowly, reaching the normal adult value of less than 1.3 mg/dL. However, in premature neonates, physiologic jaundice is more severe than in the full-term neonates, with mean peak concentrations reaching 10 to 12 mg/dL by the fifth day of life. This delay in reaching normal concentration of less than 1.3 mg/dL as compared with the full-term neonates reflects the delay primarily in maturation of hepatic glucuronyl transferase activity in the premature neonate [3]. The blood bilirubin levels observed in Alexa's case exceeded the levels observed in premature neonates (Table 1).

Bilirubin is one of the products of heme catabolism. It is a weak acid and does not dissolve in water or readily excreted at pH 7.40 without conjugation with glucuronic acid in the liver. It can penetrate the blood brain barrier and cause neurological problems. Hyperbilirubinemia is capable of producing a spectrum of neurological dysfunction in the newborn, ranging from transient mild encephalopathy to permanent severe neurological impairment secondary to neuronal necrosis [3, page 1324]. A mean peak unconjugated bilirubin concentrations in the blood of 10 to 12 mg/dL may cause bilirubin encephalopathy in certain high-risk neonates [3, page 1317].

Furthermore, auditory brainstem responses were performed in nineteen newborns with blood bilirubin levels ranging from 12 to 20 mg/dL using non filtered clicks at 100 dB SPL (peak equivalent). Absolute and inter-peak latencies of waves I and V were measured and correlated to bilirubinemia upon acoustic stimulation and maximal bilirubinemia observed during neonatal observation. Significant correlations were noted between bilirubinemia and V or V-I latencies. It was concluded that hyperbilirubinemia affects the upper auditory pathways [4].

Bilirubin usually binds with serum albumin, and this complex does not cross the brain barrier. The bilirubin binding capacity of albumin is decreased in sick premature and full-term human neonates. In addition, serum albumin is lower in these patients. Alexa suffered from an upper respiratory tract bacterial infection and the standard treatment for this illness is antibiotics. Antibiotics can displace bilirubin from albumin and make it free which enhances the CNS toxicity of bilirubin. Alexa had a moderate blood bilirubin level of 16.5 mg/dL at 5 days of age. However, the treatment with antibiotics that bind with albumin may have increased the toxicity of bilirubin in this case by increasing the level of unbound bilirubin [3].

The term kernicterus has been traditionally used to describe the pathology of bilirubin toxicity within the brain (necrosis followed by gliosis). However, approximately half of all infants with kernicterus observed at autopsy also have extraneural lesions of bilirubin toxicity. These include necrosis of renal tubular cells, intestinal cells, and pancreatic cells in association with intracellular crystals of bilirubin. On November 16, 1999, Alexa suffered from acute pancreatitis and diabetes. It is possible that Alexa's hyperbilirubinemia caused subclinical tissue injuries that increased her susceptibility to infections.

I-C. Review of Alexa's medical records from one week to fifteen months of age

Alexa was breast-fed during the first two months of her life. After two months, her diet consisted of 20% breast-milk and 80% formula. She was fed formula milk, soft baby food, and table food as she progressed in her development. However, her medical record shows that she was a picky eater, and her appetite became poor at about 10 months of age. Furthermore, her appetite gradually declined toward the time of her death in November of 1999, when she was 15 months old.

On July 20, Alexa's mother told Alexa's pediatrician that Alexa had a poor appetite for the last 2-3 weeks prior to her appointment [1]. Additionally, Alexa's daycare provider, Ms. Lorena DelGrosso stated that Alexa had a poor appetite. She ate mostly graham crackers during the day (7:30 am to 5:00 pm) at her daycare. In addition, DelGrosso reported that Alexa vomited on many occasions immediately following the eating of soft food such as mashed potatoes or yogurt and that she had loose stools [5, page 22]. She watched Alexa from August to November 15, 1999 approximately 2-3 days per week.

Alexa's father also testified that Alexa was very fussy. He said that during October and November of 1999, Alexa just wanted to take her milk bottle and she didn't want to eat her food. She didn't eat fruit or anything at the table. She ate a little bit of formula and the powder cereal mix [6].

Alexa's poor appetite, diarrhea, and vomiting between July and November of 1999 caused Alexa's poor weight gain and increased her susceptibility for fungal, bacterial, and viral infections. At 2 months of age, Alexa was in the 50th percentile for weight on the growth chart. Her weight dropped to below the 1st percentile at 15 months of age. Her length also dropped from the 25th percentile at 7.4 months of age to the 10th percentile at 12 months of age. Alexa's growth measurements are presented in Table 2.

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Table 2. Alexa's weight and length measurements

Date	Age months	Weight* lbs.	Percentile on chart	Length inches	% on chart
10/11/98	2.0	9 lb 11 oz	50	21	
12/14/98	4.1	12 lb 3 oz	25		
02/16/99	6.2	13 lb 8 oz			
03/22/99	7.4	14 lb 4 oz	10	25.9	25
07/20/99	11.3	16 lb 13 oz		27.5	
08/13/99	12.1	16 lb 6 oz	<5	28.0	10
11/16/99	15.2	19 lb 9 oz	<1		

*Alexa was born 08/11/98 with a birth weight of 6 pounds 9 ounces (2977 g) and length of 19.5 inches (49.5 cm).

Alexa suffered from fungal, bacterial, and viral infections in the time period between 10 and 15 months of age. Alexa developed white thrush on her tongue on July 20, 1999 and she was treated with Nystatin (anti-fungal) orally. On August 6, Alexa's mother called Alexa's doctor for refill of Nystatin because Alexa still had white thrush on her tongue and she did not want to eat. In addition, on August 16, Alexa's mother requested a second refill of Nystatin to treat her thrush. Alexa was treated with three consecutive courses of Nystatin and the common adverse reactions of Nystatin are diarrhea and vomiting.

Furthermore, Alexa's daycare provider, Ms. Lorena DelGrosso stated that while Alexa had been in her care, there were a few times that she had a fever and Ms. DelGrosso treated her with Tylenol. Alexa also suffered from an upper respiratory tract infection in November of 1999 prior to her hospitalization on November 16 [5, page 44]. Alexa's mother stated that Alexa was congested and had a low-grade fever the weekend prior to November 16 [7, page 21].

Alexa also developed anemia and vitamin K deficiency as indicated by several clinical tests. On July 20, her hemoglobin level was 11.4 g/dL and it was reduced to 8.3 g/dL on November 16. The hemoglobin concentration was reduced by 27% in four months. Alexa's hematocrit value was also low at 26.8% on November 16. Alexa's prothrombin time (PT) and partial thromboplastin time (PTT) were found to be highly elevated on November 16 and these are the biomarkers for vitamin K deficiency. Her PT and PTT values were 33.3 seconds (266% of normal) and more than 100 seconds (286% of normal), respectively [8-10]. Elevation of PT and PTT usually increase the susceptibility of the individual for bleeding. Vitamin K deficiency also interferes with calcium metabolism in bone.

Despite Alexa's chronic health problems (fungal infection, poor appetite, and poor weight gain), Alexa was vaccinated with the polio (IPV) and hepatitis B (Hep B) vaccines on July 20, 1999. Furthermore, a nurse in Alexa's doctor's office did not pay attention to Alexa's poor health on August 13, 1999 and proceeded to vaccinate her with four attenuated live virus vaccines [Measles, Mumps, and Rubella (MMR) and Varicella]. The recommended child age for receiving the MMR virus vaccines in a healthy child is fifteen months. Alexa received these vaccines three months earlier than the recommended schedule [1].

Alexa received the MMR and varicella vaccines at 12 months of age when she was chronically ill and these vaccines further compromised her health. The functions of her immune system were depressed, especially the T-cells, which led to a failure to neutralize the administered viruses and probably induced the infection of pancreas with the mumps virus. The clinical evidence showed that Alexa suffered from acute pancreatitis on November 16. The list of vaccines given to Alexa and their compositions are presented in Tables 3 and 4.

The vaccines administered to Alexa have been known to cause serious adverse reactions in even healthy children. The adverse reactions observed in some children include upper and lower respiratory tract infections, ear infections, fever, encephalitis, pancreatitis, diabetes mellitus, poor appetite, loss of weight, and teething. Alexa suffered from these illnesses as stated in her medical records and as described in this report. Some of the studies below describe adverse reactions to vaccines that were given to Alexa, these are summarized in Section I-D below.

Table 3. Alexa's vaccination history

Date	Age months	Vaccines given
10/01/98	1.6	Diphtheria & Tetanus Toxoids and Pertussis (DPT); Inactivated Polio vaccine (IPV), Haemophilus Influenzae B (Hib), and Hepatitis B (Hep B)
12/14/98	4.2	DPT, IPV, Hib, Hep B
03/22/99	7.3	DPT, Hib
07/20/99	11.3	IPV, Hep B
08/13/99	12.0	Measles, mumps and rubella (MMR) and varicella

I-D. Adverse reactions attributed to vaccines given to Alexa

Alexa received polio (IPV), hepatitis B (Hep B), MMR, and varicella vaccines when she was suffering from poor appetite, poor weight gain, a chronic fungal infection, and immune depression. Alexa's poor weight gain and her chronic fungal infection indicate that Alexa was suffering from depression in the functions of her immune system, especially the T-cell count and functions. Children suffering from malnutrition usually have a reduction in their T-cell counts and functions [11]. This made Alexa's response to the administered vaccines inadequate and increased her risk of developing serious adverse reactions to vaccines and infections.

Children who suffer from acute or chronic illnesses do not respond adequately to vaccines. Krober et al. examined 47 infants with colds and 51 well infants at the age of 15 to 18 months for their response to develop the measles antibody following the standard measles-mumps-rubella vaccine. Prevaccination serum samples were obtained prior to vaccine administration and post-vaccination serum samples were obtained 6 to 8 weeks later. Measles antibodies were measured in these serum samples by an indirect fluorescein-tagged antibody test. Ten (21%) of 47 infants with colds failed to develop the measles antibody, while only one (2%) of 51 well infants failed to develop an antibody [12].

Serious systemic adverse reactions have been reported in children who received the MMR vaccines. These include malaise, sore throat, cough, rhinitis, headache, dizziness, fever (101ºF-102.9ºF; 38.3ºC-39.4ºC), rash, nausea, vomiting, diarrhea, fever, regional lymphadenopathy, parotitis, orchitis, nerve deafness, vasculitis, otitis media, hearing loss, conjunctivitis,

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aseptic meningitis, measles, thrombocytopenia, and anaphylaxis [13, page 1820, 14-19].

Koga et al. described a case of a child who developed bilateral acute profound deafness and aseptic meningitis within 14 days after receiving MMR vaccines. The cause of this deafness was presumed to be the mumps vaccination. The basis for this presumption was that the meningitis following MMR vaccination was elicited by the Polymerase Chain Reaction (PCR) method to be caused by the mumps vaccine. The complication of the central nervous system (CNS) after measles vaccination occurs within 14 days after injection and the onset of vomiting and gait disturbance of the case occurred at 24 days after vaccination [14].

Furthermore, a 7 year old girl developed unilateral total loss of hearing at 13 days following MMR vaccination and the live, attenuated mumps-virus vaccine was suspected to be the cause of the injury [15]. Stewart and Prabhum also reported six individuals, who developed hearing loss after the measles, mumps, and rubella (MMR) immunization and MMR remained a possible etiology. They stated that any risk associated with attenuated viruses must be weighed against the risks of the natural diseases [16].

Cases of aseptic meningitis associated with measles, mumps, and rubella vaccines were sought in thirteen UK health districts following a reported cluster in Nottingham that suggested a risk of 1 in 4,000 doses. Cases were ascertained by obtaining vaccination records of children with aseptic meningitis diagnosed from cerebrospinal fluid samples submitted to Public Health Laboratories or discharged from hospital with a diagnosis of viral meningitis. Both methods identified vaccination 15-35 days before onset as a significant risk factor and therefore indicative of a causal association. With both, half the aseptic meningitis cases identified in children aged 12-24 months were vaccine-associated with onset 15-35 days after vaccine. This study confirmed that the true risk was substantially higher than suggested by case reports from pediatricians, probably about 1 in 11,000 doses [17].

Furthermore, in Japan, at least 311 meningitis cases suspected to be vaccine-related were identified among 630,157 recipients of the measles-mumps-rubella trivalent (MMR) vaccine. These cases were identified based on the notification of cases and the testing of mumps viruses isolated from cerebrospinal fluid for their relatedness to the vaccine by nucleotide sequence analysis [18].

Also, the Institute of Medicine of the United States of America examined putative serious adverse consequences associated with administration of diphtheria and tetanus toxoids, measles, mumps, and measles-mumps-rubella vaccines, oral polio vaccine and inactivated polio vaccine, hepatitis B vaccines, and Haemophilus influenzae type B (Hib) vaccines. The committee spent 18 months reviewing all available scientific and medical data from individual case reports (published and unpublished) to controlled clinical trials.

The committee found that the evidence favored acceptance of a causal relation between diphtheria and tetanus toxoids and Guillain-Barre syndrome and brachial neuritis; between measles vaccine and anaphylaxis; between oral polio vaccine and Guillain-Barre syndrome; and between unconjugated Hib vaccine and susceptibility to Hib disease. The committee also found that the evidence established causality between diphtheria and tetanus toxoids and anaphylaxis; between the measles vaccine and death from measles vaccine-strain viral infection; between measles-mumps-rubella vaccine and thrombocytopenia and anaphylaxis; between the oral polio vaccine and poliomyelitis and death from polio vaccine-strain viral infection; and between the hepatitis B vaccine and anaphylaxis [19].

In addition to the MMR vaccine, Alexa received the varicella vaccine at the age of 12 months when she was suffering from immune depression. The following is a list of the most frequently reported adverse reactions in children ages 1 to 12 years who received the varicella vaccine: upper respiratory illness, cough, irritability, nervousness, fatigue, diarrhea, loss of appetite, vomiting, otitis, diaper rash/contact rash, headache, teething, malaise, abdominal pain, skin rash, nausea, eye complaints, chills, lymphadenopathy, malagia, lower respiratory illness, allergic reactions (including allergic rash and hives), stiff neck, heat rash, arthralgia, eczema/dry skin/dermatitis, constipation, and itching. Furthermore, in a study consisting of 8,827 children who received the varicella vaccine, fever (102°F/38.9ºC) developed in 14.7% between 0-42 days [13, page 1910].

Three hundred sixty-five infants were inoculated with Haemophilus influenzae type B (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%) [13, page 2318]. The Hib vaccine also caused diabetes in some children. Classen and Classen analyzed data from a Hib vaccine trial and identified clusters of extra cases of insulin dependent diabetes (IDDM) caused by the vaccine that occurred between 36 and 48 months post-immunization [20].

Furthermore, approximately 116,000 children in Finland were randomized to receive 4 doses of the Hib vaccine beginning at 3 months of age or one dose starting after 24 months of age. A control-cohort included all 128,500 children born in Finland in the 24 months prior to the Hib vaccine study. The difference in cumulative incidence between those receiving 4 doses and those receiving 0 doses is 54 cases of IDDM/100,000 (P =0.026) at 7- year (relative risk = 1.26).

Most of the extra cases of IDDM appeared in statistically significant clusters that occurred in periods starting at approximately 38 months after immunization and lasting approximately 6-8 months [20]. In a second study, distinct rises in the incidence of IDDM in children occurred 2-4 years following the introduction of the MMR and pertussis vaccines [21].

Adverse reactions to IPV, DTaP, and Hepatitis B vaccines have also reported in children. Two hundred and eleven two month-old infants were vaccinated with IPV and DTaP and some infants developed systemic adverse reactions at 24 hours post-inoculation that included fever >102.2°F (39.0ºC) (0.5%), irritability (24.6%), tiredness (31.8%), anorexia (8.1%), and vomiting (2.8%) [13, page 2335].

The 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 >100.4ºF (38ºC) in 7% of total, irritability in 36.3%, drowsiness in

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34.9%, loss of appetite in 16.5%, vomiting in 5.8%, and crying for 1 hour or more in 3.9% [13, page 3063].

In addition, 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 non-fatal 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 [22].

The whole-cell DTP vaccine has also been associated with acute encephalopathy [13]. 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 the DTP vaccine 7 days preceding the 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 the 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.

The database from the 1994 National Health Interview Survey (NHIS) in the USA that included 6,515 children less than six years of age who received the hepatitis B vaccine was 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 [23].

The above selected studies clearly show that the vaccines given to Alexa cause serious health problems, even death in healthy infants and older children. The risk of developing adverse reactions to vaccines usually increases in children with pre-existing health problems. Alexa had chronic health problems at 10 months of age prior to her vaccination with IPV, hep B, MMR, and varicella (Table 3).

Alexa developed adverse reactions to these vaccines that led to her cardiac arrest on November 16, 1999. These included loss of appetite, diarrhea, teething, upper respiratory tract infections, ear infection, malnutrition, acute pancreatitis, osteomyelitis, and vitamin K deficiency. Alexa's adverse reactions to vaccines are consistent with those reported in the medical literature and the Physicians' Desk Reference [12-23]. However, the medical examiner and the other State's expert witnesses did not consider the adverse reactions of vaccines given to Alexa in their evaluations of this case.

Section II. Alexa's Cardiac Arrest and Hospitalization on November 16-18, 1999: Review of Clinical Events and Treatment History

II-A. Description of events at Kathleen Butcher's house on 11-16-1999

Alexa's mother brought Alexa to Kathleen Butcher's house at approximately 0730 on November 16, 1999. There were a total of eight children under the age of five years in Kathleen's care on November 16. Kathleen became a State licensed home day-care provider in 1996. She had no criminal record in Maryland and was approved as a home day-care provider after a rigorous routine investigation and training by the State Department of Human Resources. Kathleen cared for Alexa approximately three days per week for a total of thirteen months. On November 11, 1999, Alexa was a 15 month-old toddler. [24].

Kathleen fed Alexa breakfast in the morning and she appeared normal. At approximately 1215, she placed Alexa in a highchair to give her lunch and fed her lunch consisting of pieces of chicken, potatoes, and string beans that were provided by her mother. Kathleen told the police that while Alexa was eating lunch, she started to get sleepy, appeared to be whitish, and dozed for about five minutes. In about fifteen to twenty minutes after beginning her meal, Kathleen took Alexa out of the highchair, wiped her hands, and took her with another child upstairs to take a nap [24, page 239].

At approximately 1245, Kathleen checked on the two toddlers and noticed that Alexa was not breathing and unresponsive. She took Alexa out of the playpen, brought her downstairs to the foyer, called 911, and began performing CPR [25]. Kathleen was trained to do CPR and she did it correctly as stated by the police officers, who arrived at her house. However, Kathleen was emotionally upset and crying at that time. Kathleen told the police that Alexa was congested for a month and teething [24].

Table 4. Compositions of vaccines administered to Alexa 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 μg pertussis toxin (PT); 25 mcg filamentous hemagglutinin (FHG; 8 mcg pertacin; 2.5 mg 2-phenoxyethanol; 4.5 mg sodium chloride; and 0.1 mg formaldehyde.
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.
Haemophilus Influenzae (Hib)	Each 0.5 mL dose contains (0.4% sodium chloride) contains 10 mg of purified Haemophilus capsular polysaccharide.
Hepatitis B	Each 0.5 mL dose contains 0.25 mg aluminum; 10 μg of hepatitis B antigen; 4.5 mg sodium chloride; 0.49 mg disodium phosphate dihydrate; and 0.35 mg sodium dihydrogen phosphate dihydrate.
Measles, Mumps and Rubella (MMR)	Each 0.5 mL contains no less than the equivalent of 1,000 TCID50 (tissue culture infectious doses) of the U.S. Reference Measles live virus; 20,000 TCID50 Mumps live virus; and 1,000 TCID50 of the U.S. Reference Rubella live virus. The three live viruses are mixed before being lyophilized. It also contains additives.
Varicella virus vaccine	Each 0.5 mL dose contains a minimum of 1350 PFU (plaque forming unit) of Oka/Merck varicella virus and additives.

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II-B. Treatment given to Alexa by the Rescue Team on November 16, 1999

The Howard County Fire and Rescue Team (HCFRT) logged in a call from Kathleen Butcher's residence at 1257 on November 16 and they arrived at her home at about 1304 [6]. They saw Kathleen was performing CPR on Alexa in a correct manner. Alexa was in full cardiopulmonary arrest and she was not breathing. The HCFRT initiated Pediatric Advanced Life Support and rushed Alexa to the Emergency Room at Laurel Regional Hospital. En route they intubated her to assist in ventilation. They also provided Alexa with three rounds of cardiac defibrillation because they found that she suffered from ventricular fibrillation based on an EKG test.

At that time, the HCFRT did not see any sign of obvious injuries or trauma on Alexa's head or the rest of her body. In addition, the police officers who arrived at Kathleen's home did not see any sign of injury or trauma on Alexa [26, page 12]. The police also examined Kathleen's hands and they did not see any sign of injury or blood spots. Furthermore, the police inspected the walls and the floors of the foyer at Kathleen's home. They did not find any blood or hairs on the floor or the walls nor did they find evidence of damage on the walls [26, page 12].

II-C. Clinical findings and treatments given to Alexa at Laurel Regional Hospital

Alexa arrived at the ER in Laurel Regional Hospital at about 1322 on November 16. She was in critical condition and without spontaneous cardiac function. She was unresponsive to pain and had fixed and dilated pupils. Her core body temperature was quite low at 94.7°F (34.8ºC).

Laboratory studies of blood collected at 1419 revealed that she had an elevated serum glucose level of 504 mg/dL, high anion gap of 26 mmol/L (normal range: 5-20 mmol/L), potassium of 3.7 mmol/L, high platelet count of 659,000/μL (indicative of bone marrow hyperplasia), low hemoglobin of 8.3 g/L and hematocrit of 26.8%, low CO_2 level of 9 mmol/L, and a high white blood cell count of 31,200/μL (normal range: 6,000-14,000/μL). These values indicate that Alexa was suffering from diabetes, metabolic acidosis, anemia, and an acute bacterial infection [8].

Alexa was treated with a high therapeutic dosage of epinephrine (0.5 mg) given intravenously in four injections during an 18-minute period in order to stimulate her heart and to increase her blood pressure (Table 5). Bleeding (intracerebral, subdural and/or subarachnoid hemorrhage) is one of the serious adverse reactions of epinephrine, even when given at a low dosage level of 0.05 mg subcutaneously, which is 10% of the dosage of epinephrine given to Alexa. Furthermore, Alexa received fluid, atropine, dopamine, and sodium bicarbonate by intravenous route as shown in Table 5.

Alexa also received three rounds of cardiac defibrillation in the ER in addition to the three rounds given by the Howard County Fire and Rescue Team (HCFRT). The HCFRT's Captain and a physician who treated Alexa said that the electrical shock used to defibrillate Alexa's heart causes hyper-extension or arching of the back of the child. The body usually jerks up by an inch or two and occasionally the body will momentarily jump [25, page 54]. Captain Richards also stated that the defibrillation pads can occasionally leave marks on the skin that resemble a burn resulting from the electrical arch [25, page 59].

At about 1420, Alexa was still in a coma and unresponsive. Due to Alexa's life-threatening condition, she was transferred by Medestar Helicopter on November 16 at 1406 to Children's National Medical Center (CNMC) in Washington, DC and arrived at about 1414 [9]. The total time spent to resuscitate Alexa up to this point was about 20-30 minutes. During this time her brain suffered from severe ischemia and hypoxia that caused brain edema and the death of nerve cells.

Table 5: Treatments given to Alexa at Laurel Regional Hospital on November 16, 1999

Time	Treatment and Dosage	Mechanism of Actions
1328	0.1 mg epinephrine (IV bolus)	Sympathomimetic drug
1333	0.1 mg epinephrine (IV bolus)	
1337	0.2 mg epinephrine (IV bolus)	
1346	0.1 mg epinephrine (IV bolus)	
1332	0.2 mg atropine (IV bolus)	Parasympatholytic drug
1333	0.2 mg atropine (IV bolus)	
1440	0.2 mg atropine (IV bolus)	
1335-9	60 mL NS Fluid (IV bolus)	To treat dehydration
1345	10 mEq Sodium bicarbonate	To treat acidosis
1409	60 mg Dopamine/100 mL NS (IV) @ 2.5 mL/hr	Sympathomimetic drug

II-D. Clinical findings and treatments given to Alexa at CNMC on November 16

Alexa arrived at the Children's National Medical Center (CNMC) by helicopter on November 16 at about 1414. She was intubated and her body weight was 8.9 kg (19.6 lb). At 1420, Alexa's blood pressure was 65/31 mm Hg. She had a pulse rate of 138/minute, and a low temperature of 94.7°F (34.8ºC) (Table 6).

Dr. James Chamberlian examined Alexa at the CNMC at about 1449 and found that she had equal breath sounds, no rales or wheezes. She was flaccid with no response even to pain. The pupils were fixed at about 8 mm. Her heart sounds were normal and without murmur, gallop, or rub. However, she had weak distal pulses and the EKG test showed prolonged QT waves [10].

Dr. Chamberlian observed a boggy swelling of the scalp at the occipital and the right temporopartial areas. However, he did not see any other external signs of injuries on Alexa's head or the rest of her body that indicated that Alexa had suffered from trauma. Dr. Chamberlian also examined Alexa's abdomen and found it soft and non-distended. He did not detect any abnormal mass or organomegaly [10].

Blood analysis performed at about 1507 showed that Alexa had low blood pH of 6.92, bicarbonate level of 8 mmol/L, hemoglobin of 8.7g/dL, and hemotocrit value of 26.2%. It also showed the white blood cell count (40600/μL), platelet count (737000/μL), and prothrombin time (33.3 seconds) were elevated (Tables 8-11). A stained blood smear was examined at 1507 and revealed that the neutrophil contained toxic granules. Also there were increases in the number of immature neutrophil

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(bands and meta) [10]. These changes are compatible with the presence of an acute bacterial infection.

Alexa was treated with high therapeutic antibiotics (Cefotaxime and Vancomycin) to fight the bacterial infection between 1504 and 1540. Her white blood cell count was reduced from 40600/μL (at 1507) to 13800/μL (at 2207) by the use of antibiotics. Alexa was also treated with dopamine at 15 μg/kg/minute and epinephrine at 0.6 μg/kg/minute to increase cardiac output and blood pressure. Her blood pressure was low at 50's/20's. Alexa was also given sodium bicarbonate to treat her acidosis (Table 7).

A CT scan of Alexa's head region taken at 1615 showed diffuse and severe edema of the cerebrum with obliteration of the cortical sulci, small ventricles and basilar cisterns. Abnormal densities were seen in the interpenduncular cisterns that were consistent with the presence of focal subarachnoid hemorrhage. A marked amount of swelling and hemorrhage was also seen in the scalp soft tissues primarily on the right side at the vertex. In addition, complete opacification of the middle ear and mastoid was observed bilaterally. No displaced skull fracture was seen in this scan and the CT scan taken at 1625 to check the base of the skull and the cervical spine [10].

The CT scan taken of Alexa's abdominal region at 1639 showed an abnormal pattern of vascular enhancement in that the major arterial vessels appeared moderately bright, but small in size. This was consistent with low perfusion rate and reduced intravascular volume. There was also diffuse enhancement of the bowel wall and multiple loops of the bowel contained fluid. This finding was also compatible with a low perfusion state. Free abdominal and intraperitoneal fluid was also present. However, no evidence of discrete solid parenchymal organ injury or hollow viscous perforation was identified that suggested the presence of contusion or laceration of the internal organs. Images of the chest demonstrated some minor left lower lobe air space disease, most compatible with atelectasis [10].

A blood analysis performed at 2207 revealed that Alexa had an elevated blood glucose level of 624 mg/dL, blood pH of 7.58, bicarbonate level of 26 mmol/L, anion gap of 21, alkaline phosphatase of 143 U/L, ALT of 198 U/L, AST 470 U/L, prothrombin time (PT) of 20.8 seconds, partial thromboplastin time (PTT) of >100 seconds, and fibrin degradation product (FDP) of >20 (Tables 8, 11, 12). It also showed that Alexa had a low serum potassium level of 3.0 mmol/L, hemoglobin level of 7.5 g/dL, hematocrit value of 26%, fibrinogen level of 115 mg/dL, and red blood cell count of 2.56 x 10^6/μL (Tables 8, 9). The white blood cell count (13800/μL), platelet count (294000/μL), and serum albumin (3.2 g/dL) were within the normal range.

Alexa was given fresh frozen plasma (FFP) by IV to reduce bleeding, red blood cells to treat anemia, and epinephrine and dopamine to increase cardiac output and blood pressure (Table 7). She was also given cefetaxime, vancomycin, and nitidine to treat bacterial and yeast infections. Furthermore, she was treated with sodium bicarbonate by IV infusion. However, at that time her blood pH was critically high (7.58) and this treatment was not medically justified (Tables 7, 8).

Table 6. Alexa's vital indicators on November 16-18, 1999

Date and Time	Heart Rate/minute	Blood pressure mm Hg	Temperature °F (°C)
Nov. 16			
1420	138	65/31	94.7 (34.8)
1453	104	62/32	
1520	115	72/43	
1600	129	86/55	
1615	131	60/37	
1625	135	79/43	
1700	138	61/33	
1920	176	79/43	
2100	179	104/64	
2207	160	106/64	99.5 (37.5)
2330	195	119/80	
Nov. 17			
0160	190	104/50	
0600	161	110/77	
0700	121-197	49-124/33-98	
1355		102/62	98.6 (37.0)
Nov. 18			
0500	128	103/50	

Table 7. Treatments given to Alexa at CNMC on November 16, 1999

Time	Treatment & Dosage	Mechanism of Actions
1510	Dopamine IV infusion (15 μg/kg/min.)	Sympathomimetic drug
1510	Bicarbonate 10 mEq	To treat acidosis
1510	Cefotaxime 500 mg	Antibiotic
1510	Vancomycin 150 mg	Antibiotic
1527	Epinephrine infusion (0.6 μg/kg/min.)	Sympathomimetic drug
1540	Bicarbonate 20 mEq	Treatment for acidosis
1600	Gastrografin 10 cc/300 cc Fluid PO	Contrast Medium
1900	Albumin 5% (100 cc/Kg)	Protein replacement
1925	Cefotaxime 100 mg/8 hr	Antibiotic
1925	Vancomycin 100mg/6 hr	Antibiotic
1925	Fosphenytoin, IV (25 mg PE/0.5 mL)	Anticonvulsant
2000	Nitidine HCl (20 mg/8 hr)	Treatment yeast infection
2000	Bicarbonate 10 mEq	Treatment for acidosis
2200	Vancomycin (100 mg/6 hr)	Antibiotic
2200	Insulin (2 mL)	Treatment for diabetes
	Epinephrine (1 μg/kg/min)	Sympathomimetic drug
	Sodium bicarbonate 10 mEq	Treatment for acidosis
2207	Dopamine IV infusion (15 μg/Kg/min.)	Sympathomimetic drug
2207	Fresh frozen plasma (FFP)	To reduce bleeding
2207	Red blood cells 300 cc	Treatment for anemia
2207	Bicarbonate 20 mEq	Treatment for acidosis
2210	Cefotaxime (500 mg), IV	Antibiotic
2300	Zantac 20 mg IV	Treatment for gastric ulcer
2400	Cefotaxime 500 mg	Antibiotic
2400	Fosphenytoin, IV (25 mg PE/0.5 mL)	Anticonvulsant

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Table 8. Alexa's Metabolic Parameters on November 16-18, 1999

Date & Time	Blood PH	Glucose (mg/dL)	Bicarbonate (mmol/L)	Anion Gap (mmol/L)	Potassium (mmol/L)
Nov. 16					
1419	-	504H	-	26H	3.7
1459-1507	6.92L	-	8L	-	4.3
1528	7.06L	-	10L	28H	-
1855	7.38	-	11L	-	3.7
2207	7.58H	624H	26H	21H	3.0L
2341	7.58H	-	18	-	-
Nov. 17					
0048-56	7.50H	521H	17L	-	2.7L
0210	-	475H	-	-	-
0317	7.50H	546H	-	-	3.5
0400-29	-	537H	22	17	3.4
0523	7.56H	453H	-	-	2.5L
0639-43	-	507H	-	-	-
0803-18	-	405H	-	-	-
0903	-	322H	-	-	-
1000-4	7.52H	283H	19	-	2.2L
1156-9	7.47H	170H	22	-	2.7L
1359-409	7.11-7.43		23	-	3.2L
2005-42	-	162H	11L	9 L	4.4
2144	-	188H	-	-	4.4
Nov. 18					
0004	-	154H	23	-	3.9
0219	-	-	18	-	3.1L
0356-430	-	199H	16	9L	3.4
0529	-	200H	18	-	-
0803-29	-	171H	22	-	4.0
0923-51	-	-	21-2	-	4.0
1143-7	-	148H	22	-	4.2
1418-29	7.41	136H	21	-	3.7
1840	7.46	-	21	-	3.5
Normal Range:	**7.35-7.45**	**57-117 (mg/dL)**	**18-25 (mmol/L)**	**10-18 (mmol/L)**	**3.3-4.6 (mmol/L)**

Table 9. Alexa's white blood cell counts on November 16-18, 1999

Date & Time	White Blood Cell x 10^3/ μL	Segmented Neutro.%	Band Neutro.%	Meta Mylocy.%	Lymphocyte %
Nov. 16					
1419	32.1H				
1507	40.6H*	27	37	5	27
2207	13.8	43	24	3	23
Nov. 17					
0400	18.6H	69	10		14
2042	17.7H	52	13		18
Nov. 18					
0430	18.3H	74	18	5	
1421	23.4H	76	12	4	
Normal Range:	**6.0-17.0**	**13-42**	**0-1**	**0-1**	**35-67**

*Toxic granules + 2 and left shift with bands and metas were observed. These are indicators for acute bacterial infections.

Table 10. Alexa's hematology values on November 16-18, 1999

Date & Time	Red blood cell x 10^6/μL	Hemoglobin (g/dL)	Hematocrit %	Platelet x 10^3/μL
Nov. 16				
1419	3.1L	8.3L	28.8L	659H
1507	3.35L	8.7L	26.2L	737H
2207	2.56L	7.5L	22.2L	294
Nov. 17				
0400	4.33	13.4	38.8	214
2042	3.16L	9.5L	22.2L	117L
Nov. 18				
0430	2.53L	7.7L	22.2L	114L
1421	2.21L	6.7L	19.2L	118L
Normal Range:	**3.7-5.3**	**10.5-13.3**	**33-39**	**150-450**

Table 11. Alexa's coagulation parameters on November 16-19, 1999

Date & Time	PT* (sec.)	PTT (sec.)	INR	Fibrinogen (mg/dL)	Platelet x 10^3/μL
Nov. 16					
1507	33.3H	-	8.94H	-	737
2207	20.8H	>100.0H	3.24H	115L	294
Nov. 17					
0818	14.1H	39.9H	1.43H	269	-
2042	13.9H	36.9H	1.37H	598H	117
Nov. 18					
0430	15.0H	40.2H	1.61 H	604H	118
Normal Range:	**9.8-12.5**	**25.6-35.0**	**0.76-1.29**		**150-450**

*PT: prothrombin time; PPT: partial thromboplastin time; -: not available

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Table 12. Alexa's serum enzymes levels on November 16-18, 1999

Date & Time	Amylase (IU/L)	Lipase (IU/L)	ALK* (IU/L)	AST (IU/L)	ALT (IU/L)
Nov. 16					
1419					
2207			143	470H	198H
Nov. 17					
0048	1026H	358H	92L	505H	168H
0400			92L		
2042			171		
Nov. 18					
0430	243H		254H	126H	124H
1418	114H		333H		
Normal Range:	**<100**	**15-150**	**129-191**	**20-60**	**5-60**

*ALK: Alkaline phosphotase; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase

II-E. Clinical findings and treatments given to Alexa at CNMC on November 17

Blood analysis performed at 0050 showed very high levels of amylase (1026 IU/L) and lipase (358 IU/L) enzymes in serum (Table 12). The blood sample analyzed at 210 revealed that Alexa still had a high blood glucose level of 475 mg/dL. She also had a high blood pH of 7.58 and a low serum potassium level of 2.7 mmol/L (Table 10). At about 400, Alexa's platelet count (214,000/μL) and white blood cell count (18600/μL) reached normal levels. Her serum total bilirubin was slightly elevated (1.7 mg/dL). At 0818, the PT and PTT were reduced to 14.1 and 39.9 seconds, respectively and her red blood cell count returned to normal (4.33 x 10^6/μL) because she was treated with FFP and red blood cells by IV infusion (Table 13).

The CT scan taken at 1627 of Alexa's head region was compared with the previous CT taken on November 16. It showed interval increases in the severity of the global edema involving both the supra- and infratentorial structures. There was minimal definition of the cortical mantle and basal ganglia due to diffuse edema. There was an interval development of bilateral transtentorial herniation and probably tonsillar herniation.

There was also linear density along the left aspect of the tentorium which may represent a small amount of blood along the tentorium. However, the previously demonstrated subarachnoid blood in the interpenduncular cistern was no longer clearly noticeable. The extracranial structures were notable for interval increase in the subgaleal hematoma, which became bilateral and diffuse and it was more marked on the right side.

A CT scan of the abdomen and pelvis taken at 1627 showed a mild amount of periportal edema in the liver in addition to fluid in the gallbladder fossa with enhancement of the gallbladder wall mucosa and the bowel wall. The abdominal cavity also contained a moderate amount of fluid. The spleen, kidneys, adrenal glands, and the pancreas appeared normal. A limited evaluation of the lung bases demonstrated a consolidation of the posterior aspect of the right lower lobe.

A radiological bone survey taken at 1713 demonstrated an erosion of the inferior endplate of the T-10 vertebrae which appeared irregular but well defined. This may represent a lytic bone lesion but osteomyelitis of the vertebral body was not ruled out. The disk spaces were preserved. The lesion in T-10 did not have the expected appearance of a fracture. Additionally, no fracture or dislocation was noted in this bone survey.

Table 13. Alexa's treatment at CNMC on November 17, 1999

Time	Treatment & Dosage	Mechanism of Actions
0010	Insulin 1 U/IV	Treatment for diabetes
0100	Fosphenytoin (IV) (25 mg pE/0.5 mL)	Anti-epileptic drug
0240	KCl (mEq IV)	Treatment for hypokalemia
0240	Insulin (1 unit/cc)	Treatment for diabetes
0400	Vancomycin (100 mg each 6 hr)	Antibiotic
0700	Dopamine IV infusion (15 μg/Kg/min.)	Sympathomimetic drug
0700	Epinephrine infusion (0.6 μg/Kg/min.)	Sympathomimetic drug
0700	Albumin 5% 100 cc/kg	To correct protein loss
0700	FFP 300 cc	To reduce bleeding
0700	KCl	Treatment for Hypokalemia
0700	Insulin drip 0.2 U/Kg	
0800	Cefotaxime 500 mg	Antibiotic
0800	Nitidine HCl (20 mg per 8 hr)	Treatment for yeast infection
1000	Vancomycin (100 mg/6 hr)	Antibiotic
1000	Fosphenytoin (25 mg PE/0.5 mL)	Anti-epileptic drug
1100	Insulin 1U/cc in Ns 6 cc	Treatment for diabetes
1100	Albumin 25%	
1100	Vasopressin 1 U/100 mL NS	Anti-diuretic
1120	Dopamine IV infusion (15 μg/kg/min.), total 300 mg	Sympathomimetic drug
1120	Epinephrine infusion (0.35μg/kg/min.), total 6 mg	Sympathomimetic drug
1130	Heparin (1000 unit/500 mL) 2 mL per hour	Anticoagulant
1130	10 mEq KCl/500 mL	
1600	Nitidine HCl (20 mg per 8 hr)	Treatment for yeast infection
1600	Cefotaxime 500 mg	Antibiotic
1600	Vancomycin (100 mg each 6 hr)	Antibiotic
1600	Gastrograffin 10 cc in 300 cc fluid	
2200	Fosphenytoin (25 mg)	Anticonvulsant
2255	Epinephrine 0.05 μg/kg/min	Sympathomimetic drug
2255	Insulin	Treatment for diabetes
2400	Nitidine HCl (20 mg per 8 hr)	Treatment for yeast infection
2400	Cefotaxime 500 mg	Antibiotic

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II-F. Clinical findings and treatments given to Alexa at CNMC on November 18

A single view of the chest taken at 0100 demonstrated some linear basilar atelectasis in the right lung. There was a bulbar deformity of the posterior aspect of the 8th rib on the left side that suggests the possibility of a healing fracture. At 0430, Alexa's PT (15.0 seconds), PTT (40.2 seconds), glucose level (199 mg/dL), and white blood cell count (18300/μL) were still elevated.

In addition, the fibrinogen level in serum was increased to 604 mg/dL from an initial level of 115 mg/dL. Alexa's red blood cell count (2.53 x 10^6/μL), hemoglobin level (7.7 g/L), hematocrit (22.2%), and platelet count (114000/μL) were reduced significantly from the levels observed on November 17. These values indicate that Alexa had experienced a major bleeding episode within the previous 24 hours.

Alexa was treated with dopamine, epinephrine, potassium, and antibiotics (Table 14). She was officially pronounced dead at 1020 on November 18. The organ harvesting procedure, description of abnormal findings, and the adverse reactions to heparin are described in Section III.

Table 14. Alexa's treatment at CNMC on November 18, 1999

Time	Treatment and Dosage	Mechanism of Actions
0420	Insulin (1.5 U/cc), 15 cc	Treatment for diabetes
0500-1900	Dopamine IV infusion (15 μg/Kg/min.)	Sympathomimetic drug
0500-1900	Epinephrine infusion (1.0 μg/Kg/min.)	Sympathomimetic drug
1530	Ticarcillin Clavulanate 500 mg IV/ each 6 hr.	Antibiotic
1800	20 mEq KCl/L IVF	Treatment for hypokalemia

Section III. Organ Harvesting Procedure, Abnormal Findings, Heparin Dose Given, and Adverse Reactions

III-A. Organ's harvesting procedure and abnormal findings

Alexa was pronounced brain dead at 1020 on November 18. The Chief Medical Examiner for the District of Columbia gave his permission to harvest Alexa's organs for donation. The organ harvesting procedure was performed by Dr. Carlos Fernandez-Bueno and Dr. Johann Johnson [10]. They started at 0355 on November 19 at the Children's National Medical Center in Washington, D.C.

Alexa was given anesthetic, ringer lactate solution, mannitol (10 g/IV), dextrose 50% (100 ml over 10 min), lasix (30 mg IV), and 5000 IU of heparin (IV). Alexa's body weight was 8.9 kg and the resulting heparin dose was 562 IU/Kg. The recommended therapeutic dose of heparin for infants and children is 50 IU/Kg IV. The dose of heparin given to Alexa prior to organ harvesting was 11.2 times the therapeutic dose for a child similar to Alexa's body weight [13, page 3306]. The Medical Examiner and the State's other expert witnesses clearly did not take into consideration Alexa's adverse reactions to heparin when they evaluated this case. Heparin usually causes very serious bleeding even when given at the therapeutic dose level as described below (II-C).

Furthermore, the surgeons opened Alexa's abdominal cavity and found (1) bloody intraperitoneal fluid, (2) severe induration of the root of mesentery with inflammatory process and fibrin exudates, (3) hematoma of the right upper omentum area between the proximal transverse colon and second and third portions of the duodenum portal, and (4) inflammation in the area of the infrahepatic vena cava and the upper portion of the right kidney [10]. Photographs were obtained of these lesions as requested by the medical examiner and sent to his office, but unfortunately, they were lost and the medical examiner never saw them.

The surgeons observed no evidence of perforation in the gastrointestinal tract that indicated trauma. In addition, the liver and kidneys were removed, examined, and appeared normal. No evidence of parenchymal damage or damage to their vascular supplies was present that could indicate that the abdominal region had been struck by blunt trauma [27, page 27]. Furthermore, Dr. Carlos Fernandez-Bueno did not notice any bruises on Alexa's back or the abdominal region [27, page 69]. After the removal of organs (heart, liver, kidneys, adrenals, spleen, and a large portion of the diaphragm), Alexa's body was transported to the morgue and kept in the cooler until the time of autopsy at 1300 on November 19, 1999.

III-B. Microscopic examination of abnormal tissue taken from Alexa's mesenteric

On November 19, the surgical team removed a tissue sample from the mesenteric near the iliac artery that appeared abnormal when microscopically examined. It consisted of a single, round to oval fragment of tan, hemorrhagic tissue approximately 0.9 x 0.7 x 0.7 cm. Bisection of this tissue revealed firm white tissue throughout. Half of the sample was frozen and the remainder was embedded in one cassette, sectioned, and stained with H & E [10].

Dr. Dena Selby examined the tissue sample and the H & E stained tissue section on November 22, 1999. The microscopic examination revealed that the abnormal tissue consisted of fibroplasias response with some acute hemorrhage. There was no evidence of malignancy. The medical examiner and the State's other expert witnesses did not even mention or refer to the result of this investigation in their reports or in their court testimonies [10].

III-C Heparin dose given to Alexa and adverse reactions to heparin

Heparin is a heterogeneous group of straight-chain anionic mucopolysaccharides, called glycosaminoglycans, having anticoagulant properties [13]. It inhibits reactions that lead to the clotting of blood and the formation of fibrin clots both in vitro and in vivo, acting on multiple sites in the normal coagulation cascade. Clotting time is prolonged by full therapeutic doses of heparin in most cases. Alexa was given 5000 IU of heparin sodium by IV injection. Taking into consideration Alexa's weight at the time of treatment was 8.9 kg, the resulting effective heparin dose would have been 562 IU/Kg per hour. The Physicians' Desk Reference (PDR) recommends 50 IU/Kg, IV as an initial dose for infants and children. Therefore, the dose of heparin given to Alexa prior to harvesting her organs was *11.2 times* the recommended therapeutic dose. [13, page 3306].

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Hemorrhage can occur at virtually any site in patients receiving heparin. Patients suffering from anemia, unexplained symptoms, and/or those that have low blood pressure are at greatest risk of having serious hemorrhagic events after receiving therapeutic doses of heparin. Alexa had hypotension and her hematocrit was very low (25.3%). Bleeding is the chief sign of heparin over-dosage. Nose bleeds, blood in the urine, or tarry stools may be noted as the first signs of bleeding. Easy bruising or petechial formations may precede frank bleeding [28, page 1287].

The surgical teams who harvested Alexa's organs did not notice any bleeding on Alexa's back and abdomen prior to harvesting the organs. The Medical Examiner observed nine purple contusions in various places on Alexa's back that measured 1/8-3/8 inches in size [29]. The microscopic examination of the skin and the subcutaneous tissues of one of these bruises showed that the bleeding was less than 24 hours old. It is obvious that Alexa's treatment with excessive amounts of heparin caused the fresh bleeding on Alexa's back and other locations described by the medical examiner, which I will explain in Section V-B of this report.

Section IV. Analysis of Clinical Events and Causes That Led to Alexa's Cardiac Arrest, Bleeding, and Edema

Alexa suffered from cardiac arrest and apnea between 1230 and 1245 on November 16, 1999. The clinical data described in Sections II and III clearly shows that her cardiac arrest was triggered by acute pancreatitis and diabetes mellitus and not by violent shaking and blunt trauma as the State alleged. Alexa did not breathe for approximately 30 minutes following her cardiac arrest. As a result, her brain suffered from severe ischemia and hypoxia, which caused severe diffuse edema and nerve damage.

Alexa also suffered from vitamin K deficiency, anemia, acute bacterial infections, and complications of acute pancreatitis and diabetes. These complications included metabolic acidosis, reduction of potassium levels in cardiac muscles and nervous tissues, edema, bleeding, disseminated intravascular coagulation (DIC), acute peritonitis, hypovolemia, cardiac arrest, and apnea. Furthermore, Alexa's treatment with high therapeutic doses of epinephrine during resuscitation, and her treatment with epinephrine and heparin during her hospitalization caused bleeding in the subdura, retina, skin, and other locations.

The medical evidence shows that the vaccines given to Alexa on July 20 (IPV and Hep B) and August 13, 1999 (MMR and Varicella) induced Alexa's illnesses. During that time, Alexa suffered from a poor appetite, poor weight gain, and chronic fungal infection. She was treated with nystatin (an antifungal drug) for six weeks. Children suffering from malnutrition usually have lowered T-cell counts and reduced functions [11]. That made Alexa's response to the administered vaccines inadequate and increased her risk of developing serious adverse reactions to vaccines and infections. Furthermore, the MMR live viruses were received three months earlier than the recommended schedule [1, 11, 13]. Alexa suffered from acute pancreatitis and the mumps virus vaccine was the likely cause.

Additionally, rhinitis, upper and lower respiratory infections, and otitis media have been observed in some children who received MMR and varicella vaccines as I described in Section I-D of this report. The middle ear is connected to the nasopharynx via the eustachian tube. When this tube is blocked, fluid collects in the middle ear and mastoid cavities, providing a culture medium for the bacteria present. Acute otitis media (AOM) or middle ear infection may result. Viral respiratory tract infections that can cause edema of the eustachian tube mucosa, often precede or accompany episodes of AOM [11].

Streptococcus pneumonia and Haemophilus influenza are the major causes of bacterial ear infections in children [11, page 182]. The incidence of pneumococcal bacteremia is relatively high among infants up to 2 years of age. In addition to otitis media, S. Pneumonia and H. Influenza also cause osteomyelitis in children [11, page 871]. Alexa had osteomyelitis of the T-10 vertebrae and it is very likely that it was caused by S. pneumonia and/or H. influenza. Below are detailed descriptions of the pathogenesis and pathology of the clinical events that led to Alexa's cardiac arrest and death in November of 1999.

IV-A. Alexa suffered from acute pancreatitis

On November 16, 1999, Alexa suffered from acute pancreatitis and the mumps vaccine probably caused her illness. The pancreas is a large gland behind the stomach and close to the duodenum that secrets alkaline fluid (pH 8.0) containing about 20 enzymes. These enzymes include amylase, lipolytic enzymes (lipase, phospholipase A, and cholesterol esterase); proteolytic enzymes (trypsin, chymotrypsin, carboxypeptidases, aminopeptidases, and elastase), and ribonucleases [11, page 1741]. These enzymes are needed to perform the major digestive activity of the gastrointestinal tract.

IV-A1. The pathology and symptoms of acute pancreatitis

The pathologic spectrum of acute pancreatitis varies from edematous pancreatitis to necrotizing pancreatitis. The inflammation of pancreas usually causes the release of the digestive enzymes to the adjacent tissues, abdominal and peritoneal cavities, and the blood stream. Activated proteolytic enzymes, especially trypsin, not only digest pancreatic and peripancreatic tissues but also can activate other enzymes such as elestase and phospholipase.

Active enzymes digest cellular membranes and cause proteolysis, edema, interstitial hemorrhage, vascular damage, coagulation necrosis, fat necrosis, and parenchymal cell necrosis. In addition, cellular injury and death result in liberation of activated enzymes. Activation and release of bradykinin peptides and vasoactive substances (e.g., histamine) are believed to produce vasodilation, increased vascular permeability, and edema in distant organs [11, page 1742].

The local and systemic complications of acute pancreatitis may include ascites, massive intraperitoneal hemorrhage, atelectasis mostly of the left lung, hypotension, hypovolemia, nonspecific ST-T changes in electrocardiogram, disseminated intravascular coagulation (DIC), erosive gastritis, gastrointestinal hemorrhage, hyperglycemia, hypoalbuminemia, hypocalcemia, and fat necrosis. Jaundice occurs infrequently and when present, it usually is due to edema of the head of the pancreas compressing the intrapancreatic portion of the common bile duct. Additionally, erythematous skin nodules may occur due to subcutaneous fat necrosis [11, page 1742].

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Shock is not unusual in cases of acute pancreatitis and may result from 1) hypovolemia secondary to exudation of blood and plasma proteins into the retroperitoneal space, 2) increased formation and release of kinin peptides, which cause vasodilation and increased vascular permeability, and 3) systemic effects of proteolytic and lipolytic enzymes released into the circulation.

IV-A2. List of medical evidence that shows Alexa was suffering from acute pancreatitis (AP) on November 16

1) High levels of amylase and lipase enzymes in serum: Acute pancreatitis (AP) can occur suddenly and the diagnosis of AP is usually established by the detection of increased levels of serum amylase and lipase. Values of amylase equal to threefold or more above normal virtually clinch the diagnosis of AP if overt salivary gland disease and gut perforation or infarction are excluded. After 48 to 72 hours, even with the continuing evidence of pancreatitis, total serum amylase values tend to return to normal. However, pancreatic lipase levels may remain elevated for 7 to 14 days. Serum lipase activity increases in parallel with amylase activity, and finding elevated levels of serum lipase increases the diagnostic yield of AP [11, page 1743, 30-33].

On November 16, Alexa's serum amylase level was 1026 IU/L, which was more than 10-fold higher than the normal level of <100. Her serum lipase level (358 IU/L) was also elevated and it was at least threefold higher than the mean normal level (Table 12). Chapoy et al. evaluated nine cases of acute non-traumatic pancreatitis in children and found that the average serum amylase level was 1045 IU/L [30]. In a second study, eleven children (14 months to 9.5 years of age) with acute pancreatitis (AP) and diagnosis of pancreatic injury was suggested by hypera-amylasemia [32]. In addition, thirty-one children (average age 7.9 years, range 2-15; 55% males) who suffered from AP had elevated levels of amylase in serum [34].

2) Hyperglycemia: Alexa's blood glucose level at 1419 on November 16 was 504 mg/dL and was increased to 624 mg /dL at 2207. These levels are about fivefold higher than the normal level (Table 8). Alexa's blood glucose levels continued to be elevated on November 17 and 18 although she was receiving insulin (Tables 13, 14). In acute pancreatitis, hyperglycemia is common and it is due to multiple factors including decreased insulin release, increased glucagons release, and an increased output of adrenal glucocorticoids and catecholamines [11, page 1743]. Chapoy et al. evaluated nine cases of acute non-traumatic pancreatitis in children and found that three of these cases suffered from hyperglycemia [30].

3) Elevated white blood cell count: On November 16, Alexa's white blood cell count was 32,100/μL at 1419 and increased to 40,600/ μL at 1507 [Table 9]. Leukocytosis occurs frequently in cases of acute pancreatitis [11, page 1743]. Toxic granules and Dohle bodies were observed in Alexa's neutrophils which are indicators for bacterial infection. In patients with conditions associated with a systemic inflammation reaction, neutrophil granules may appear larger than normal and stain more darkly, often assuming a dark blue-black color. This has been called toxic granulation [35, page 14]. Dohle bodies are light blue round or oval bodies about 1 to 2 μm in diameter and may be seen in the cytoplasm of neutrophils of patients with infections, burns, and other inflammatory stress. The blue staining is due to Ribonucleic Acid (RNA) [35, page 14].

In addition, in certain inflammatory disorders, morphologic changes occur in blood neutrophils. The best-known alteration is the "shift to the left" which donates the presence of bands, metamyelocytes, and sometimes myelocytes in the blood [35, page 735]. Alexa's white blood cell differential count showed a left shift, which also indicates bacterial infection. At 1507, her band was 37% (normal range: 0-1%) and her metamylocyte was 5% (normal range: 0-1%).

On November 16, Alexa was given four IV injections of epinephrine (0.5 mg), starting at 1328 and the last injection was given at 1346. The treatment with epinephrine is known to cause an increase in the white blood cell count (WBC) due to a shift of cells from the marginated to the circulating pool. However, the increase in the WBC due to epinephrine is not associated with the appearance of toxic granules and Dohle bodies in the neutrophil or the left shift observed in Alexa's case [35, page 755]. In addition, a peak leukocytosis usually occur in 5 to 10 minutes following administration of epinephrine that rarely last more than 20 minutes. Alexa's white blood cell count was found to be 32,100/ μL at 1419 which was 31 minutes following the last epinephrine treatment.

Furthermore, Alexa's white blood cell count increased to 40,600/ μL at 1507 and no epinephrine was given to Alexa between 1419 and 1507. Alexa was also treated with epinephrine by IV infusion at 0.6μg/kg/min at 1527 and by 2207 she received a very high dose of epinephrine (2.13 mg). However, Alexa's white blood cell count (WBC) was reduced from 40,600/ μL at 1507 to 13,800/μL at 2207. The reduction in Alexa's WBC was caused by her treatment with high therapeutic doses of antibiotics (Cefotaxime and Vancomycin) at 1507 (Table 7). Neutrophils disappear from the circulation with a half-time of 6.7 hours and this explains the reduction in WBC observed at 2207 by her treatment with antibiotics [35, page 755].

Additionally, Alexa was treated with high doses of epinephrine on November 17 and 18 and her white blood cell counts did not go up (Tables 9, 12, 13). These data indicate that Alexa's white blood cell counts were elevated not as a result of her treatment with epinephrine but because she had an acute bacterial infection.

4). Hypovolemia, inflammation, hypotension, and edema: A CT scan of the abdominal region taken at 1639 on November 16 showed that Alexa was suffering from hypovolemia with the presence of free abdominal and intraperitoneal fluid. Multiple loops of the bowel were also filled with fluid. In addition, the CT scan of the abdominal region taken one day later (at 1627 on November 17) showed a mild amount of periportal edema in the liver without any focal mass. There was also fluid in the gallbladder fossa with enhancement of the gallbladder wall mucosa and the bowel wall which was indicative of a severe hypotensive episode. The abdominal cavity also contained a moderate amount of fluid.

Furthermore, lesions of acute pancreatitis were also described by the surgeons who opened Alexa's abdominal cavity on November 19 at 0400. They found bloody intraperitoneal fluid, severe induration of root of mesentery with inflammatory process and fibrin exudates, hematoma of the right upper omentum area between the proximal transverse colon and second and

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third portions of the duodenum portal, and inflammation in the area of the infrahepatic vena cava and the upper portion of the right kidney. Microscopic examination of abnormal tissue taken from the mesentery on November 19 showed fibroplasias responses with some acute hemorrhage [10].

These inflammatory changes described above were caused by the release of digestive enzymes (amylolytic, lipolytic, and proteolytic) from the pancreas to the abdominal cavity and not as a result of trauma as the Sate's expert witnesses claimed. Pancreatic exudates containing toxins and activated pancreatic enzymes permeates the retroperitoneum and the peritoneal cavity, inducing a chemical burn and increasing the permeability of blood vessels. It causes extravasation of large amounts of protein-rich fluid from the systemic circulation into "third spaces," producing hypovolemia and shock.

In addition, the activated enzymes and toxins enter the systemic circulation and increase capillary permeability throughout the body. They may also reduce peripheral vascular tone, thereby intensifying hypotension. Circulating activated enzymes may damage tissue directly (i.e., phospholipase A_2 is thought to injure alveolar membranes of the lungs) [33]. Chapoy et al. evaluated nine cases of non-tramatic acute pancreatitis in children and found four of them suffered from shock [30]. Also, Berney et al. evaluated 21 children who had acute pancreatitis and found that one third (33%) had hypovolemic shock-related pancreatitis [36].

Furthermore, Krumberger stated that in the course of acute pancreatitis, the patient may present with signs of hypovolemic shock with associated sequestration of fluid in the peritoneum as a result of inflammatory and mediated responses. Multisystem failure can occur in necrotizing acute pancreatitis as a result of mediators that are activated by the proteolytic enzymes normally produced by the pancreas, and released into the peritoneum by injured cells [31].

No evidence indicating trauma was observed by the CT scans taken on November 16 and 17. There was no evidence of parenchymal organ injury, injuries to their blood supplies, or perforation of the intestine. In addition, the surgeons who harvested Alexa's organs on November 19 and the medical examiner who performed the autopsy excluded the possibility that Alexa's abdomen was struck by blunt trauma. The development of lesions in Alexa's abdominal cavity case was progressive and caused by the release of pancreatic enzymes and mediators.

The hematoma of the right upper omentum observed by the surgeons on November 19 formed because the released pancreatic elestase damaged the blood vessels wall in the mesentery. The clinical evidence presented in Table 10 shows that the bleeding occurred between 0400 and 2042 on November 17. At 0400 Alexa had a normal red blood cell count of 4.33 x $10^6/\mu L$, hemoglobin of 13.4 g/dL, hematocrit of 38.8% and platelet count of 214,000/μL. However, at 2042 the red blood cell count, hemoglobin, hematocrit, and platelet count were reduced to 3.16 x $10^6/\mu L$, 9.5 g/dL, 22.2%, and 117,000/μL, respectively.

Other clinical evidence that demonstrates the changes in Alexa's abdominal cavity occurred in a progressive fashion is the elevation of serum bilirubin levels and the presence of severe induration of root of the mesentery. The total bilirubin level in serum became elevated (1.7 mg/dL) on November 17 due to the pressure on the bile duct as a result of edema. Elevations in serum bilirubin levels have been observed in 15 to 25% of patients suffering from acute pancreatitis because pancreatic edema compresses the common bile duct [33].

The severe induration of root of the mesentery and the inflammation resulted from the actions of the pancreatic enzymes in digesting the fat and connective tissue, proliferation of fibroblasts, and the deposition of calcium. Intraperitoneal saponification of calcium by fatty acids in areas of fat necrosis occasionally occurs in cases of acute pancreatitis [11].

The abdominal computed tomographic (CT) scans of 28 children with acute pancreatitis were reviewed. CT scans were evaluated for pancreatic size and distribution of intra- and extrapancreatic fluid collections. Extrapancreatic fluid was classified as peritoneal, retroperitoneal, mesenteric, or ligamentous. Fourteen children (50%) had complicated pancreatitis associated with fluid collections. Intrapancreatic fluid was identified in only two patients (7%); whereas, extrapancreatic fluid was seen in 14 (50%). Extrapancreatic fluid was most often seen in the anterior pararenal space followed by the lesser sac, lesser omentum, and transverse mesocolon [37].

5) Abnormal changes in the lungs and in the electrocardiogram (EKG): Abnormal changes in lungs have been also observed in patients suffering from acute pancreatitis. Chest x-rays of these patients may reveal atelectasis or a pleural effusion (usually left-sided or bilateral) [33]. In 10 to 20 percent of patients there are pulmonary findings including basilar rales, atelectasis, and pleural effusion, the latter most frequently left-sided [11]. Also, approximately 25 percent of patients suffering from acute pancreatitis have hypoxemia (arterial $PO_2 \leq 60$ mmHg) [11, page 1743].

Alexa's CT scan of the chest taken at 1639 on November 16 demonstrated some minor left lower lobe air space disease most compatible with atelectasis. Alexa's CT scan of the chest taken one day later (at 1627 on November 17) showed a consolidation of the posterior aspect of the right lower lobe [10]. In addition, the medical examiner evaluated tissue sections from the lungs microscopically and found one section was almost totally atelectatic; the other had alternating zones of atelectasis and compensatory emphysema. These sections were taken on November 19 at the time of autopsy [29].

The electrocardiogram (EKG) of patients suffering from acute pancreatitis is occasionally abnormal, showing ST-segment and T-wave abnormalities simulating myocardial ischemia [11, page 1743]. Alexa's EKG taken on November 16 showed prolonged QT waves [10].

The changes in the lungs and heart of people suffering from acute pancreatitis (AP) usually result from the actions of the pancreatic enzymes and the inflammatory mediators that reach these organs via blood circulation [11, page 1742]. Death during the first several days of acute pancreatitis is usually caused by cardiovascular instability (with refractory shock and renal failure) or respiratory failure and occasionally by heart failure. Circulating enzymes and toxins are thought to play a large role in the early death of individuals suffering from AP [33].

6) Elevation of serum enzymes: Alexa's serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels were 470 IU/L (eight-fold higher than normal) and 198 U/L (five-fold higher than normal), respectively on November

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16 (Table 12). The levels of creatine kinase (941 U/L) and alkaline phosphate (333 U/L) became elevated on November 18. These data indicate that Alexa suffered damage in the liver, heart, and muscles resulting from the actions of pancreatic enzymes and other mediators. High levels of serum alkaline phosphotase, aspartate aminotransferase (AST), and lactic dehydrogenase (LDH) have also been reported in patients suffering from acute pancreatitis [11, page 1743].

IV-A3. The common causes of acute pancreatitis in children and the predisposing factors in Alexa's case

Acute pancreatitis (AP) involving children is most commonly caused by non-traumatic causes. Alexa suffered from acute pancreatitis. Causes for her injuries other than trauma should have been considered in this case by both the medical examiner and the State's other expert witnesses. Some of the causes of AP in children include infection with the mumps virus (even in the absence of parotitis), hepatitis B virus, coxsackie B5 virus, Epstein-Barr virus, and influenza B virus, mycoplasma, diabetes mellitus (ketoacidosis), malnutrition, drugs, and idiopathic vasculitis [11, 38]. Chapoy et al. evaluated nine cases of AP and none of those cases were caused by trauma [30].

In addition, a retrospective review of 49 cases of childhood AP (males and females) was evaluated in three Scottish pediatric centers. The most common causes of AP were mumps (39%) and trauma was found to be the cause of AP in only 14% of these cases [39]. Also, 31 children (2-15 years old; 55% males) who suffered from acute pancreatitis (AP) were evaluated. Infection, gallstones, drugs, and idiopathy were the causes found in 80% of these cases and trauma was identified as the cause of AP in only 6.5% [34].

Furthermore, the hospital records of 50 children (2-17 years old) who had acute pancreatitis (AP) were reviewed and trauma was identified as the cause of the disease in only 5 cases (10%). Biliary disease, viral infection, pancreatic duct abnormalities, genetic disease, and idiopathic causes were identified in 90% of the cases [40]. Also, Weizman evaluated 61 patients (1 to 18½ years of age) with acute pancreatitis (AP) and found trauma to be the cause of AP in only 15% of these cases [41].

Additionally, Ziegler et al. evaluated 49 patients with AP, ranging in age from 1 month to 18 years and found trauma was the cause in only one third of these cases. One third of these patients had biliary tract disease as an etiology with nearly half of these being related to underlying hematologic disease, usually sickle cell anemia. Other etiologies were systemic disease (6 patients), congenital anomalies (8 patients), and idiopathy (3 cases). [42]. In a separate study, Berney et al. evaluated 21 children who had acute pancreatitis (AP) and found that trauma was the cause of the disease in only 29% of the patients [36].

Alexa had several predisposing factors that may have caused her pancreatitis: (1) Alexa was given the mumps virus vaccine when she was suffering from depression in the functions of her immune system, especially her T-cells as indicated by her chronic fungal infection and poor weight gain. Children who suffer from poor appetite, poor weight gain, diarrhea, and vomiting as in Alexa's case usually have low T-cell counts and reduced thymus size. The mumps virus probably overcame the immune system and infected the pancreas. (2) The vaccines given to Alexa also cause vasculitis. (3) Alexa suffered from diabetes mellitus (ketoacidosis). (4) Alexa suffered from malnutrition and it increased her risk for bacterial infections.

IV-B. Alexa's diabetes mellitus and the adverse reactions of sodium bicarbonate

The symptoms of diabetes include weight loss, increased susceptibility to infections, hyperglycemia, metabolic acidosis, and coma. Bacterial, viral, and mycotic infections complicate the life of the diabetic in whom hyperglycemia is poorly controlled due to multiple abnormalities in the host response to infections. The leukocyte functions are compromised and immune response is therefore blunted in diabetic patients [11]. Chronic yeast infection can be an early sign. If these infections are very frequent or difficult to clear up with appropriate treatment, diabetes should be considered.

Alexa displayed all of the classical symptoms of diabetes. She suffered from hyperglycemia, anemia, metabolic acidosis, and fungal, viral, and bacterial infections. She had poor weight gain and her weight dropped from the 50% in April of 1999 to less than the 1st percentile on the growth chart in November of 1999 (Table 2). She had a chronic fungal infection for six weeks and she was treated with three consecutive courses of anti-fungal drugs (Section I). Furthermore, on November 16, Alexa suffered from an acute bacterial infection as shown by her blood analysis described in Section IV-A2.

Alexa's serum glucose level was 504 mg/dL at 1419 and it was increased to 624 mg/dL (normal range: 70-110 mg/dL). Her blood glucose levels remained elevated until November 18 (Table 8) despite of her treatment with insulin (Tables 12, 13). 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 [11]. Stress hyperglycemia, usually associated with infections and other life-threatening illnesses, is due to the release of glucagons and catecholamines [11, page 2061].

Also, on November 16, Alexa had acidemia with a blood pH of 6.92, bicarbonate level of 8 mmol/L, anion gap of 26 mmol/L, and CO_2 level of 9 mmol/L (Table 8). 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 (i.e., lactate, free fatty acids, phosphates) contribute [11]. Alexa's acidemia was treated with high doses of sodium bicarbonate by IV infusion that elevated the blood pH to a high level of 7.58. She was also treated with sodium bicarbonate at 2207 on November 16 even though her blood pH was already high at 7.58 and continued to stay elevated until 1159 on November 17 (Table 8). Treatment with high doses of sodium bicarbonate usually causes metabolic alkalosis, hypokalemia, hypoxia, and brain edema [11].

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 (11, page 2073). Alkalinization increases the avidity of hemoglobin to bind oxygen, thus impairing the release of oxygen in peripheral tissues. The hemoglobin-oxygen dissociation curve is normal in

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diabetic ketoacidosis because of opposing effects of acidosis and the 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 Alexa's case and that her treatment with high doses of bicarbonate contributed to her severe hypoxia and cerebral edema [43-45].

Alexa's serum potassium level was normal (3.7 mmol/L) on November 16. However, it dropped to a low level of 2.2 mmol/L on November 17 following her treatment with high doses of sodium bicarbonate which elevated the blood pH to 7.58 (Table 8). She was treated with potassium chloride solution by IV infusion several times on November 17 and 18 to correct her hypokalemia (Tables 12, 13). Potassium concentrations in the blood usually fall rapidly during therapy with sodium bicarbonate because it leaves the blood and goes back inside the cells, predisposing the patient to cardiac arrhythmias and/or paralysis of the respiratory muscles [11, page 2060].

Harrison's Principles of Internal Medicine states that in metabolic acidosis resulting from diabetes, initial serum potassium concentrations are normal to high, despite depletion of body stores. Potassium usually leaves the intracellular environment because the intracellular proteins bind with hydrogen which leads to cardiac arrest and paralysis of the respiratory muscles [11, page 2060]. Alexa arrived to the ER at the Laurel Regional Hospital at approximately 1322 on November 16, she presented with cardiac arrest and apnea. The low levels of potassium contained within her muscles and nerves contributed to her subsequent cardiac arrest and other symptoms.

Additionally, Alexa suffered from cerebral edema as a result of being diabetic. In diabetic children, cerebral edema is a common cause of death and it occurs more frequently than in adults [11, 46, 47]. Alexa had cerebral edema as shown by the CT scans and stated in the autopsy report [29]. At the time of Alexa's admission in the hospital, her brain edema was mild; nonetheless her treatment with high doses of sodium bicarbonate and ischemia, and hypoxia increased the severity of the edema in the brain as indicated by the CT scans.

The CT scan of the brain taken at 1627 on November 17 showed interval increases in the severity of global edema involving both the supra- and infratentorial structures when compared with the CT scan of the brain taken on November 16. There was a minimal definition of the cortical mantle and basal ganglia due to diffuse edema. There was interval development of bilateral transtentorial herniation and probably tonsillar herniation as well.

Furthermore, Alexa suffered from hypoxia as a result of her anemia, as shown by her very low hemoglobin of 8.3 g/dL and hematocrit of 28.8%, and red blood cell count of 3.1 x 10^6/μL. Her anemia resulted from malnutrition because at 1419 on November 16, the CT scan of the brain did not show any significant bleeding in the subdura. Alexa's hemoglobin levels were 11.4 g/dL and 8.3 g/dL on July 20 and November 16, respectively.

Alexa lost 27% of her hemoglobin level during the four months prior to her hospitalization due to her chronic loss of appetite, diarrhea, and vomiting. On November 16, Alexa had high platelet count of 659,000/μL and it indicated that she had bone marrow hyperplasia which is a response reaction to anemia and systemic infections. Her cardiac arrest, apnea, and hypotension also resulted in severe hypoxia and general ischemia of the brain. She was not breathing for approximately 30 minutes.

Alexa's risk factors for developing diabetes included: (1) malnutrition increased her susceptibility to bacterial and viral infections and 2) vaccination with MMR live viruses increased her risk of infections with these viruses because she had depression in her cellular immunity. It has been established that diabetes tends to be more common following outbreaks of mumps or rubella [48].

IV-C. Clinical evidence and causes of vitamin K deficiency in Alexa's case

On November 16, Alexa's prothrombin time (PT) and partial thromboplastin time (PTT) were highly elevated and these are considered important indicators for vitamin K deficiency [33,49-53]. Alexa's PT and PTT values were 33.3 (266% of normal) and more than 100 seconds (286% of normal), respectively. PT and PTT were measured in fifteen infants who suffered from vitamin K deficiency and were found to be highly elevated. They were reduced sharply within 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 [52].

Vitamin K is essential because it has a coagulation activity and is important for calcification of bones. 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. Chronic treatment with antibiotics, anti-fungal, or other drugs that inhibit bacterial growth and/or diarrhea can cause vitamin K deficiency. The deficiency of vitamin K causes bleeding in both children and adults. It also increases the risk for bone fractures.

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 [33, 49].

Infants who develop vitamin K deficiency usually suffer from bleeding in the brain and other locations. In a study conducted in Japan, intracranial hemorrhage was observed in 353 (75%) cases out of 473 infants who suffered from vitamin K deficiency [54]. Additionally, bleeding in the brain was ob-

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served in eleven infants who developed vitamin K deficiency. The localizations of the intracranial hemorrhage were as follows: intracerebral (91%), subarachnoid (46%), subdural (27%), and intraventricular (27%) [55].

Furthermore, fifteen infants who developed bleeding in the brain and other locations were found to be suffering from vitamin K deficiency. 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 the cerebrospinal fluid. Skin bleeding (ecchymosis) was also observed in three patients [52].

In addition to the bleeding in the brain and other locations, infants who suffered from vitamin K deficiency also experienced the following: seizures, drowsiness, diarrhea, vomiting after meals, lack of neurologic response, and tense or bulging fontanelle [52, 55]. In a study that included fifteen infants, convulsions and irritability were observed in 47% and 33% of patients, respectively. Upon physical examination there was a bulging or full fontanel in ten patients [52]. A second study included eleven infants who developed vitamin K deficiency. The presenting complaints were seizures (91%), drowsiness (82%), vomiting (46%), fever (46%), and acute diarrhea (27%). On examination, a tense or bulging fontanelle was observed in 73% of the patients [55]. Alexa had a seizure in the hospital on November 16 for which she was treated with Fosphenytoin (anticonvulsant) on November 16 and 17 (Tables 7, 12).

Alexa's vitamin K deficiency was caused by her chronic loss of appetite, long-term treatment with an anti-fungal drug, frequent bowel movements and vomiting. At two months of age, Alexa was in the 50th percentile for weight on the growth chart. By 15 months of age her weight dropped to below the 1st percentile (Table 2). Her length also dropped from the 25th percentile at the age of 7.4 months to the 10th percentile at 12 months of age. Alexa suffered from fungal, bacterial, and viral infections in the time period between 11 and 15 months of age. At approximately 11 months of age, Alexa developed white thrush on her tongue. On July 20, 1999 she was treated with Nystatin (anti-fungal) orally for at least six weeks (Section I-B). The common adverse reactions of Nystatin are diarrhea and vomiting.

Diarrhea and malabsorption in children and adults can predispose them to vitamin K deficiency. 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 [56].

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 [57]. Hanawa et al. evaluated 57 infants from 2 weeks to 4 months of age and they discovered that the infants had experienced bleeding episodes due to vitamin K deficiency. The main causes of vitamin K deficiency were hepatobiliary lesions, chronic diarrhea, and long-term antibiotic therapy [54].

The levels of serum liver enzymes were elevated at the time of Alexa's admission to the hospitals on November 16 suggesting liver damage (Table 12). The synthesis of the coagulation factors occurs in the liver and liver damage can cause bleeding problems. The above should have been considered in Alexa's differential diagnosis.

Alexa's prothrombin time (PT) and partial thromboplastin time (PTT) were found to be highly elevated on November 16 and were reduced by more than two-fold by November 18 because Alexa was treated with fresh frozen plasma (FFP) as shown in Tables 7 and 13. FFP is efficacious for treatment of factors II, V, VII, IX, X, and XI deficiency. For example, for anticoagulated patients who are actively bleeding or who require emergency assistance as a result of factors II, VII, IX, and X deficiency, FFP can be used to achieve immediate hemostasis. FFP is also used for intravascular volume replacement in acute blood loss [58].

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 [33]. The final diagnostic confirmation of vitamin K deficiency is reached following a rapid, therapeutic response to vitamin K1 or fresh frozen plasma (FFP) administration as it happened in Alexa's case (Table 11). It is unfortunate that the medical examiner and other physicians who evaluated this case overlooked the overwhelming medical evidence that clearly shows that Alexa was suffering from vitamin K deficiency.

Section V. Review of Medical Examiner's Autopsy Report in Alexa's Case and My Observations

Alexa Marie Shearer was pronounced brain dead on November 18 at 1020. She was kept alive on a life support system until November 19 at 0400 to harvest her organs for donation. The Chief Medical Examiner for the District of Columbia gave his permission to harvest Alexa's organs. Dr. Carlos Fernandez-Bueno gave Alexa 5000 IU of heparin prior to harvesting her organs, which was 11.2 times the therapeutic dose. He removed the heart, liver, kidneys, adrenal glands, spleen, and a large portion of the diaphragm. He kept Alexa's body in the morgue's cooler until the time of autopsy.

Dr. Fernandez-Bueno noticed abnormalities in the abdominal cavity that indicated Alexa suffered from acute pancreatitis. He described the abnormal lesions in his report and took pictures of the lesions and sent them to the Medical Examiner [10]. However, the Medical Examiner's office lost these pictures and the Medical Examiner never saw them. In addition, the Medical Examiner did not review Dr. Fernandez-Bueno's report [10]. The organ harvesting procedure, descriptions of lesions observed in the abdominal cavity, and the adverse reactions to heparin are all described in Section III of this report.

The Medical Examiner performed Alexa's autopsy at 1300 on November 19 (Autopsy Case No. 99-4143). The main objective of this autopsy was to establish the causes of injuries and death in this case. Detective Glenn Case from the Howard County Police Department attended the autopsy**.** At the end of

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the autopsy, the Medical Examiner told Detective Case that Alexa was killed by blunt force trauma to her head. The police investigation turned into a homicide and the suspect was Kathleen Butcher. However, at that time the Medical Examiner did not do any microscopic examination of the brain, dural membranes, retina, skin, or the T-10 vertebrae.

In addition, he did not review Alexa's medical records prior to her hospitalization on November 16 and during her hospitalization on November 16 through 18. He examined the brain grossly about 4 months after he told Detective Case that Alexa was killed by blunt trauma to the head. The Medical Examiner did not find any evidence of bleeding in the brain or other injuries that indicated Alexa suffered from trauma to the head. He released his autopsy report describing his findings in this case on April 6, 2000 [29].

Detective Case interviewed Kathleen on November 19 at 2120 to get her statement and she said that she did not hurt Alexa. However, Kathleen was arrested and incarcerated December 3, 1999 based on the Medical Examiner's theory that Kathleen killed Alexa. She was then pregnant with her fourth child [6, pages 54].

Prior to making the arrest, Detective Case spoke with the Medical Examiner between November 19 and December 3 to confirm the cause of death in this case. He told the Medical Examiner that he was planning to arrest Kathleen and wanted to be absolutely clear that he was dealing with a homicide investigation. The Medical Examiner confirmed that Alexa died as a result of blunt head trauma, her injuries occurred after she ate lunch at Kathleen's house, and the manner of death was homicide [6, pages 107].

I reviewed the Medical Examiner's autopsy report regarding Alexa's case as well as his court testimony. I found that the Medical Examiner's investigation of this case was incomplete and his conclusions were unsupported by the medical evidence, even by his own findings. Alexa's injuries and death were not caused as a result of violent shaking and blunt head trauma as he claimed. Vaccines, infections, and adverse reactions to medications caused her injuries and death. Below are the descriptions of the Medical Examiner's findings and the medical evidence that supports my findings and assertions.

V-A. Alexa's weight and development

The Medical Examiner stated that the body of Alexa was that of a normally developed, well-nourished white female, but slightly small for the reported age of 15 months. I disagree with the Medical Examiner's statement that Alexa's development was normal and she was well-nourished. Her medical records show that she suffered from chronic health problems such as loss of appetite, diarrhea, vomiting, anemia, fungal infection, an upper respiratory tract infection, ear infection, osteomyelitis, vitamin K deficiency, and diabetes mellitus.

She was treated with three consecutive courses of Nystatin (anti-fungal) and the common adverse reactions of Nystatin are diarrhea and vomiting. At two months of age, Alexa was in the 50th percentile for weight on the growth chart and her weight dropped to below the 1st percentile at 15 months of age. Her length also dropped from the 25th percentile at 7.4 months of age to the 10th percentile at 12 months (Table 2). Sections I-IV contain detailed information describing Alexa's numerous health problems.

V-B. The factual causes of the external lesions observed in Alexa's case

The subcutaneous bleeding and extracranial accumulation of fluid in Alexa's case progressively developed following her admission to the hospital. They were not caused by trauma as stated by the Medical Examiner and the State's other expert witnesses. These lesions developed as a result of the synergistic actions between infections, their complications, and medications administered to Alexa in the hospital which include hypotention, acute pancreatitis; hypoxia, brain ischemia, and Alexa's treatment with high therapeutic doses of epinephrine, dopamine, sodium bicarbonate, and fluids (Tables 7, 13, 14). She was also given excessive amounts of heparin prior to harvesting her organs for donation (Section III).

Detective Glenn Case and the Howard County Fire and Rescue Team (HCFRT) did not see any visible sign of injury or trauma on Alexa's head or the rest of her body when they arrived on the scene at Kathleen's house at approximately 1300 on November 16 [26]. The first sign of scalp swelling was observed in the hospital at about one hour following her treatment with epinephrine, dopamine, sodium bicarbonate, atropine and IV fluid which increased her blood pressure and her intravascular volume (Table 5). Dr. James Chamberlian examined Alexa at the Children's National Medical Center (CNMC) at about 1449 and he did not notice signs of injuries on Alexa's head, except for a boggy swelling of the scalp at the occipital and the right temporopartial areas. In addition, he did not see any other sign of external injuries on Alexa's chest, abdomen or her back at that time.

Furthermore, the results of the CT scans of the head region taken on November 16 and the 17 also showed that the extracranial lesions developed during Alexa's hospitalization. At 1615 on November 16, the CT showed a marked amount of swelling and hemorrhage in the scalp's soft tissues, primarily on the right side at the vertex. The severity and the magnitude of these lesions increased significantly within the subsequent 24-hour period as shown by the CT taken on November 17. It indicated interval increase in the subgaleal hematoma, which became bilateral and diffuse (more marked on the right than the left).

Alexa suffered from acute pancreatitis and the mediators released during the progression of her illness caused vasodilation as described in Section IV-A of this report. Also during that period, Alexa was treated with high therapeutic doses of medications and fluids by IV infusion that increased Alexa's cardiac rates and her blood pressure. This influenced the intravascular hydrostatic and osmotic pressure and led to the leakage of fluid outside of the blood vessels (Tables 5, 7, 12).

Drs. Bell and Fernandez-Bueno provided evidence that shows that the bleeding on Alexa's back occurred on November 19 and that it did not happen on November 16. Dr. Bell examined Alexa on November 18 and he did not see any bleeding on Alexa's back. He only saw bruising over Alexa's right eye which became visible at 8:30 a.m. on November 18. Additionally, Dr. Fernandez-Bueno examined Alexa's body while harvesting her organs on November 19. He did not report seeing

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any bruising on Alexa's back, chest, or abdomen during his examination of her at 0400.

During autopsy, on November 19, the Medical Examiner observed nine purple contusions in various locations on Alexa's back, measuring 1/8-3/8 inches in size. He also observed bruises on her right cheek and below her right ear lobe. He claimed that these bruises were caused by trauma on November 16. The following is a list of medical evidence that disproves the Medical Examiner's theory. 1) Numerous physicians and police examined Alexa prior to 0400 on November 19. They did not see nor report any marks on Alexa's back, chest, and abdomen nor did they see the bruises described by the Medical Examiner. Their observations support the fact that the bruises observed by the Medical Examiner developed following her treatment with excessive amounts of heparin on November 19 at 0400. 2) The microscopic examination of the skin and the subcutaneous tissues of one of those bruises performed by the Medical Examiner also revealed that the bleeding was fresh. It showed mild and focal hemorrhage in the deep subcutaneous fat, along a septum and within the fat lobule. No inflammation or hemosiderin were seen in the area of the bleeding. These findings indicate that the bleeding occurred within 12 hours of autopsy.

The Medical Examiner also confirmed that the bleeding under Alexa's skin was less than 12 hours old when he was asked in court as provided below [24, page 51; 59, page 190].

> **Defense Attorney:** "No inflammation or hemosiderin in the skin associated around the area around those bruises, would that be consistent with an injury or a collection of blood, that took place within twelve hours of death?"
> **Medical Examiner:** "That factor alone, yes it is."
>
> **Prosecutor:** "On cross-examination you had indicated that the microscopic findings were consistent with the bruising occurring within twelve hours of death, do you recall that testimony?"
> **Medical Examiner:** "Yes, I do."

I do not understand the logic the Medical Examiner used when he asserted that the bleeding under the skin on Alexa's back, observed at autopsy, was caused by trauma on November 16. His findings undisputedly show that the bleeding was less than 12 hours old and he performed the autopsy at approximately 96 hours following Alexa's admission in the hospital. The bleeding on Alexa's back was clearly caused by the excessive amounts of heparin given to her about seven hours prior to autopsy.

V-C. Alexa's brain edema and subdural hemorrhage were not caused by trauma

On November 19, the Medical Examiner found that Alexa's brain was severely swollen and the changes were diffuse and symmetrical. He also observed mild diffuse subdural hemorrhage over both cerebral convexities. He told Detective Case who was present at the autopsy that the damage in Alexa's brain had been inflicted by blunt trauma to the head. The Medical Examiner did not consider the factual causes involved in this case that caused the brain edema and subdural bleeding. He also did not wait to finish his examination of the brain and the dura before rendering his professional opinion regarding the manner of death.

The Medical Examiner examined one section of the dura at a later date and found fresh subdural hemorrhage that indicated the bleeding was less than 24 hours old. The red blood cells were largely intact and there were platelet-fibrin zones. The hemorrhage also contained a moderate mixed inflammatory infiltrate and rare hemosiderin granules, but there was no evidence of organization (no fibroblasts). These changes indicate that the bleeding was less than 24 hours old. In the area of bleeding or inflammation, sometimes as early as 24 hours after injury, fibroblasts and vascular endothelial cells begin proliferating to form granulation tissues by three to five days [60, pages 71-72].

In addition, the Medical Examiner waited until March 30, 2000 (about 4.5 months after the autopsy) to examine the fixed brain grossly [29]. He examined sections from different parts of the brain and he found evidence of severe edema, but he did not notice bleeding in any part of the brain. He determined that there was no need to examine the brain microscopically because he would not find diffuse axonal injuries.

The clinical evidence shows that the subdural bleeding and the brain edema progressed rapidly during Alexa's hospitalization following her treatment with many medications, fluid, and heparin (Sections II-IV). The CT scan of the brain taken on November 16 at 1615 showed the presence of focal subarachnoid hemorrhage and not diffuse hemorrhage as described in the Medical Examiner's autopsy report [10, 29]. Alexa was treated with high therapeutic doses of epinephrine (0.5 mg) given intravenously in four injections as well as epinephrine by IV infusion prior to the CT scan having been taken (Tables 5, 7).

Some of the serious adverse reactions of epinephrine are intracerebral, subdural and/or subarachnoid hemorrhage even when given at a low dosage of 0.05 mg subcutaneously, which is less than 10% of the epinephrine dose given to Alexa [61]. Additionally, Alexa suffered from acute pancreatitis and vitamin K deficiency that increased her tendency to bleed as described in Sections II-IV. To compound this, Alexa was given excessive amounts of heparin on November 19 that also contributed to the bleeding.

The result of the CT scans of Alexa's brain showed that edema rapidly progressed during Alexa's hospitalization. The CT taken at 1615 on November 16 showed diffuse and severe edema of the cerebrum with obliteration of the cortical sulci, small ventricles and obliterated basilar cisterns. Twenty-four hours later, the CT showed interval increases in the severity of the global edema involving both the supra- and infratentorial structures. There was a minimal definition of the cortical mantle and basal ganglia due to diffuse edema. Also, there was interval development of bilateral transtentorial herniation and probably tonsillar herniation.

There were several factors involved in this case that led to the formation of edema in Alexa's brain. (1) Alexa suffered from cardiac arrest and apnea. She did not breathe for about 30 minutes and this led to ischemia, hypoxia, and edema of the brain. It caused hypoxic/ ischemic encephalopathy. (2) Alexa

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suffered from diabetes mellitus and brain edema is associated with diabetes in children (Section II). (3) Alexa suffered from acute pancreatitis which led to vasodilatation, hypotension, and edema (Section IV). (4) Alexa was treated with high doses of sodium bicarbonate that caused metabolic alkalosis (blood pH 7.58), hypoxia, and brain edema. (5) Alexa was treated with relatively large volumes of fluid and high therapeutic doses of epinephrine and dopamine by IV. These medications increased her blood pressure and the intravascular volume thus contributing to the pathogenesis of edema in this case (Sections II and IV).

V-D. The factual causes of the bleeding in Alexa's eyes

At autopsy, the Medical Examiner observed bilateral optic nerve sheath hemorrhage as well as tissue hemorrhage surrounding the left optic nerve. He also observed small hemorrhage in the retina of the right eye. He stated that these lesions were caused by violent shaking of the head on November 16. The Medical Examiner's conclusion is not supported by medical evidence. He neglected to examine H & E stained tissue sections of the bleeding areas by microscope to check for evidence of inflammation and he did not estimate the age of the bleeding. Additionally, he failed to consider or evaluate other factors involved in this case that are known to cause bleeding in the eye such as diabetes and the treatment with excessive amounts of heparin.

My evaluation of the medical evidence in this case revealed that the bleeding in the retina and optic nerve sheath were caused by the synergistic actions of the following factors. (1) Alexa suffered from diabetes and bleeding in the eye (inner retina, superficial nerve fiber layer, and pre-retinal hemorrhage) is commonly described in patients suffering from diabetes [11]. (2) Alexa suffered from severe hypoxia as a result of her cardiac arrest and apnea, anemia, hypotension, metabolic and respiratory acidosis, and metabolic alkalosis (from the excessive treatment with sodium bicarbonate in the hospital). Hypoxia caused damage in the small blood vessel walls that led to bleeding. (3) Alexa was treated with excessive doses of epinephrine and bleeding is one of the adverse reactions of epinephrine. (4) Alexa was given excessive amounts of heparin on November 19 that caused bleeding. (5) Alexa had severe brain edema that caused pressure on the retina.

V-E. The causes of subdural bleeding in the spinal cord in Alexa's case

At autopsy, the Medical Examiner examined the spinal canal at approximately the T-8 vertebrae and found localized fresh epidural hemorrhage. The cerebrospinal fluid surrounding the spinal cord was diffusely bloody. The examination of serial transverse sections of the spinal cord revealed normal architecture, except in the cervical cord, where the tissues were more softened and friable with loss of architecture.

Sections of the spinal cord were submitted (two cervical and two thoracic, all in one cassette) for microscopic examination. He found that all sections had eosinophilic anterior horn neurons (hypoxic change), with focal extravasation of erythrocytes from blood vessels with early wall necrosis. The parenchyma appeared to be diffusely necrotic, as well, with poor staining and loss of detail and architecture [29, 59].

The Medical Examiner stated the necrosis in the spinal cord at the cervical and the thoracic regions was caused by hypoxia, however, he did not evaluate the causes of hypoxia in this case. Alexa suffered from cardiac arrest and she was not breathing for approximately 30 minutes on November 16 and that subsequently led to ischemia and hypoxia. In addition, she was treated with excessive doses of sodium bicarbonate that caused hypoxia.

Furthermore, the Medical Examiner described the bleeding observed in the thoracic region of the spinal cord as fresh but he did not investigate the role of the high doses of epinephrine and the excessive amounts of heparin given as the causes of bleeding in this case.

The Medical Examiner performed the toddler's autopsy on November 19. If the bleeding occurred on November 16 in Kathleen's home as the Medical Examiner believed, then the age of the bleeding would have been about 72 hours old. By 72 hours, we would expect to see a proliferation of fibroblasts and the deposition of hemosiderin in the areas of hemorrhage and not fresh blood.

V-F. The causes of acute otitis media and mastoiditis in Alexa's case

The CT scan of the head region taken on November 16 showed opacification of the middle ear and mastoid bilaterally thereby indicating the presence of infection. Dr. Vezina, radiologist, stated that Alexa's middle ear and mastoid contained soft tissue, fluid, or blood in both sides [24, page 133]. Normally, the middle ear and mastoid contain air. The possibility of a chronic ear infection in Alexa's case was also stated by Dr. Michael James Bell who reviewed the CT scan and treated Alexa on November 16 [25, page 222].

The results of Alexa's blood test taken on November 16 also indicated that Alexa was suffering from a bacterial infection. The evidence that shows she had this infection includes elevated white blood cell count (40,600/ μL), the presence of toxic granules and Dohle bodies in the neutrophils, and the presence of elevated numbers of immature neutrophils (37% band and 5% metamylocyte). However, the Medical Examiner did not make any attempt to examine the middle ear or mastoid at autopsy nor did he investigate the link between the bilateral ear infections and the causes of injuries and death in this case.

Dr. Michael Baden, a forensic pathologist who reviewed this case for the defense, was surprised to learn that the Medical Examiner did not examine the middle ear and mastoid bone at autopsy. He stated that it is very important to look at the mastoid air cells and the middle ear with the naked eye. It helps us to see if there was any abnormality or infection in the ears which had been suggested in the medical records. He also stated that it only takes about five minutes to drill into the middle ear and to see if there was an occipital infection [5, page 102].

The middle ear is connected to the nasopharynx via the eustachian tube. When this tube is blocked, fluid collects in the middle ear and mastoid cavities providing a culture medium for the bacteria present. Acute otitis media (AOM) or middle ear infection may result. Viral respiratory tract infections that can cause edema of the eustachian tube mucosa often precede or accompany episodes of AOM [PN1].

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Following her vaccination with MMR and varicella vaccines, Alexa suffered from upper respiratory infections during the last three months of her life prior to her cardiac arrest. As I described in Section I-D, rhinitis, upper and lower respiratory tract infection, and otitis media have been observed in some children who have received these vaccines. Alexa also suffered from poor appetite, poor weight gain and diabetes mellitus all of which are known risk factors for developing upper respiratory tract and ear infections.

Streptococcus pneumonia and Hemophilus influenza are the primary causes of bacterial ear infection in children [11, page 182]. S. pneumonia was identified in approximately 40 to 50 percent of cases of reported otitis in children. Prior infection by a respiratory virus was thought to contribute significantly to these pneumococcal infections by causing congestion of the openings to the eustachian tubes or the paranasal sinues [11, page 871]. The incidence of pneumococcal bacteremia is relatively high among infants up to 2 years of age (160 cases per 100,000 population). In addition to otitis media, S. pneumonia and Hemophilus influenza are also known to cause osteomyelitis in children [11). Alexa had osteomyelitis of the T-10 vertebrae.

IV-G. Alexa's osteomyelitis

A radiological bone survey on Alexa was performed on November 17 and the radiologist, Dr. Gilbert Vezina observed erosion of the inferior endplate of the T-10. It appeared irregular but well defined. He stated that about one eighth of the whole vertebrae was missing and that this may have represented a lytic bone lesion, however, osteomyelitis of the vertebral body could not be ruled out. He also stated that this finding did not have the expected appearance of a fracture [10, 24].

Furthermore**,** Dr. Michael James Bell, the pediatric intensive care physician reviewed the CT scan and stated that there was erosion of the inferior end plate of the T-10 vertebrae. It was seen in osteomyelitis caused by bacterial infection. He also stated that there was no clear evidence of a fracture of the 10^{th} thoracic vertebrae [25]. In addition, Dr. Darryl Garfinkle, a diagnostic pediatric radiologist reviewed the CT scan and he concurred stating that there was no evidence of fracture in the T-10 vertebrae. He said that there was destruction of the bone in the lower half of the vertebral body and that bone was missing, which is consistent with lytic bone disease [7, page 138].

Dr. Garfinkle explained the process of forming lytic lesion in bone by infection as follows: lytic bone results from infection caused by increased metabolic activity within the bone. The organism produces certain types of chemicals that can destroy the bone. When the organism is lodged in the bone, it grows and produces certain chemicals, called enzymes, which have the destructive potential to destroy bone and its support tissues [7, page 138].

The Medical Examiner examined the T-10 vertebrae at the time of autopsy on November 19 and stated that the body of the T-10 vertebrae had a recent fracture in it with no bony growth or other evidence of healing. This fracture had fresh red blood associated with it and fresh blood around the crack [59]. He also stated that the fresh hemorrhage in the T-10 occurred within ten to twelve hours of the infliction of that injury. The Medical Examiner stated that the fracture of the T-10 resulted from overextending the spine due to the arching of Alexa's back. It was a hyperextension injury and was not the result of trauma to the abdomen [59, page 101].

The following list of medical facts disproves the Medical Examiner's theory that the fracture of Alexa's T-10 vertebrae was caused by Kathleen Butcher and that it happened on November 16.

1) The CT scan on November 17 showed a lytic lesion in the bone and part of the bone was missing. Dr. Garfinkle, radiologist, stated that we can sometimes see more bone destruction radiographically than is visible by gross examination [7, page 148]. In addition, no one can rule out with medical certainty the possibility of bacterial infections in the bone without examining tissue sections of the bone microscopically and doing a bone scan. Bone scans detect hot spots in the bone which may represent bone regeneration (healing) [24, page 156]. The Medical Examiner did not take a sample of the bone to be examined by microscope in this case.

2) The clinical evidence indicated that Alexa was suffering from bacterial infection on November 16. These include elevated white blood cell count (40,600/ μL), the presence of toxic granules and Dohle bodies in her neutrophils, and the presence of elevated number of immature neutrophils (37% band and 5% metamylocyte). Alexa also had otitis media and the primary causes of bacterial ear infection in children are S. Pneumonia and Haemophilus influenza. These bacteria are also known to cause osteomyelitis in children [11].

3) The Medical Examiner stated that the hemorrhage in T-10 was fresh and occurred within ten to twelve hours of the infliction of that injury. That means that the bleeding occurred on November 18 or 19 and not on November 16. It is very clear that the bleeding in this case was caused by the excessive amounts of heparin given to Alexa at approximately 9 hours prior to autopsy (Section III).

4) The Medical Examiner observed fresh bleeding in the areas of the T-8 and T-10 but not in the area of the T-9. The Medical Examiner and the State's Expert Witness stated that the bleeding in the vertebra resulted from someone holding Alexa from the back and shaking her to crack T-10. If their theory was correct, then we expect to see bleeding in the tissue associated with the T-9 vertebrae as well. The evidence clearly shows that their theory is wrong and that the fresh bleeding was caused by the excessive treatment with heparin.

The clinical evidence presented above clearly indicates that the lytic lesion in the T-10 vertebra represents osteomyelitis. It was not a result of trauma as the Medical Examiner and the other state's expert witnesses had suggested. Osteomyelitis is an infection of the bone and marrow. It most commonly results from bacterial infection, although fungi and viruses can infect the bone and marrow as well. Infection can reach the bone via the blood stream. Hematogenous spread via arterial and venous routes can result in lodgment of organism in bone marrow of the vertebrae and then spread to the vertebral body [62, page 2326].

Clinical manifestations of osteomyelitis varies with the virulence of the organisms and the nature of the host resistance. General findings include fever, malaise, anorexia, and weight loss [62, page 2425]. Alexa suffered from these symptoms at least during the three months prior to her hospitalization on

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November 16. Alexa had several risk factors that predisposed her a bacterial infection of the bone that included diabetes mellitus, poor weight gain, anemia, ear infection, and upper respiratory infection.

The logical explanation for the presence of a fresh crack in the vertebral body of T-10 observed by the Medical Examiner at autopsy was the use of the defibrillator on November 16. It was used six times to defibrillate Alexa's heart. The rescue team's captain and the physician who treated Alexa said that the electrical shock used to defibrillate Alexa's heart caused hyperextension or arching of the back of the child. The body jerks up by an inch or two and occasionally the body will momentarily jump [25, page 54]. Alexa's T-10 vertebrae was very fragile and about one-eighth of the whole vertebrae was missing due to the erosion caused by infections and its disposition to crack easily. It is important to note that the Medical Examiner also confirmed that the fracture of the T-10 vertebrae was caused by hyperextension of the back and that it was not a result from trauma to the abdomen [59, page 101].

V-H. Fracture of rib #8, who did it?

A single view of Alexa's chest x-ray taken on November 18 showed a deformity of the posterior aspect of the 8th rib on the left side that suggests the possibility of a healed fracture. Dr. Darryl Garfinkle, a radiologist, also examined the x-ray and said that the healed left eighth rib fracture was probably at least ten days old [7]. At autopsy, the Medical Examiner observed a slight nodular expansion at the posterior region of the left 8th rib and cutting through this region revealed solid bone growth. He said that this lesion represented a healed fracture and it was more likely months old or older [59]. However, he did not attempt to investigate the possible cause(s) of this old fracture as required by law.

Investigating the cause(s) of the left 8th rib fracture in a scientific manner may have revealed the factual cause(s) of the fracture and could have helped in explaining the causes of Alexa's cardiac arrest that occurred on November 16. Some experts who testified in this case stated that this fracture was caused by previous child abuse. If this was the case, then, who did it?

The likely cause for the bone lesion observed in Alexa's rib #8 was vitamin K deficiency. People who suffer from vitamin K deficiency also suffer from bone problems. Alexa suffered from vitamin K deficiency as I described in Section IV-C of this report. Her prothrombin time (286% of normal) and partial thromboplastin time (266% of normal) were highly elevated and these are important indicators for vitamin K deficiency.

In addition to controlling coagulation factors, vitamin K is also important for bone metabolism. The carboxylation process of two bone matrix proteins necessary for normal bone metabolism are vitamin K-dependent. These proteins contain (a) the amino acid γ-carboxyglutamic acid and (b) the carboxyl groups of the glutamic acid residues that are needed for binding calcium and phospholipid [33,49]. Low vitamin K intakes were associated with an increased incidence of hip fractures in a cohort study involving 335 elderly men and 553 elderly women [63].

The lesion in the rib observed in Alexa's case may likely represent a local bone defect caused by vitamin K deficiency and followed by healing as shown in the following two cases. Fenton et al. evaluated two American infants who had massive intracranial hemorrhage, no history of trauma, and radiographic findings that were initially interpreted as linear parietal fractures. The radiographic findings raised the possibility of non-accidental trauma in these infants.

The investigation revealed that both infants had severe coagulopathy, one due to hemorrhagic disease of the newborn (vitamin K deficiency) and the other due to disseminated herpes simplex virus infection. Both infants died. At autopsy, the parietal bone abnormalities were found not to be fractures but proved to be an anomalous suture in one infant and a connective tissue fissure in the other [64].

Section VI. Kathleen Butcher's Jury Trial and Analysis of the Events

Kathleen Butcher's jury trial was held in the Circuit Court of Howard County, Maryland on February 21, 2001 and lasted for 13 days (Criminal case No. 13-K-99-38775). The Honorable Judge Raymond J. Kane, Jr. presided on this trial. Attorneys Kim Oldham and Danielle Duclaux represented the State and attorneys Joel Abramson and Ralph Lotkin represented the defendant in this case. Kathleen was convicted of involuntary manslaughter and child abuse in the death of Alexa Marie Shearer and sentenced to 10 years and 5 years, respectively, to serve concurrently in prison. I reviewed the trial documents and I found that the State did not present any medical evidence that showed Kathleen Butcher harmed Alexa nor that Alexa's injuries were caused by violent shaking and trauma [5-7, 24-27, 59, 65-68].

VI-A. The State's theory and names of expert witnesses

The State's theory was that Kathleen Butcher inflicted Alexa's fatal injuries on November 16, 1999 after Alexa ate lunch at approximately 1215. They alleged that repeated violent shaking and blunt trauma to the head and abdomen caused Alexa's injuries. The prosecutors stated that Kathleen probably became overwhelmed taking care of eight children, lost her temper for a few moments and injured Alexa. Kathleen has stated numerous times that she is innocent and that she took very good care of Alexa.

The medical examiner and five physicians provided technical and expert testimonies for the State in this case. The medical examiner testified twice for the State in this case. The physicians who testified were the following:

1. The Chief Medical Examiner for the District of Columbia
2. State Expert Witness, consultant in child abuse cases
3. Dr. Michael James Bell, Pediatric Intensive Care Physician
4. Dr. Carlos Fernandez-Bueno, surgeon
5. Dr. Gilbert Vezina, pediatric neurologist
6. Dr. Mary Julia Marcin, pediatrician

VI-B. The defense's theory and names of expert witnesses

The defense's theory was that Alexa's injuries were caused prior to bringing her to Kathleen's house at 7:30 a.m. on No-

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vember 16, 1999. They believed them to have been caused by an individual other than Kathleen. One expert witness said that Alexa suffered from a bacterial infection and that her infection may have caused some of Alexa's injuries. The following physicians testified for the defense in this case.

1. Dr. Jack Daniel, pathologist
2. Dr. Michael Baden, forensic and clinical pathologist
3. Dr. Darryl Garfinkle, a diagnostic pediatric radiologist

VI-C. Testimonies of the State's expert witnesses

The Medical Examiner and the State's Expert Witness were the key expert witnesses for the State in this case. They stated that Alexa's fatal injuries occurred after she ate lunch at 1215 on November 16, 1999. They alleged that violent and repeated shaking along with blunt trauma to the head caused her injuries. In addition, the State's Expert Witness declared that Alexa had also suffered from blunt trauma to her abdomen.

I reviewed both the Medical Examiner and the State's Expert Witness' medical reports along with their court testimony. I found that their investigations pertaining to this case were incomplete and their conclusions were not supported by medical facts. They neglected to evaluate Alexa's adverse reactions to vaccines and medications that were responsible for causing her injuries and death as described in Sections I-V of this report. They also misinterpreted the clinical data in this case. It also should be pointed out that the Medical Examiner performed an incomplete autopsy in this case.

I presented my analysis for the Medical Examiner's autopsy report and his findings in Section V of this report. Below is my analysis with regards to the State's Expert Witness' report and her testimony in this case. I will present a list of observations that show the State's Expert Witness' investigation was incomplete and that her conclusions were not supported by medical facts.

VI-D. Analysis of State's Expert Witness report and her testimony in Alexa's case

The State's Expert Witness was asked by the prosecutors to review Alexa's case and to provide her opinion concerning the causes of injuries and death in this case. The State's Medical Witness is a physician. She stated that she had evaluated all of the medical evidence in Alexa's case thoroughly and found Alexa was growing and thriving normally during her life. She did not believe that Alexa had suffered from any significant health problems that led to her cardiac arrest on November 16, 1999. The State's Medical Witness concluded that Alexa's fatal injuries were sustained after she ate lunch at 1215 on November 16, 1999 at Kathleen's home. She alleged that her injuries were caused by violent and repeated shaking of the head along with blunt trauma to the head and abdomen [2, 26].

I reviewed the State's Expert Witness' report and her court testimony and I totally disagree with her findings and her conclusions in this case based on the medical evidence described in this report. My findings clearly show that Alexa suffered from chronic illnesses that led to her cardiac arrest and subsequent death in November of 1999. Below is a list of observations that support my assertions.

1) Alexa suffered from jaundice and an upper respiratory tract bacterial infection during the first week of her life (Section I-A). Her blood bilirubin level was 16.5 mg/dL at five days after birth, which was about 8 times the expected normal level of 2 mg/dL. Neurological damages have been observed in infants who had blood bilirubin levels > 12 mg/dL (Section I-B).

2) Alexa suffered from poor appetite beginning around 10 months of age. Her appetite gradually worsened until the time of her death at 15 months of age. For example, on July 20, Alexa's mother told her pediatrician that Alexa had exhibited a poor appetite during the 2-3 weeks prior to her appointment. Also of special importance is the fact that Alexa vomited on many occasions and had frequent bowel movements. This led to a significant reduction in her food intake and contributed to her anemia, vitamin K deficiency, and immune depression [Section I-C].

3) Alexa suffered from poor weight gain. At two months of age she was in the 50th percentile for weight on the growth chart and her weight dropped to below the 1st percentile at 15 months of age. Her length also dropped from the 25th percentile at 7.4 months of age to the 10th percentile at 12 months (Table 2).

4) Alexa suffered from fungal, bacterial, and viral infections in the time period between 10 and 15 months of age. On July 20, 1999, at about 10 months of age, Alexa developed white thrush on her tongue. Consequently she was treated with three consecutive courses of Nystatin (anti-fungal) orally which caused vomiting and diarrhea. She also suffered from an upper respiratory tract infection, congestion, and a low-grade fever. In addition, the blood tests and CT scan taken on November 16 showed that Alexa suffered from bacterial infections, diabetes, acute pancreatitis, anemia, osteomyelitis of the T-10 vertebrae, otitis media, and mastotidis (Sections I-VI).

5) The State's Expert Witness stated, "I saw no evidence that Alexa Shearer had any kind of serious infection whatsoever in this case." The medical evidence described in this report (Sections II-V) clearly shows that Alexa suffered from an acute bacterial infection. She also stated that Alexa's white blood cell count was high on November 16 because of her treatment with epinephrine and it was not a result of bacterial infection [26, page 163]. The medical evidence described below clearly shows that bacterial infection was the primary cause that led to the elevation of Alexa's white blood cell count and it was not the treatment with epinephrine.

On November 16, Alexa had an acute bacterial infection as indicated by her elevated white blood count (WBC) of 40,600/μL, neutrophil band of 37%, metamylocyte of 5% and the presence of toxic granules and Dohle bodies in her neutrophils. Treatment with epinephrine does not cause elevation in the number of immature neutrophils in the blood circulation nor does it induce the formation of toxic granules and Dohle bodies in the neutrophils.

Additionally, Alexa's treatment with epinephrine began at 1328 and ended at 1346 on November 16. A peak leukocytosis usually occurs within 5 to 10 minutes following the administration of epinephrine and rarely lasts more than 20 minutes. Alexa's white blood cell count was found to be 32,100/μL at 1419, which was 31 minutes after the last epinephrine treatment.

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Furthermore, Alexa's white blood cell count increased from 32,100/μL at 1419 to 40,600/μL at 1507 and there was no epinephrine given to Alexa during that time. Also, Alexa was treated with epinephrine by IV infusion at 0.6μg/kg/min at 1527 and by 2207 she received a very high dose of epinephrine (2.13 mg). However, Alexa's white blood cell count (WBC) was reduced from 40,600/μL at 1507 to 13,800/μL at 2207. The reduction in Alexa's WBC was caused by her treatment with high therapeutic doses of antibiotics (Cefotaxime and Vancomycin) at 1507. Neutrophils disappear from the circulation with a half-time of 6.7 hours and this explains the reduction in Alexa's WBC after her treatment with the antibiotics.

Alexa was treated with high doses of epinephrine on November 17 and 18 and her WBC did not increase (Tables 9, 12, 13). These data indicate that Alexa's WBC likely increased not as a result of her treatment with epinephrine, but because she had a bacterial infection.

6) The State's Expert Witness stated that Alexa's prothrombin time (PT) and partial thromboplastin time (PTT) were initially elevated as a response to head trauma and they returned to more normal values within 1-2 days on their own. The State's Expert Witness' statements are not supported by medical facts. Alexa's PT (33.3 seconds) and PTT (> 100 seconds) levels were elevated on November 16 because she was suffering from vitamin K deficiency. PT and PTT are important markers for vitamin K deficiency (Section IV-C). Her PT and PTT levels were reduced by more than two-fold by November 18 because of her treatment with fresh frozen plasma (FFP) as shown in Tables 7 and 13. FFP is efficacious for treatment of factors II, V, VII, IX, X, and XI deficiency.

7) The State's Expert Witness stated that Alexa's pancreatic enzymes were elevated because Alexa suffered from trauma to the head and abdomen. Once again, the State's Expert Witness' theory is not supported by medical facts. Serum amylase and lipase were elevated in this case because Alexa suffered from acute pancreatitis (AP) as I described in Section IV. All physicians who treated Alexa including the medical examiner observed no evidence of abdominal trauma in this case.

On November 16, Alexa's serum amylase level (1026 IU/L) and serum lipase level (358 IU/L) were more than ten-fold and three-fold higher than normal levels, respectively (Table 12). The diagnosis of acute pancreatitis (AP) is usually established by the detection of an increased level of serum amylase and lipase. In addition, the abnormal changes observed by the surgical team who harvested Alexa's organs on November 19 indicated that Alexa suffered from acute pancreatitis. These included bloody intraperitoneal fluid, severe induration of root of mesentery with inflammatory process and fibrin exudates, and hematoma of the right upper omentum (Section III).

8) The State's Expert Witness stated that Alexa had hyperglycemia on November 16 because she suffered from head trauma. The medical evidence presented in this report indicates that Alexa suffered from diabetes (Section IV). Alexa's blood glucose level at 1419 on November 16 was 504 mg/dL and it increased to 624 mg/dL at 2207. These levels are about fivefold higher than the normal level (Table 8). Alexa's blood glucose levels remained elevated on November 17 and 18 although she was receiving insulin (Tables 13, 14).

In acute pancreatitis, hyperglycemia is common and it is due to multiple factors including decreased insulin release, increased glucagons release, and an increased output of adrenal glucocorticoids and catecholamines (Section IV). In addition, Alexa suffered from metabolic acidosis, elevated anion gap, low serum bicarbonate level, and hypokalemia, all of which are indicators of diabetes mellitus.

9) Alexa's blood test on November 16 at 1419 revealed that her hemoglobin concentration (8.3 g/dL) and hematocrit value (28.8%) were low. The State's Expert Witness stated that Alexa suffered from anemia or blood loss but she did not investigate to find the factual cause of her anemia, which was vital for the outcome of this case. My investigation revealed that Alexa's anemia was not a result of blood loss on November 16 but instead it resulted from her chronic loss of appetite, occasional vomiting, and her frequent bowel movements.

On July 20, Alexa's hemoglobin level was 11.4 g/dL and it was reduced by 27% to 8.3 g/dL in four months. The CT scan of the brain taken at 1615 on November 16 showed the presence of focal subarachnoid hemorrhage and the amount of blood released was less than 2 mL. Alexa's body weight was 8.9 Kg and her blood volume should be about 534 mL. Normal blood volume in human is about 6-7% of body weight. The loss of 2 mL of blood would have reduced the concentration of her hemoglobin by about 0.4% to bring it to 11.39 g/dL and not 8.3 g/dL as occurred in this case. This simple calculation clearly proves that Alexa's anemia did not result from bleeding on November 16.

10) The State's Expert Witness stated that the bleeding and edema observed in Alexa's case (subdural hemorrhage in the brain and spinal cord, brain edema, bleeding in the eye, extracranial bleeding and edema, and bleeding under the skin on Alexa's back) were caused by blunt trauma to the head and abdomen and violent shaking. The clinical evidence described in Sections II-V of this report clearly shows that the bleeding and edema described above progressively developed following Alexa's admission to the hospital and that they were not caused by trauma as the State's Expert Witness asserted.

They developed as a result of the synergistic actions between bacterial infections, complications of infections, and medications given to Alexa in the hospital. These included acute pancreatiti, hypotention, hypoxia, and Alexa's treatment with high therapeutic doses of epinephrine, dopamine, sodium bicarbonate, fluid, and excessive amounts of heparin (Tables 7, 13, 14).

11) The State's Expert Witness stated that blunt trauma to the abdomen and violent shaking broke Alexa's T-10 vertebrae and caused damage in her abdominal cavity. The State's Expert Witness stated that in shaking, the mechanism of vertebral injury is related to the abuser's hands encircling the rib cage. She claims that it explains the vertebral column soft tissue hemorrhaging and the epidural and subarachnoid hemorrhaging seen around the 8^{th} and 10^{th} thoracic vertebrae identified during the autopsy.

The clinical evidence presented in Sections II-IV and summarized in Section V-F of this report contradicts the State's Expert Witness' theory. The lytic lesions described in the T-10 vertebrae represent osteomyelitis and not a fresh fracture. The hemorrhage was fresh and less than 12 hours old. Alexa's T-9 vertebrae and the soft tissue surrounding it were free of hemor-

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rhage and injuries. If Kathleen had put tremendous pressure on Alexa's vertebral column to break T-10 and cause bleeding in T-8, then we would expect to see bleeding in the tissues associated with the T-9 vertebrae as well.

Furthermore, the development of lesions observed in Alexa's abdominal cavity were progressive. There was no evidence of parenchymal organ injury, injuries to their blood supplies, nor perforation of the intestine that indicated trauma as shown by the CT scans taken on November 16 and 17. Also, the surgeons who harvested Alexa's organs on November 19 as well as the Medical Examiner who performed the autopsy stated that there was no evidence that indicated that the abdominal region had been struck by blunt trauma in this case.

Additionally, the hematoma of the right upper omentum observed by the surgeons on November 19 was caused due to the damage of the blood vessel walls by the pancreatic elestase and not by trauma. The clinical evidence presented in Table 10 shows that the bleeding occurred between 0400 and 2042 on November 17. At 0400 Alexa had a normal red blood cell count of 4.33 x 10^6/μL, hemoglobin of 13.4 g/dL, hematocrit of 38.8% and platelet count of 214,000/μL. At 2042, the red blood cell count, hemoglobin, hematocrit, and platelet count were reduced to 3.16 x 10^6/μL, 9.5 g/dL, 22.2%, and 117,000/μL, respectively.

It seems that the State's Expert Witness overlooked the markers of acute pancreatitis in Alexa's case which included elevated levels of amylase and lipase enzymes in serum, bloody intraperitoneal fluid, induration of root of mesentery with inflammatory process and fibrin exudates, severe inflammation in the area of the infrahepatic vena cava and the upper portion of the right kidney, hematoma of the right upper omentum, coagulopathy; hypotension, and edema. She also overlooked the fact that Alexa was given excessive amounts of heparin (11.2 times the therapeutic dose) on November 19 that further exacerbated the bleeding.

12) The State's Expert Witness stated, "Alexa's left posterior 8th rib fracture had callus formation indicating that it had begun healing and was estimated to be at least several weeks old at the time of her death. This type of fracture is only consistent with inflicted trauma and could not have been the result of normal toddler activities." The State's Expert Witness did not do any investigation to find out who caused the rib fracture in this case. However, she used twisted logic in her report to link Kathleen indirectly as the person who caused the fracture [2]. She stated,"forceful squeezing of the rib cage and violent shaking are both mechanisms of injury associated with posterior rib fractures." She also claimed that forceful squeezing of Alexa's back and violent shaking caused the fracture and the bleeding in the T-10 and T-8 vertebrae.

There is an obvious problem with the State's Expert Witness' logic. The rib fracture was several weeks old, yet the T-10 fracture was supposedly fresh and occurred on November 16. These two events did not occur at the same time. The likely cause for the bone lesion observed in Alexa's rib #8 was vitamin K deficiency as I explain in Section V-G of this report. Vitamin K is important for bone calcification. Alexa's bone lesion was likely caused by a defect in bone metabolism locally, which followed by healing.

13) The clinical data show that Alexa suffered from otitis media and mastoditis bilaterally. The State's Expert Witness did not think that there was a link between Alexa's ear infection and her injuries on November 16 and therefore ignored this important issue. My investigation revealed that Streptococcus pneumonia and Haemophilus influenza are the primary causes of bacterial ear infection in children. It is likely that Alexa's ear infections were caused by these bacteria as I described in Section V-H of this report. In addition to otitis media, these bacteria also cause osteomyelitis in children and Alexa also had osteomyelitis of the T-10 vertebrae.

The clinical data presented above and in this report clearly indicate that the State's Expert Witness did not conduct a scientific investigation in this case, that her investigation was incomplete and her conclusions were invalid. She did not consider the adverse reactions of vaccines and medications given to Alexa in her evaluation of this case. I believe that the State's Expert Witness' unscientific investigation and invalid conclusions strongly influenced and prejudiced the jury into believing that Alexa died as a result of violent shaking and blunt trauma, which affected the outcome of the trial.

I was shocked to read the State's Expert Witness' unscientific criteria that she set to diagnose cases of Shaken Baby Syndrome. She presented these criteria in her court testimony [26]. She stated that children who suffered from repeated violent shaking had the following features in common:

1) They may have outside external injury or they may not.
2) They may have bone fractures or they may not.
3) They may have retinal hemorrhage in two eyes, or one eye, or they may not.
4) They may have evidence of blood in the brain; a brain bruise or they may not. (She stated that sometime, we do not see bleeding in the brain even in fatal cases).
5) They may have evidence of diffuse axonal injury in the brain or they may not.
6) They do not have a lot of swelling in the brain, except in severe cases.
7) They have small subdural hemorrhage and in a typical case of SBS the hemorrhage was about five to ten cc's and sometimes a little more.
8) Their parents or caretakers usually deny that they harmed the children.

It seems that the criteria in which the State Expert Witness employs to diagnose cases of Shaken Baby Syndrome (SBS) will include any infant or toddler who develops minimal subdural hemorrhaging in the hospital or outside the hospital. Serious adverse reactions to epinephrine that is administered to children in the emergency room are intracerebral, subdural, and/or subarachnoid hemorrhage, even when given at a low dosage of 0.05 mg subcutaneously. Epinephrine is given to almost all children suffering from cardiac arrest and shock. In addition, vitamin K and vitamin C deficiency, liver disease, and treatment with heparin also cause bleeding in children.

In this case, Alexa was treated with a high therapeutic dose of epinephrine (0.5 mg) given intravenously immediately following her admission to the hospital on November 16 (Table 5). In addition, she was treated with large doses of epinephrine

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In addition, she was treated with large doses of epinephrine during her hospitalization. The amount of epinephrine given to Alexa in the hospital was more than thirty-fold the dose of epinephrine that is capable of causing bleeding in the subdura. Alexa also suffered from vitamin K deficiency, was treated with excessive amounts of heparin, and suffered from acute pancreatitis that caused bleeding. However, the State's Expert Witness did not consider any of these factors in her evaluation of this case.

I believe that the State's Expert Witness' involvement in this case caused great harm to Kathleen, her family and friends. She misled the court and Alexa's family to believe that Kathleen killed Alexa. Furthermore, the criteria used by the State's Expert Witness to diagnose cases of SBS are not supported by medical facts. The use of her criteria will likely put many innocent parents and daycare providers at great risk of being falsely accused and imprisoned for harming their children by violent shaking. The State's Expert Witness' work in this case and her involvement in other cases of alleged SBS should be reviewed by a medical board in order to save innocent people from being accused of horrible crimes that they did not commit.

Section VII. Conclusions and Recommendations

Alexa suffered from cardiac arrest and apnea between 1230 and 1245 on November 16, 1999. The clinical data described in this report clearly show that Alexa's cardiac arrest was triggered by acute pancreatitis and diabetes mellitus and not by violent shaking and blunt trauma as the State alleged. Alexa also suffered from vitamin K deficiency, anemia, acute bacterial infections, oesteomyelitis, otitis media, and mastotidis.

In addition, Alexa did not breathe for about 30 minutes and her brain suffered from severe ischemia and hypoxia that led to diffuse edema and nerve damage. The complications of acute pancreatitis and diabetes caused hypovolemia, metabolic acidosis, reduction of potassium levels in cardiac muscles and nervous tissues, edema, bleeding, and disseminated intravascular coagulation (DIC). Vitamin K deficiency caused bleeding and affected calcium metabolism in the bone.

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

Alexa was vaccinated with four attenuated live viruses vaccines (measles, mumps, rubella, and varicella) on August 13, 1999 when she was suffering from serious chronic health problems. She suffered from immune depression, fungal infection, poor appetite and poor weight gain, frequent bowel movements and soft stool, and vomited on many occasions. She was treated with Nystatin (anti-fungal) for six weeks and the common adverse reactions of Nystatin are diarrhea and vomiting. In addition, she received the MMR vaccines three months earlier than the recommended age in a healthy child (15 months).

Alexa's poor weight gain and her chronic fungal infection caused significant depression in the functions of her immune system, especially the T-cells. It made her response to the administered vaccines inadequate and increased her risk for developing serious adverse reactions to vaccines and predisposed her to infection. The MMR and varicella vaccines caused the following serious illnesses that led to Alexa's cardiac arrest and apnea on November 16, 1999.

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

2) The MMR and varicella vaccines and the viral and bacterial infections caused Alexa to eat less, lose weight, develop anemia, vitamin K deficiency, and led to significant immune depression, especially in the T-cell count and functions. The mumps virus from the vaccine probably overcame Alexa's weakened immune system and infected the pancreatic tissues. The clinical tests and the pathological findings in the abdominal cavities indicated that Alexa suffered from acute pancreatitis, which led to her cardiac arrest and apnea on November 16, 1999.

I reviewed the Medical Examiner's autopsy report and his court testimony in this case and discovered that his autopsy and investigation pertaining to this case were incomplete (Section V). He misinterpreted the clinical data including the results of his own tests. He presented the wrong conclusions to the police and the court regarding the causes of injuries and death in this case (Section V). His work led to the false accusation, arrest, and conviction of Kathleen Butcher for a horrible crime that she did not commit. The Medical Examiner's work should be reviewed by the government and state medical board in order to save other innocent people from being falsely accused of killing children by the so called "Shaken Baby Syndrome."

I also reviewed the State's Expert Witness' report and her testimony given in this case (Section VI). She declared that Alexa's injuries were caused by violent and repeated shaking and blunt trauma to the head and abdomen after she ate lunch at 1215 on November 16. The State's Expert Witness' theory and conclusions are not supported by the medical facts and evidence presented in this report. She mainly based her conclusions on the Medical Examiner's incomplete autopsy report. Additionally, she added a new false allegation to this case by alleging that Alexa had been struck by blunt trauma to the abdominal region. Her accusation was not supported by the surgeon who harvested Alexa's organs, the results of the CT scans taken of Alexa's abdominal region, or by the findings observed by the medical examiner during autopsy.

I believe that the State's Expert Witness' involvement in this case misled the court and Alexa's family into believing that

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Kathleen killed Alexa. She caused great harm to Kathleen and her family. Furthermore, the criteria used by the State's Expert Witness to diagnose cases of SBS are not supported by medical facts. The use of her criteria will likely put many innocent parents and daycare providers at great risk of being falsely accused, wrongly convicted, and imprisoned for harming children by violent shaking. The State's Expert Witness' work in this case and her involvement in other cases of alleged SBS should be reviewed by a medical board in order to save many innocent people from being accused of horrible crimes that they did not commit.

The extensive medical evidence presented in this report clearly shows that Alexa, her family and Kathleen, and her family are all the victims of a broken medical system that need to be urgently fixed. Alexa died as a result of adverse reactions to vaccines and medications. Kathleen was convicted and put in prison because of sloppy and incomplete medical investigations. Alexa was given four live virus vaccines without any consideration for her chronic illnesses and her immune depression. The nurse and the physician who gave those vaccines to Alexa should bear the responsibility for injuring her.

I believe that Howard County and the State of Maryland have the responsibility to review the evidence presented in this report. It clearly shows that Kathleen is innocent and that they should take immediate action to free her from prison so that she may be reunited with her five children, (ages 2-11 years) and husband. Additionally, the State should investigate the involvement of the Medical Examiner and the State's Expert Witnesses in similar cases that have resulted in the conviction of parents or caretakers accused of killing children by SBS. The physicians who caused Alexa's death and her family's suffering should be held responsible for compensating Alexa's family for the loss of their child, as well as for their suffering. Furthermore, I believe that these doctors should compensate Kathleen and her family for their pain and suffering, including payment of all expenses relating to this ordeal.

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

I spent approximately 300 hours evaluating the medical evidence in this case in order to find the factual causes of death and to write this detailed report. It is my hope that the State of Maryland, our Federal Government, physicians, and our society will take the time to review the evidence and that they will act promptly to resolve the problems. The Shaken Baby Syndrome theory should be re-evaluated. This is the third case that I have evaluated within a 10-month period involving child, who died as a result of adverse reactions to vaccines. In all three cases, either their parents or their caretaker were falsely accused of murder and imprisoned. Differential diagnosis should be used to solve complicated medical problems such as these, as I used in this case to find the factual causes of the problem.

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