Analysis of causes that led to baby Alan Ream Yurko's cardiac arrest and death in November of 1997

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Abstract

In November of 1997, Alan R. Yurko was accused of, and arrested for, killing his son, the 2½-month-old baby Alan Ream Yurko, by vigorous shaking of the head. Mr. Yurko was convicted by a jury in 1999 and sentenced to spend his life plus ten years in prison. I evaluated baby Alan's case by reviewing the baby's medical records, H & E stained tissue sections of Alan's organs obtained at the time of autopsy, the autopsy report, Francine's medical record during her pregnancy with Alan, the trial document and testimonies of expert witnesses, and related published medical literature. I used differential diagnosis to evaluate the contribution of causes and the synergistic actions among these causes that led to the cardiac arrest, apnea, subdural bleeding, and death in this case. I determined that baby Alan died as a result of adverse reactions to vaccines and medications that were given to him by the healthcare providers and his father is innocent. Furthermore, the shaken baby syndrome theory is not supported by science and should be re-evaluated. A better understanding of the biological mechanisms of various medications and vaccines well help to save infant lives and protect vital resources in regard to the caretakers.

Keywords: Shaken Baby Syndrome, adverse vaccine reactions, subdural bleeding, death

1. Introduction

In November of 1997, Mr. Alan R. Yurko was accused of, and arrested for, killing his son, the 2½-month-old baby Alan Ream Yurko, by vigorous shaking of the head. Mr. Yurko was convicted by a jury in 1999 and sentenced to spend his life plus ten years in prison. Mr. Yurko and his wife, Francine, requested that I evaluate their case to find the factual cause(s) that led to baby Alan's cardiac arrest and death in November of 1997. I evaluated their case by reviewing the baby's medical records, H & E stained tissue sections of Alan's organs obtained at the time of autopsy, the autopsy report, Francine's medical record during her pregnancy with Alan, the trial document and testimonies of expert witnesses, and related published medical literature. I used differential diagnosis to evaluate the contribution of causes and the synergistic actions among these causes that led to the cardiac arrest, apnea, subdural bleeding, and death in this case.

I present my review and analysis of Francine's health problems during her pregnancy with Alan in Section 2 of this report. Section 3 contains a detailed description of baby Alan's health problems from the time of birth on September 16, 1997 to the day of his cardiac arrest on November 24, 1997, and my analysis. In Section 4, I describe the clinical events during Alan's five days in Princeton and Florida hospitals, and my analysis of these events. My detailed review and analysis of the medical examiner's autopsy report and his court testimony are presented in Section 5. My review of the testimonies of the other state witnesses and the defense witness is described in Section 6. Section 7 contains my conclusions and recommendations.

Baby Alan was born five weeks premature on September 16, 1997 by induced labor because his mother, Francine, suffered from oligohydramnios. Francine also suffered from multiple chronic illnesses during her pregnancy that included gestational diabetes, anemia, loss of appetite, spastic colon, urinary tract and vaginal bacterial infections. She gained only two pounds during her entire pregnancy (Section 2). The baby spent the first week of his life in the hospital because he suffered from respiratory distress syndrome, jaundice, hypoxia, hypoglycemia, and bacterial infections (Section 3). At day three following his birth, Alan's serum bilirubin level was l7.4 mg/dL which is capable of causing encephalopathy.

Review of the medical literature revealed that gestational diabetes, oligohydramnios, and jaundice have tremendous negative impact on the prenatal, natal, and postnatal developments. These conditions caused increases in mortality rate, congenital anomalies, growth retardation, skeleton deformities, rate of premature labor, respiratory distress syndrome, hypoxia, hypoglycemia, encephalopathy, and rate of infections in the newborn (Section 3.1).

Baby Alan was released from the hospital one week following his birth, with jaundice and respiratory system problems. He continued to have symptoms of chest congestion and difficulty in breathing following discharge from the hospital. He

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gained only 0.5 pounds (227 g) in his first twenty-four days of life. However, his growth was improved during his second month of life. He gained 2.7 pounds (1.22 kg).

On November 11, 1997, at two months of age, Alan was given six vaccines (DTaP, Hib, OPV and Hepatitis B) and sent home without monitoring or medical supervision. At l0 or 11 days following the vaccine injections, the baby developed a high-pitched cry, his skin became warm to touch, and there was increasing lethargy. His mother had been told in the doctor's office that these symptoms might result following these vaccinations. This led her not to worry about her baby's symptoms and not to call his doctor. On November 24th, at 13 days post vaccination, the baby had a cardiac arrest and apnea episode, and his father took him to the emergency room at Princeton Hospital in Orlando Florida (Section 3.2).

Serious adverse reactions, such as apnea, cardiorespiratory problems, and oxygen desaturations that require medical intervention are commonly associated with vaccination of preterm infants. Preterm babies who were vaccinated at 70 days of age or less, similar to baby Alan, developed the most serious adverse reactions to vaccines. The authors of many well-documented studies concluded that the risk and benefit of vaccination in preterm infants should be evaluated prior to administering vaccines (Section 3.3). They also emphasized that preterm infants who received vaccines should be monitored. Adverse reactions to vaccines that were administered to baby Alan are not limited to preterm infants. They have also been reported in full term infants. For example, in the US, reports to the passive Vaccine Adverse Event Reporting System (VAERS), concerning infant immunization against pertussis between January 1, 1995 and June 30, 1998 revealed 285 cases of death and 971 cases of nonfatal serious illnesses (Section 3.3).

On November 24th, baby Alan was admitted to Princeton Hospital with cardiac arrest and apnea; he was then resuscitated. The first examination revealed that he was flaccid, his corneas were somewhat cloudy, and he had gastric ulcer. There was no injury caused by trauma found on his head or his body, except for a small reddish linear bruise under the right eye. His four-year-old sister caused this minor injury accidentally when she was handing a baby bottle to her father. The baby's first blood test revealed that he suffered from metabolic and respiratory acidosis (pH of 7.18), diabetes (blood glucose level of 337 mg/dL and Anion gap level of 22 mEq/L), anemia, elevated serum liver enzymes and LDH. He also had elevated white blood cell count (20,900/μL) and platelet count (571,000/μL). The baby was treated with high therapeutic doses of three antibiotics, rocephin, gentamicin, and Claforan (cefotaxime sodium), to fight the bacterial infections. He was also given IV fluids and dopamine, then transferred to Florida Hospital at about 2:00 PM on November 24th.

At Florida Hospital, the baby's temperature rose to 105.8ºF (41.0ºC), and his blood glucose reached 397 mg/dL on November 24th. The treatment with three types of antibiotics reduced his temperature, blood glucose level, and serum enzymes. On November 26th, his serum glucose level dropped to a normal level of 95 mg/dL (76% reduction); the LDH, alkaline phosphate, and SGPT levels dropped by 70%, 47%, and 19%, respectively; and the white blood cell count by 35%. These data clearly indicate that the baby had liver and pancreas bacterial infection and maybe heart bacterial infection, and that the infections were resolved because of the treatment with antibiotics. The baby also suffered from hypotension, dysrhythmia, dehydration and weight loss (lost 1.05 pounds, or 476 g, in five days). The baby was given IV fluid, plasmanate and red blood cells, heparin, potassium, dopamine, and antidiuretic hormones.

Furthermore, the baby was treated with excessive amount of sodium bicarbonate on November 24th. The blood pH increased from 7.10 to 7.67, and this treatment caused metabolic alkalosis, hypoxia, hypokalemia, and cerebral edema. At high blood pH, the release of oxygen from hemoglobin to the tissues is reduced significantly. In addition, the baby was also given heparin at 2:45 PM at an estimated high dose level of 219 IU/kg per hour. At 3:15 PM, blood analysis showed elevated prothrombin time and fibrinogen split product level. Heparin given to patients suffering from anemia, hypotension, and unexplained symptoms similar to baby Alan's has caused serious hemorrhagic events. A computerized tomography scan of the brain taken at 7:50 PM showed a subdural hematoma on the right side of the brain, and intraparenchymal hemorrhage. Based on the hourly heparin dose (219 IU/kg per hour), the estimated total dose infused in five hours was 1095 IU/kg, which is about 8.8 times the recommended maintenance dose for infants of 125 IU/kg per five hours.

Unfortunately, the baby was treated again with excessive doses of sodium bicarbonate and heparin on November 25th, despite his problems with metabolic alkalosis (pH 7.61) and bleeding in the brain. This treatment caused metabolic alkalosis, hypoxia, hypokalemia, cerebral edema, and bleeding. His serum potassium level dropped from 4.9 mEq/L to 2.3 mEq/L. Also, baby Alan suffered from disseminated intravascular coagulation (DIC) as a result of his treatment with heparin. The platelet count prior to the administration of heparin on November 24, 1997 was 571,000/μL of blood, and dropped to 397,000/μL (30.5% reduction) on November 25th. Heparin increases the tendency of the platelets to aggregate and form a clot. Also, blood analysis performed at about 30 minutes post-heparin infusion, shows increased fibrinogen split product level (160 μg/mL) and prothrombin time (14.6 seconds). These values are 1600% and 115% of normal, respectively. These values returned to normal on November 26th following the cessation of the treatment with heparin.

On November 24th, chest x-ray showed bilateral pulmonary infiltrates and healing fracture of the 6th rib. My review of the medical literature revealed that rib fractures have been reported to occur during labor, and that these fractures were missed during the initial examination of the baby. In addition, the mechanism of rib fractures during labor was explained in the medical literature (Section 5.10). It has also been stated that the specific clinical manifestations of ribs fractures are often absent, making diagnosis difficult.

Baby Alan was pronounced (brain) dead on November 27, 1997—about 75 hours after the initial hospital admission. On November 29, the Chief Medical Examiner performed an autopsy whose main objective was to establish the cause(s) of death. Prior to autopsy, the baby's heart, liver, pancreas, and a portion of the intestine were taken by Translife for organ transplant. The baby was given a megadose of heparin (22,950 IU) prior to and during organ harvesting, to prevent the formation

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of blood clots in organs. The Chief Medical Examiner concluded that baby Alan died of bleeding in the brain resulting from vigorous shaking of the baby by his father, Alan Yurko.

My review of the Chief Medical Examiner's autopsy report indicates that it lacks the accuracy and the expected minimum scientific detail to make it reliable and useful for revealing the cause of death. For example, the Chief Medical Examiner described the histology of the heart in his autopsy report, but the heart was donated prior to autopsy. Therefore, he did not have the chance to examine it. He did not present a description of the microscopic appearance of the meninges and the presence of axonal injury in the brain and spinal cord. There is no description of his x-ray findings on the rib fractures. In addition, the Chief Medical Examiner's measurement of 22 cm for the head circumference was obviously wrong. It was 37.5 cm at 18 days prior to the autopsy (Table 4).

Furthermore, the Chief Medical Examiner's description of the bleeding in the subdural spaces of the brain and spinal cord indicates that the bleeding occurred in at least three stages during a 2-5-day period, and it does not support his claim that bleeding occurred within a few minutes or a few seconds. Also, the presence of hemorrhage in the lungs, brain, and spinal cord, and the presence of cerebral edema does not support his claim that the bleeding was caused by vigorous shaking of the head, but shows that it was caused by metabolic and cardiovascular problems.

Alan R.Yurko's jury trial took place February 22 to 24, 1999 in the state of Florida. The prosecutor provided four major witnesses testifying for the state, and two of these were called for repeat appearances before the jury, following that of the defense witness. Against these witnesses the defense provided a single witness. The state witnesses were: 1) the Chief Medical Examiner; 2) Dr. Gary Pearl, a consultant neuropathologist (testified twice); 3) the Treating Physician; and 4) the General Pediatrician (testified twice). The defense witness was Dr. Douglas Radford Shanklin, a pathologist. Three state witnesses (the Chief Medical Examiner, the Treating Physician, and the General Pediatrician) said that baby Alan died as result of Shaken Baby Syndrome. However, none of them provided medical evidence to prove their case, and their testimonies were based only on a theory. The fourth state witness, Dr. Pearl reported that the injuries in the brain and spinal cord were acute injuries and did not start at birth or shortly after birth. He did not say that these injuries were caused by Shaken Baby Syndrome. None of the state witnesses reviewed the baby's prenatal record, his birth record, his doctor's charts during his two months of weekly visits, or adverse reactions to vaccines and medications given to the baby. Also, they did not interview his parents to get the case history. Furthermore, none of these witnesses presented evidence in court to show the presence of axonal injury in the brain (Sections 5 and 6).

In addition, the Chief Medical Examiner presented statements in court that are not supported by his autopsy findings. He stated in court that the cerebrospinal fluid (CSF) was mixed with blood, but in his report he described that the CSF was clear. He also stated in court and in his report that the heart was donated prior to his examination, but he described the histology of the heart in his report. Furthermore, the Chief Medical Examiner stated that the baby did not suffer from meningitis, but his autopsy report and the clinical evidence described in the baby's chart, as well as the pathology evidence presented by Dr. Pearl and Dr. Shanklin, indicate that the baby suffered from meningitis. I also examined the H & E section of the meninges and observed evidence of acute meningitis. The lesions and symptoms described by the pathologists, autopsy report, and the baby's chart that indicate the presence of acute meningitis include swollen blood vessels, congestion, infiltration of meningial tissue with inflammatory cells, brain edema, high white blood cell count, and elevated body temperature of 105.8ºF (41.0ºC). Also, the Chief Medical Examiner overlooked the influence of the treatment with antibiotics on the severity of the lesions in the meninges.

The Chief Medical Examiner presented the minor retinal bleeding in the right eye as evidence that baby Alan died as a result of "Shaken Baby Syndrome," but he did not investigate the factual causes that led to retinal bleeding, such as diabetes, infections, and hypoxia. Furthermore, the Chief Medical Examiner did not provide x-ray findings to prove that Alan had fractures of ribs #5, 7, and 10. Also, he did not search the medical literature to find out if rib fractures occurred during labor. However, he showed in court two photographs of minor contusions in the temporal areas of the head that had occurred in the hospital at about one day prior to autopsy and had no relation to the cause of death in this case. I believe that he did it to influence the thinking of the jury that physical force was used.

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

The Treating Physician and Florida Hospital contacted the Orange County Sheriff's Office and the Child Protective Office on November 24, 1997, and filed a report of child abuse based on the assumption that baby Alan was injured as a result of abuse by his father. Mr. Yurko was arrested on November 26, 1997, while his son was still alive in Florida Hospital. The Treating Physician assumed that Mr. Yurko was guilty of child abuse, but his own examination of baby Alan revealed no injuries caused by trauma except a minor bruise under the right eye. In fact, he treated the baby with excessive doses of sodium bicarbonate and heparin that caused hypoxia and bleeding. Heparin should not be given to an individual suffering from anemia, hypotension, bleeding, and tissue inflammations similar to baby Alan's.

The defense witness, Dr. Shanklin, made very important contributions to this case. He stated that baby Alan's kidneys were not fully developed. His finding might explain the mother's problem with oligohydramnios (Section 3.1.2). He also stated that the baby suffered from pneumonia and meningitis of the brain and spinal cord. His findings might explain the susceptibility of these organs to bleeding caused by treatment with heparin and sodium bicarbonate. I also examined the H &

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E tissue sections of the brain, spinal cord, and lungs, and I observed evidence of acute meningitis in the brain, fresh bleeding in the subdural spaces of the brain and spinal cord, bleeding in the brain and lung, and interstitial pneumonia. The inflammation in these regions affected the integrity of the blood vessels, and this would have predisposed them to leak fluid and blood when the child was treated with excessive doses of heparin and sodium bicarbonate. Additionally, Dr. Shanklin described old neurological injuries to the brain and spinal cord. I believe that the high levels of bilirubin observed in the first week following birth caused these injuries.

During Mr. Yurko's jury trial, the prosecutor, Ms. Wilkinson presented only one theory—that baby Alan died of "Shaken Baby Syndrome" (SBS), and that Alan Yurko, his father, did it. My review of the medical evidence and the trial transcript revealed that the state did not prove that the injuries were caused by trauma, or that Mr. Yurko abused his child. However, the prosecutor still achieved her goal of getting Alan Yurko convicted of a horrible crime he did not commit. He received a life sentence plus ten years. I believe that the prosecutor used questionable practices that violated Mr. Yurko's right to a fair trial. In Section 6 of this report is a list of the prosecutor's unfair tactics, with evidence that the state did not prove its case. For example; the prosecutor did not investigate other causes, such as adverse reactions to medications and vaccines. Also, the prosecutor allowed the Chief Medical Examiner to present as evidence two photographs of minor contusions in the temporal areas of the head that occurred in the hospital at about one day prior to autopsy. The medical examiner's main objective should have been to find the causes of injuries and death, not prejudicing his findings by showing irrelevant photos that would influence a jury.

As a result of problems with our current medical system—the policy of vaccinating premature babies (the treatment given to baby Alan in the hospital) and the bias of the testimony of state witnesses in evaluating evidence, as exemplified in this case—Alan Yurko and his family suffered two tragedies. The first tragedy was the loss of baby Alan because of the adverse reactions to vaccines and the treatment using excessive doses of heparin and sodium bicarbonate given at the hospital. The Yurkos' second tragedy was the conviction of Mr. Yurko of a horrible crime he did not commit. He was convicted because the state's four expert witnesses did not take the time to review the evidence or the related published literature. They did not take the time to sort out the facts and their testimonies were based on theories, not on medical evidence. The prosecutor contributed to the problem by focusing on only one theory.

I believe that the state of Florida has a responsibility to review the evidence presented in this report and to free Mr. Yurko from prison as soon as possible. The state and the medical system should be focused on finding the facts—the causes of the injury and death of children in cases such as this—and preventing these problems from happening again. Accusing innocent parents of abusing and killing their children based on unsupported theory, as it happened in the case of baby Alan, will not prevent the death of another child by vaccines and wrong medications. But it certainly puts innocent people in prison and causes their families unimaginable suffering. It also costs the taxpayers huge sums of money to pay for trials and legal fees. I spent more than 250 hours working on the Yurko case to find the factual causes of death and to write a detailed report. I hope that the state of Florida, the medical system, and our society will take advantage of this opportunity to see the real problems facing premature babies who are receiving vaccines, and, hopefully, take action to put an end to such tragedies.

2. Review of Francine Ream Yurko's medical records during her pregnancy and analysis of her health problems

Francine Ream Yurko is a white female. She was 27 years old when her son, Alan Ream Yurko was born five weeks premature on September 16, 1997. The review of her medical records revealed that she suffered from multiple chronic illnesses during her pregnancy with Alan. She suffered from chronic spastic colon, loss of appetite, dehydration, gestational diabetes, anemia, chronic urinary infection, vaginal infection with group B Streptococcus, and oligohydramnios [1-5]. Her weight was 130 lb (59.0 kg) at the start of her pregnancy in January of 1997, dropped to 120 lb (54.4 kg), and then returned to 130 lb (59.0 kg) on July 19, 1997. At the time of delivery on September 16, 1997, her weight was 132 lb (59.9 kg), so that she had gained only 2 lb (0.907 kg) during her entire pregnancy. The currently recommended weight gain for pregnancy is 25 to 30 lb (or 11.3 to 13.6 kg).

The results of Francine's blood analysis and her glucose tolerance tests performed during her pregnancy with Alan are presented in Tables 1 and 2, respectively. These data indicate she suffered from chronic anemia and gestational diabetes. Her red blood cell count, hemoglobin levels, and the hematocrit were low, and she had high blood and urine glucose levels. The elevated fasting blood glucose levels and the abnormal results of the glucose tolerance tests indicate that her gestational diabetes was serious. The glucose levels in urine of both tests were high. The date of urine and blood analysis indicates that Francine's problem with diabetes probably started at least three months prior to delivery.

Furthermore, the high white blood cell counts and the results of urine culture show that Francine was also suffering from chronic bacterial infections. A urine culture test for bacteria was performed on June 25, 1997 and August 13, 1997. It revealed she had urinary tract Escherichia coli (E. coli) infection. E. coli was identified in both tests at growth levels of 100,000 colony per mL of urine. She was treated with antibiotics.

Furthermore, she was diagnosed with a vaginal infection with group B Streptococcus and treated with amoxicillin and ampicillin on September 15, 1997 at one day prior to delivery. She was also treated with acetaminophen for pain and fever; propoxyphene napsylate for pain; and ferrous sulfate for anemia. In addition, on September 15, 1997, her gynecologist performed an ultrasound prenatal exam and discovered that she had oligohydramnios. She lost the amniotic fluid completely without noticing. This suggests that she lost the fluid gradually over a period of days or even weeks, and/or there was a serious reduction in the production of the fluid.

The discovery of oligohydramnios led to the decision by her doctor to induce labor chemically on September 15, 1997 at 35 weeks gestation. The labor was induced by pitocin. She was

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also given pain medications (nalbuphrine, butorphanol, and promethazine). Baby Alan was born on September 16, 1997 at 2:15 PM, five weeks premature. Francine left the hospital on September 17, 1997 at 4:35 PM without her baby. Baby Alan stayed in the Intensive Care Unit because of his respiratory distress syndrome and other health problems. A detailed description of Alan's health problems following birth is presented in Section 3 below.

Table 1. Blood analysis values for Francine, June through September, 1997

Measurements	6/25*	7/23	8/11	9/15	9/17	Normal Values
Glucose		**175 H**	**201 H**			70-110 mg/dL
WBC	**12.1 H**	9.9	8.6	**12.7 H**		3.0-12.0 x 10^3/ μL
RBC	**3.64 L**	**3.4 L**	**3.4 L**	**3.5 L**		4.16-5.7 x 10^6/μL
Hemoglobin	**11.6 L**	**10.9 L**	**10.9 L**	**11.3 L**		12.1-17.3 g/dL
Hematocrit	**34.3 L**	**31.8 L**	**32.3 L**	**32.9 L**	**29.9 L**	36.5-52%
MCV	94.1	92.6	94.4	93.2		82-99 FL
MCH	31.9	31.8	31.8	31.9		28-40 PG
MCHC	33.8	34.3	33.7	34.2		29-37 g/dL
Platelet	189	165	157	176		150-400 x 10^3/μL

*H: High value; L: Low value

Table 2. Results of the glucose tolerance tests performed during Francine's pregnancy with Alan

Time of Glucose Test (mg/dL)	August 16, 1997	August 25, 1997	Reference Values
Blood			
Fasting	75	83	70-125
1/2 Hour	150	168	70-190
1 Hour	**199 H***	**220 H**	70-190
2 Hour	**167 H**	**199 H**	70-165
3 Hour	**154 H**	143	70-145
Urine			
Fasting	Negative	Negative	Negative
1/2 Hour	Negative	Negative	Negative
1 Hour	**250 H**	**500 H**	Negative
2 Hour	**1000 H**	**1000 H**	Negative
3 Hour	**1000 H**	**1000 H**	Negative

*H: High value

3. Review of Alan Ream Yurko's medical records from the time of birth on September 16th, to November 24, 1997, and analysis of his health problems

3.1 Alan's health problems during the first week of life

Baby Alan was born five weeks premature on September 16, 1997 at 2:15 PM. His weight was 5 lb and 9 ounces (2.52 kg), and head circumference was 31.3 cm. Immediately following birth, the baby had grunting respirations with sternal and rib retractions. The mother observed a persistent grayish color to the baby. At approximately two hours after birth, a blood glucose test revealed the baby had a glucose level of 37 mg/dL, and his follow-up glucose level was 32 mg/dL. His blood glucose levels were below the low normal value of glucose in infants of 45 mg/dL. The baby suffered from hypoglycemia. Furthermore, his arterial blood gasses on room air revealed that he suffered from hypoxia and acidosis. The PCO_2 and PO_2 levels were 42 and 43 mm Hg, respectively. Also, the baby had a low serum creatinine level of 0.4 mg/dL which is 80% of low normal (normal range is 0.6-1.2 mg/dL). Creatinine is a marker of muscle development and a low value indicates that the baby had low muscle mass. The infant was placed in an oxyhood with 50%O_2 to treat his hypoxia. Also, he was treated with ampicillin and gentamicin to fight bacterial infections.

The baby's 7-day hospital course was complicated by continuing respiratory distress and he spent three days in the intensive care unit. A chest x-ray showed persistent pulmonary infiltrates. Furthermore, Alan had neonatal jaundice as indicated by elevated serum bilirubin levels, with a maximum bilirubin level of l7.4 mg/dL at 3 days of age. His serum bilirubin levels are presented in Table 3. These health problems are commonly reported in preterm infants. For example, Sanchez et al. conducted a prospective surveillance of 97 (50 girls and 47 boys) preterm infants younger than 37 weeks of gestation. They found that the majority (64%) of infants had hyaline membrane disease, and 41% developed chronic lung disease (CLD). Also, 93 (96%) infants had experienced apnea of prematurity. The maximum intervention for apnea was theophylline therapy in 21 infants, nasal continuous positive airway pressure (CPAP) in 23 infants, and mechanical ventilation in 45 infants. In addition, 47 infants (48%) had an intraventricular hemorrhage. A total of 33 episodes of sepsis occurred among 26 infants, and 3 of them developed meningitis [6].

Table 3. Alan's Serum Bilirubin levels

Date	Time	mg/dL
9/17/1997	6:20 AM	4.3
9/17/1997	6:00 PM	**6.5 H**
9/18/1997	5:40 AM	**8.0 H**
9/19/1997	10:15 PM	**12.8 H**
9/19/1997	5:15 AM	**17.4 H**
9/20/1997	4:30 AM	**14.6 H**
9/21/1997	5:45 AM	**14.8 H**
9/22/1997	6:30 AM	**12.6 H**
9/23/1997	4:55 AM	**13.2 H**

*H: High value

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Gestational diabetes, oligohydramnios, and jaundice usually have tremendous impact on fetal and postnatal development. Below are brief descriptions of health problems observed during pregnancy and in children associated with these conditions.

3.1.1 Gestational diabetes and associated health risk in fetus and infant

Diabetic mothers have high blood glucose levels. Glucose crosses the placenta and leads to excessive fetal insulin secretion. Infants of diabetic mothers frequently are hypoglycemic at birth, as it happened in the case of baby Alan. His blood glucose level was 32 mg/dL, which is below the low normal value for newborn of 45 mg/dL. The pancreatic islets of these infants are hyperplastic because the mother called on the insulin supply of the fetus during gestation [7-10]. Furthermore, pregnancy in diabetics is usually associated with a higher prenatal mortality (3–5% vs. 1–2% in nondiabetic women) and a higher incidence of congenital anomalies (6–12% vs. 2–3% in nondiabetic) [7].

Also, hyperinsulinemia has been linked to hypoxemia in the fetus. It leads to an increase in oxygen consumption and a decrease in arterial oxygen content. When such a fetus becomes hypoxic, the maternal hyperglycemia accentuates the rise in lactate and the decline in pH in the fetus. There is also increased erythropoietin-induced red blood cell production in response to fetal hypoxia, resulting in polycythemia in the neonate [10:283]. Alan was suffering from hypoxia and acidosis at birth. His arterial blood gasses on room air were 42 mm Hg for the PCO_2 and 43 mm Hg for the PO_2.

Gestational diabetes can also lead to fetal macrosomia (large body size) and increases the risk for birth trauma [7-9]. Macrosomia usually results from the increase of body fat and the stimulation of growth by insulin [9]. Baby Alan's birth weight was 5 lb and 9 ounces (2.52 kg). However, his serum creatinine level was 0.4 mg/dL, which is 80% of low normal (normal range is 0.6-1.2 mg/dL). Creatinine is a marker of muscle development and low value indicates low muscle mass.

Also, infants of diabetic pregnancies can develop hyperbilirubinemia, and factors implicated have included preterm birth and polycythemia with hemolysis. Venous hematocrits of 65–70% have been observed in as many as 40% of these infants. Renal vein thrombosis has also been reported to result from polycythemia [9]. Baby Alan had neonatal jaundice as indicated by high levels of serum bilirubin reaching a maximum level of l7.4 mg/dL at 3 days of age (Table 3).

Furthermore, infants of diabetic mothers, as contrasted with those of nondiabetic mothers, have almost a six-fold increased risk of developing respiratory distress syndrome (RDS), even when all confounding variables are taken into account. The incidence of RDS is also inversely proportional to the gestational age. It is estimated to occur in about 15–20% of those born between 32 and 36 weeks. Immaturity of the fetal lung in preterm infants is the basis of surfactant deficiency. The production of surfactant increases gradually after the appearance of type II alveolar cells, but the largest increase occurs after 35 weeks of gestation [11:483]. It appears that baby Alan had two risk factors for RDS–premature birth at 35 weeks and a diabetic mother. These facts explain the severity of his RDS condition. He spent three days in the intensive care unit immediately after birth and one week in the hospital as a result of this illness. Also, his respiratory problem continued after leaving the hospital.

3.1.2 The impact of oligohydramnios on the infant's health and development:

On September 15, 1997, Francine visited her gynecologist for a prenatal exam. He performed an ultrasound exam and discovered that she was suffering from oligohydramnios. She lost the amniotic fluid completely without noticing. Her condition indicated that she had premature rupture of the membrane and/or there was a serious fetal development problem that led to a reduction in renal output. The discovery of oligohydramnios led to the decision by her doctor to induce labor chemically on September 15, 1997 at 35 weeks of gestation. Labor was induced by pitocin.

Oligohydramnios is thought to be a reflection of fetal compromise in most circumstances. A decrease in placental perfusion results in decreased blood flow and therefore decreased oxygen delivery to the fetus. There is also decreased renal perfusion by the preferential shunting of blood to the fetal brain. Decreased renal perfusion results in decreased urine output, which leads to a decreased amniotic fluid volume. Amniotic fluid production increases from 120 mL per day at 20 weeks to 1200 mL per day at term [12].

Dr. Douglas Shanklin, the defense expert witness (pathologist) examined the H & E stained kidney section of baby Alan microscopically and found that his kidneys were not fully developed. He concluded that the child had a developmental problem [13]. Dr. Shanklin's finding may explain the mother's problem with oligohydramnios. Alan's kidneys were underdeveloped and that led to the reduction in both urine output and the volume of the amniotic fluid.

Oligohydramnios is commonly associated with fetal growth retardation, increased risk of preterm delivery, and admission to a neonatal intensive care unit. In a study involving 7582 high-risk obstetric referral patients, the corrected prenatal mortality (PNM) rate for patients with normal amniotic fluid volume was 1.97 in 1000. In patients with oligohydramnios, the PNM rate increased over 5-fold to 10.4 in 1000 [11:141].

The patient with oligohydramnios is three times more likely to deliver preterm and 30 times more likely to be induced for fetal indications than those with normal amniotic fluid volume [12:354]. Birth weight less than 10th percentile for gestational age at delivery is also common in pregnancies with oligohydramnios. Chauhan et al. did a MEDLINE search and reviewed all studies published in English between 1987 and 1997 that correlated antepartum or intrapartum amniotic fluid index with adverse peripartum outcomes. They found that an antepartum or intrapartum amniotic fluid index of $\leq$5.0 cm is associated with a significantly increased risk of cesarean delivery for fetal distress and with a low Apgar score at 5 minutes. They also found a few reports linking amniotic fluid index and neonatal acidosis [14].

Also, fetal deformities have been observed in conditions of chronic oligohydramnios. It is generally associated with genitourinary tract anomalies that inhibit urination (renal agenesis, polycystic kidneys, and urinary obstruction). Prolonged oligo-

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hydramnios, particularly during the critical period of fetal pulmonary development, can cause pulmonary hypoplasia. Positional deformities (skeletal and facial abnormalities) are also common in chronic oligohydramnios (12). Furthermore, the antepartum testing records of 779 women seen over a 12-month period were reviewed and it has been found that antepartum oligohydramnios is associated with an increased risk of fetal heart rate abnormalities [15].

3.1.3 The pathology of jaundice in infants

Baby Alan suffered from neonatal jaundice. His serum bilirubin levels were l7.4 and 13.2 mg/dL at 3 and 7 days of age, respectively (Table 3). In the full-term newborn, physiologic jaundice is characterized by a progressive rise in serum 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. During the period from the fifth to tenth day of life in white infants, serum bilirubin concentrations 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 [12].

Bilirubin is one of the products of heme catabolism. It is a weak acid and not water soluble 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 dysfunctions in the newborn ranging from transient mild encephalopathy to permanent severe neurological impairment secondary to neuronal necrosis (12:1324). A mean peak unconjugated bilirubin concentrations of 10 to 12 mg/dL may cause bilirubin encephalopathy in certain high-risk, low-birth-weight neonates [12:1317]. Brain stem auditory evoked response (BAER) studies showed significant prolongation of latencies of waves III, IV-V, and interpeak I-III and I-V in neonates with moderate unconjugated hyperbilirubinemia (10 to 20 mg/dL) compared with those of similar gestational and postnatal ages without hyperbilirubinemia. This suggests interference with brain stem conduction.

Furthermore, 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. The term kernicterus has been traditionally used to describe the pathology of bilirubin toxicity within the brain (necrosis followed by gliosis).

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. Ampicillin and other antibiotics can displace bilirubin from albumin and make it free, which enhances the CNS toxicity of bilirubin. Hypoxemia also increases CNS permeability to bilirubin. Baby Alan had moderate bilirubin levels of l7.4 and 13.2 mg/dL at 3 and 7 days of age, respectively. However, his treatment with antibiotics (ampicillin and gentamicin) that bind with albumin might have increased the toxicity of bilirubin by increasing the level of unbound bilirubin [12]. He also suffered from hypoxia, which also increases bilirubin toxicity.

Dr. Douglas Shanklin, the defense expert witness (pathologist), examined the H & E stained sections of the brain and spinal cord in Alan's case and found evidence of neurological damage in both brain and spinal cord that occurred at about 10-12 weeks prior to the baby's death. He found evidence of nerve cells necrosis and axonal injury in the brain and spinal cord. He also reported the replacement of nerve tissue by hundreds of small blood vessels. Furthermore, he observed an old infarction and deposition of calcium in the spinal cord [13]. I believe that bilirubin caused these old lesions in the brain and spinal cord in this case.

3.2 Case history of baby Alan from one week of age to the time of his hospitalization on November 24, 1997

Baby Alan was released from the hospital after one week following his birth with jaundice and respiratory system problems. His mother stated that Alan continued to have symptoms of chest congestion and difficulty breathing following discharge from the hospital. She observed grunting and raspy breathing patterns with occasional brief periods of apnea. Also, the baby remained grossly jaundiced for a month after returning home, much longer than would have been expected from benign neonatal jaundice [4]. He also showed very slow growth rate in the first four weeks of his life. He gained only 0.5 lb (227 g) during his first twenty-four days of life (Table 4). Furthermore, review of his doctor's chart from October 10, 1997 through November 11, 1997 revealed that he suffered from nasal congestion and constipation [16].

Table 4. Baby Alan's growth measurements

Date (mm/dd)	Weight (lb)	Height (Inches)	Head Circum. (cm)	Age (weeks)	Remarks
09/16	5.56			Birth weight	35 weeks gestation
10/01	5.63	18.5	33.3	2.0	Doctor visit
10/10	6.13		34.5	3.4	Doctor visit
11/11	8.88	21.0	37.5	7.9	Vaccination
11/24	10.05			9.7	Hospital admission weight
11/29	9.00	22.0	**22.0***	10.4	Autopsy

*This value obtained from the Medical Examiner's autopsy report. It is obviously wrong.

Despite Alan's multiple health problems, as described above and the five-week premature birth, he was administered six vaccines simultaneously on November 11, 1997 at approximately 8 weeks of age and sent home without monitoring and

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medical supervision. The vaccines included DTaP, Hib, OPV and Hepatitis B. The compositions of the vaccines as reported in the PDR [17] are presented in Table 5.

His mother stated that the baby developed a high-pitched cry, his skin became warm to touch, and there was increasing lethargy at about l0 or 11 days following administration of these vaccines (3-4 days prior to his cardiac arrest on November 24, 1997). She had been told by his doctor that he might experience these symptoms and this led her not to worry about her baby's symptoms and not to call his doctor [4, 16]. These vaccines have been known to cause serious health problems, especially in premature infants. A detailed description of adverse reactions of vaccines given to baby Alan in premature and healthy children is presented below.

Table 5. Composition of vaccines administered to baby Alan on November 11, 1997 as described in the Physicians' Desk Reference (PDR)

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

3.3 Adverse reactions to vaccines in premature and healthy children

Serious adverse reactions to the vaccines given to baby Alan (Table 5) that require medical intervention (such as apnea and cardiac problems) are commonly observed in preterm infants. The authors of many well-documented studies concluded that the risk and benefit of vaccination in preterm infants should be evaluated prior to administering the vaccines. They also emphasized that preterm infants who receive vaccines should be monitored. The following are descriptions of several selected studies conducted in the US and other countries to illustrate these points.

3.3.1 Case histories of 45 preterm babies who were vaccinated with DTP/Hib (diphtheria, tetanus toxoids, and pertussis/Haemophilus influenzae type B conjugate) in the neonatal intensive care unit of the Royal Gwent Hospital, Newport, UK between January 1993 and December 1998 were studied retrospectively [18]. Apparent adverse events were noted in 17 of 45 (37.8%) babies: 9 (20%) had major events, i.e., apnea, bradycardia or oxygen desaturations, and 8 (17.8%) had minor events, i.e., increased oxygen requirements, temperature instability, poor handling and feeding intolerance. Age at vaccination of 70 days or less was significantly associated with increased risk ($p < 0.01$). Of 27 babies vaccinated at 70 days or less, 9 (33.3%) developed major events compared with none when vaccinated over 70 days. The authors concluded that vaccine-related cardiorespiratory events are relatively common in preterm babies. Problems were much more common if vaccine is administered at or before 70 d. These babies should therefore be monitored post-vaccination. Baby Alan was vaccinated at 57 days of age and sent home without monitoring and medical supervision.

3.3.2 Apnea is a respiratory pause of 20 seconds or longer, usually associated with bradycardia, heart rate less than 80 beats/minute. After the occurrence of apnea in two preterm infants following immunization with DTP and Hib, Sanchez et al. conducted a prospective surveillance of 97 preterm infants (50 girls, 47 boys) younger than 37 weeks of gestation who were immunized with DTP (94 also received Hib at the same time) in a neonatal intensive care unit in Texas, US to assess the frequency of adverse reactions, and, in particular, the occurrence of apnea. For each infant, data were recorded for a 3-day period before and after receipt of the immunization [6]. Their study showed that apneic episodes occurred in 34 infants (34%) after immunization. Twelve (12%) infants experienced a recurrence of apnea, and 11 (11%) had at least a 50% increase in the number of apneic and bradycardiac episodes in the 72 hours after immunization. This occurred primarily among smaller preterm infants who were immunized at a lower weight ($p = 0.01$) and who had experienced more severe apnea of prematurity ($p = 0.01$) and had chronic lung disease ($p = 0.03$). Some of these infants required new medical interventions for the increased episodes [6].

3.3.3 Botham et al. conducted a prospective study of 98 preterm infants (53 males, 45 females), of gestational age 24-31 weeks who were immunized at approximately 2 months postnatal age with diphtheria-tetanus-whole-cell pertussis vaccine (DTPw) in the neonatal intensive care unit (NICU) at King George V Hospital in Sydney, Australia. Half the infants also received Haemophilus influenzae type b conjugate vaccine (Hib) simultaneously. All infants were monitored for apnea and bradycardia in the 24 hr. periods pre- and post-immunization. The study showed that only one infant had apnea and/or bradycardia pre-immunization, compared with 17 post-immunization. For 12 infants these events were brief, self-limiting and not associated with desaturations (oxygen saturation < 90%). However, for five infants (30%), these events were associated with oxygen desaturation, and two of these infants required supplemental oxygen. When considering immunization for preterm infants, the benefits of early immunization must be balanced against the risk of apnea and bradycardia [19].

3.3.4 Slack et al. from the United Kingdom stated that four premature infants developed apneas severe enough to warrant

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resuscitation after immunization with diphtheria, tetanus, pertussis (DTP), and Haemophilus influenzae B (Hib). One required intubations and ventilation. They also reported that although apneas after immunization are recognized they are not well documented. They concluded that it is time for further research to elucidate the best time to immunize such infants [20].

3.3.5. Botham et al. conducted a prospective study of 97 preterm infants who were immunized with diphtheria-tetanus-pertussis to document respiratory and cardiac events [21]. The mean gestational age at birth was 28.1 weeks (range 24-34) and the mean age at immunization was 80.6 days (range 44-257). They found that nineteen (20%) infants developed apnea or bradycardia within 24 hours of immunization. The infants who developed apnea and/or bradycardia had a younger gestational age at birth than those who did not (P = 0.03), were artificially ventilated for longer (P = 0.01), and were more likely to have a diagnosis of chronic lung disease (P = 0.006). Two infants who developed concurrent upper respiratory tract infections required additional oxygen, and one of these was treated with oral theophylline. Botham et al. stated that cardiorespiratory function should be monitored after immunization in very preterm infants who had prolonged ventilatory support and/or chronic lung disease.

Adverse vaccine reactions that baby Alan experienced are not limited to preterm infants–they have also been reported in full term infants. Below are brief descriptions of four selective studies that describe the incidence of illnesses associated with vaccinations in children. Some of these studies are described in the PDR [17].

1. In the US, 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, there were 285 reports involving death, 971 nonfatal serious reports (defined as events involving initial hospitalization, prolongation of hospitalization, life-threatening illness, or permanent disability), and 4514 less serious reports after immunization with any pertussis-containing vaccine [22].

2. Systemic adverse events occurring within 3 days following vaccination of 4,696 Italian infants with DTP at 2, 4, and 6 months of age were recorded. These included fever of more than 100.4ºF (38.0ºC) in 7% of total; irritability in 36.3%; drowsiness in 34.9%; loss of appetite in 16.5%; vomiting in 5.8%; and crying for one hour or more in 3.9% [17:3063].

3. The whole-cell DTP vaccine has been associated with acute encephalopathy [17]. A large case-control study that included children 2 to 35 months of age who suffered from serious neurological problem was conducted in England. Acute neurological disorders, such as encephalopathy or complicated convulsion(s) occurred in children who were more likely to have received DTP vaccine the seven days preceding onset than their age-matched controls. Among children presumed to be neurologically normal before entering the study, the relative risk (estimated by odds ratio) of a neurological illness occurring within seven-day period following receipt of DTP dose, compared to children not receiving DTP vaccine in the seven-day period before onset of their illness, was 3.3 ($p < 0.001$).

4. Three hundred sixty-five infants were inoculated with Hib, and some of them developed systemic adverse reactions. The following adverse reactions and their percentages occurred in two-month-old infants during the 48 hours following inoculation: Fever > 100.8ºF or 38.2ºC (0.6%); irritability (12.6%); drowsiness (4.9%); diarrhea (5.2%); and vomiting (2.7%) [17:2318].

The above selected studies clearly show that serious health problems and even death can result from vaccinating infants and children, especially among the premature infants. The authors of these studies emphasized that premature infants should be monitored following the administration of vaccines. The PDR stated that physicians should inform the parents or guardians about the potential for adverse reaction of pertussis-containing vaccines (17:3062). The parent or guardian should be given the Vaccine Information Materials required by the National Childhood Vaccine Injury Act of 1986 prior to immunization.

It is unfortunate that baby Alan was given six vaccines (Table 5) and sent home without any consideration of being born five weeks premature and suffering from multiple health problems. His mother stated that the baby developed a high-pitched cry, his skin became warm to touch, and there was an increasing lethargy with a falling-off feeding pattern at about l0 or 11 days following the vaccines (3-4 days prior to his cardiac arrest on November 24, 1997). She was told that these symptoms might result following these vaccinations. On November 24th, the father was alone at home with the baby and his 4-year old sister. The father observed that, in rapid succession the baby began wheezing, next spit up, and then stopped breathing. While attempting to restore breathing and going (daughter in tow) to a neighbor's house to borrow a car, the father rushed the baby to Princeton Hospital in Orlando, Florida where the baby was eventually resuscitated.

The baby stayed five days in Florida Hospital. Review of the hospital charts from Princeton and Florida hospitals revealed that at the time of admission on November 24, 1997, baby Alan suffered from diabetes and complications of diabetes, such as metabolic acidosis, gastric ulcer, hypokalemia, apnea, cardiac arrest, hypotension, respiratory acidosis, and infections. Unfortunately, his doctor overlooked the fact that his symptoms resulted from diabetes, and the baby was treated with excessive amounts of sodium bicarbonate and heparin which caused severe hypoxia, cerebral edema, and hemorrhage in brain, lungs, and spinal cord. Detailed description of the hospital events and my analysis of these events are presented in Section 4. The medical evidence indicates that Alan's diabetes had resulted from infections induced by the vaccines received on November 11, 1997.

4. Review of Alan Ream Yurko's medical records during his hospitalization on November 24 Through 29, 1997, and analysis of his health problems

4.1. Clinical events and treatments at Princeton Hospital

Review of the medical records from Princeton Hospital revealed that Alan Ream Yurko was brought into the emergency department of Princeton Hospital by his father, Alan Yurko, at

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about 11:30 AM on November 24, 1997. Mr. Yurko drove a borrowed automobile from his house to the hospital [23]. The child arrived at the hospital either in total cardiac arrest or nearly so. The child was blue and not breathing. The emergency department physicians resuscitated baby Alan and started an interosseous line in the left tibia.

The first electrocardiogram was recorded at 11:36 AM and showed only a very slow and ineffective heart rate. At 11:49 AM, the heart rate became adequate. The child was intubated at this time. The first blood gas was done at 12:09 PM, showing a pH of 7.179, a PCO_2 of 74 mm Hg, and a PO_2 of 585 mm Hg. Other laboratory work drawn at 12:09 PM showed a glucose level of 337 mg/dL, a creatinine level of 0.5 mg/dL, LDH level of 2411 IU/L, SGOT level of 207 IU/L, SGPT level of 121 IU/L, a CO_2 level of 13 mEq/L, and anion gap level of 22 mEq/L. The white blood cell count was 20,900 per μL (band 5%, segs 26%, monocytes 8%, lymphocytes 61%), hematocrit value of 23%, hemoglobin concentration of 7.8 g/dL, and the platelet count was 571,000 per μL. Other laboratory work drawn at that time showed a BUN level of 6 mg/dL, total protein level of 5.6 g/dL, albumin level of 3.3 g/dL, sodium level of 139 mEq/L, potassium level of 4.9 mEq/L, and chloride of 104 mEq/L.

Furthermore, his admitting temperature was 93ºF (33.9ºC). The Treating Physician examined the baby who was flaccid with fixed and dilated pupils. He showed no signs of spontaneous movement except for slow, agonal respirations. The corneas were somewhat cloudy. The tympanic membranes were clear and there was no hemotympanum. There was a small reddish linear bruise under the right eye, but no other injuries to the head. The mouth was free of injury externally. The trachea was midline. Examination of the thorax, both anterior and posterior, did not reveal any bruise or other injury. The breath sounds were equal, and chest movement was adequate in the intubated patient. The abdomen was soft, somewhat distended and there were no bowel sounds. Examination of the extremities showed them to be flaccid but grossly normal to inspection. There were no bruises or injuries to the extremities. He developed bleeding from the gastrostomy tube, and the Treating Physician stated that this was probably from a stress ulcer. His admission weight was 10.05 pounds (4.57 kg).

In the unit, the Treating Physician started a central line in the right femoral vein and gave the child 15 mEq of sodium bicarbonate in an effort to alter his acid-base status. He also gave him three types of antibiotics—rocephin, gentamicin, and Claforan (cefotaxime sodium)—at high therapeutic doses to fight infections. In addition, he treated the baby with fluid for dehydration, and with other medications to stimulate his heart and respiration. The list of medications given to the baby and doses is presented in Table 6. The baby's temperature was 98ºF (36.7ºC) at 1:10 PM, and it reached 103ºF (39.4ºC) at 1:45 PM. His blood pressure was unobtainable at 1:10 PM, and rose to 129/88 at 1:45 PM because of the treatment received. At this time his blood pH was 7.3 and his blood PCO_2 and PO_2 were 31 and 114 mm Hg, respectively.

Table 6. Medications given to Alan at Princeton Hospital on November 24, 1997

Time	Medications	Dose	Route	Class/type
1:00 PM	**Rocephin**	**300 mg**	**IV**	**Antibiotic**
1:00 PM	1/2NS	20 cc/hr	IV	Fluid for dehydration
1:10 PM	**Gentomycin**	**10 mg**	**IV**	**Antibiotic**
1:30 PM	N-Saline	80 cc (20 cc/kg)	IV	Fluid to treat dehydration
1:40 PM	N-Saline	80 cc (20 cc/kg)	IV	Fluid to treat dehydration
1:55 PM	N-Saline	80 cc (20 cc/kg)	IV	Fluid to treat dehydration
2:02 PM	N-Saline	80 cc (20 cc/kg)	IV	Fluid to treat dehydration
2:00 PM	**Claforan**	**22 mg/8 hours**	**IV**	**Antibiotic**
2:00 PM	Red blood cells	15 cc/kg	IV	To treat anemia
2:05 PM	Dopamine	5 mg/kg/min.	IV	Stimulate respiratory system
2:09 PM	Dopamine	10 mg/kg/min.	IV	Stimulate respiratory system

4.2. Events and treatments at Florida Hospital

Baby Alan was transferred to Florida Hospital, Orlando and arrived at about 2:15 PM on November 24, 1997 [24]. His blood pressure and temperature were 40/20 and 98ºF (36.7ºC), respectively. He was placed on life support. Blood analysis at 2:40 PM revealed that his blood pH was 7.10, PCO_2 of 60.5 mm Hg, PO_2 of 151 mm Hg, and bicarbonate of 17.9 mEq/dL. The baby was treated with a long list of medications to treat dehydration, hypokalemia, metabolic and respiratory acidosis, infections, and fever. Also, he was given red blood cells and heparin. The list of medications given between 2:15-11:05 PM on November 24th is presented in Table 7. The blood pH rose from 7.10 at 2:40 PM to 7.67 (metabolic alkalosis) at 11:00 PM as a result of the bicarbonate treatment (Table 8). His temperature was 98ºF (36.7ºC) at 2:20 PM and became 105.8ºF (41.0ºC) at 6:00 PM. It dropped to 103ºF (39.4ºC) at 8:00 PM due to the treatment with high therapeutic doses of antibiotics, Motrin®, and Tylenol® (Tables 7 and 8).

Furthermore, it seems that baby Alan was given heparin at a dose level of 2 cc per hour (500 IU/mL) by intravenous infusion at 2:45 PM. With the baby's weight at 4.57 kg, the resulting effective heparin dose was 219 IU/kg per hour (or 2 cc/hr x 500 IU/mL/4.57 kg). The PDR recommends the following pediatric dosage schedule: initial dose of 50 units/kg IV drip, and maintenance dose of 100 units/kg (IV drip) every four hours, or 25 units/kg per hour [17:3306]. Heparin inhibits reactions that lead to the clotting of blood and the formation of fibrin clots both in vitro and in vivo. Heparin acts on multiple sites in the normal coagulation. Clotting time is prolonged by full therapeutic doses of heparin in most cases. Heparin also induces the formation of white clot due to the aggregation of platelets. At 3:15 PM, at about 30 minutes post-heparin infusion, blood analysis showed increased prothrombin time and fibrinogen split product level (Table 9).

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The PDR states that bleeding can occur at virtually any site in patients receiving heparin. Fall in hematocrit, fall in blood pressure, or any other unexplained symptoms should lead to serious consideration of a potential hemorrhagic event. Heparin sodium should be used with extreme caution in disease states in which there is increased danger of hemorrhage. Baby Alan had hypotension (Table 10) and his hematocrit was very low (25.3%). The normal range for hematocrit is 36.5-52%.

Table 7. Medications given to baby Alan in Florida Hospital on November 24, 1997

Time	Medications	Dose	Route	Class/type
2:15 PM	Dopamine	10 μg/kg/min.	IV	Stimulate respiratory system
2:20 PM	½ NS +KCl	20 meq KCl/20 cc	IV	Potassium + Fluid
2:22 PM	Gentomycin	10 mg	IV	Antibiotic
2:22 PM	Claforan	22 mg/8 hours	IV	Antibiotic
2:24 PM	Dopamine	5 μg /kg/min.	IV	Stimulate respiratory system
2:45 PM	**Heparin 1:1**	**2 cc/hr**	**IV**	**Anticoagulant**
2:45 PM	Bicarbonate	5 meq	IV	Treat acidosis
2:45 PM	Norcuron	0.4 mg	IV	Neuromuscular blocking agent
2:50 PM	Bicarbonate	5 meq/hr	IV	Treat acidosis
3:15 PM	Adenosine	0.05 mg/Kg	IV	Stimulate respiratory system
3:15 PM	Plasmanate	10 cc/kg (40 mL)	IV	Protein
3:15 PM	Red Blood Cells	15 cc/kg (60 mL)	IV	Treat anemia
4:00 PM	Plasmanate	10 cc/kg (40 mL)	IV	Protein
4:00 PM	N. Saline + KCl	20 cc/hr + 20 mg KCl	IV	Treat hypokalemia and dehydration
4:45 PM	Plasmanate	10 cc/kg (40 mL)	IV	Protein
6:10 PM	Tylenol	85 mg	NG	Treat fever
7:00 PM	Motrin	45 mg	NG	Treat fever
10:00 PM	Norcuron	0.4 mg	IV	Neuromuscular blocking agent
10:30 PM	Gentomycin	10 mg/hr	IV	Antibiotic
10:30 PM	Claforan	200 mg/8 hours	IV	Antibiotic
11:00 PM	Tylenol	45 mg	NG	Treat fever
11:00 PM	Motrin	45 mg	NG	Treat fever
11:00 PM	Plasmanate	45 cc	IV	Protein
11:05 PM	Versed	0.5 mg	IV	Sedative
11:05 PM	Versed	0.5 mg/hr	IV	Sedative

Table 8. Baby Alan's blood gases Measurements*

Date	Time	pH	PCO_2 mm Hg	HCO_3 mEq	PO_2 mm Hg
11/24/97	12:23 PM	7.18	74		585
	2:40 PM	7.10	60.5	17.9	151
	3:40 PM	7.19	54.9	20.2	108
	5:39 PM	7.25	64.0	26.4	142
	8:30 PM	7.39	51.9	30.9	54.8
	10:05 PM	7.36	59.4	33.0	137
	11:00 PM	7.67	22.1	25.9	218
11/25/97	12:15 AM	7.70	19.6	24.9	254
	1:30 AM	7.55	32.8	28.8	247
	3:40 AM	7.61	29.6	30.2	
	10:50 AM	7.61	29.2	29.3	190
	12:30 PM	7.55	33.8	30	119
	5:15 PM	7.45	46.1	32.1	109
Normal range		7.35-7.45	35-45	22-26	80-100

Table 9. Coagulation parameters for Alan Ream Yurko November 24-26, 1997

Coagulation parameter	Date (mm/dd/yyyy) and Time 11/24/1997 3:15 PM	11/25/1997 5:54 AM	11/26/1997 10:35 AM	Normal Range
Fibrinogen	263.00	-	320	190-400 mg/dL
Fibrinogen split product	160.00 H*	-	10 H	<10 ug/mL
Prothrombin time	14.60 H	14.5 H	11.6	8.7-12.7 seconds
APPT	24.00	34.1 H	14.9 L*	23.1-32.7 seconds

*H: High value; L: Low value

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Table 10. Baby Alan's blood pressure, heart rate, and temperature

Date	Time	Blood pressure	Temp. ºF	Heart Rate
11/24/97	12:05 PM	0	93.0	0
	1:10-1:40 PM	Unable to obtain	98-99	-
	1:45 PM	129/88	103.0	-
	2:00 PM	58/31	99.1	-
	2:07 PM	48/34	100.4	-
	2:14 PM	107/48	100.1	214
	2:15 PM	40/20	-	-
	2:20 PM	107/88	98.0	214
	2:35 PM	119/83	-	226
	2:45 PM	98/80	>102.0	-
	3:15 PM	-	-	260
	3:16 PM	-	-	180-230
	4:45 PM	-	103.8	-
	5:45 PM	86/65	105.0	226-230
	6:00 PM	89/67	105.8	226-240
	6:30 PM	90/68	105.1	221-240
	7:00 PM	114/63	105.7	212-240
	8:10 PM	102/54	103.5	199-240
	8:30 PM	106/58	103.0	191-238
	9:00 PM	104/55		192-240
	11:40 PM	-	-	170
11/25/97	2:50 AM	72/31 to 85/39	-	-
	3:30 AM	97/46	101.7	
	4:00 AM		100.7	
	10:30 AM	130/80	-	180
	11:00 AM	-	101.0	-
	12:00 Noon	-	102.6	-
11/26/97	3:50 AM	-	101.3	-
	5:00 AM	-	100.7	-
	6:00 AM	-	99.8	-

A computerized tomography scan of the brain taken at 7:50 PM (at about five hours following the start of heparin infusion) showed a right subdural hematoma, intraparenchymal hemorrhage in the right frontal region and some mass affect in the right cerebral hemisphere. An ophthalmologist examined Alan's eyes and found minimal right internal hemorrhages. At 4:21 PM, chest x-ray showed bilateral pulmonary infiltrates and healing fractures of the 6th rib. Dr. Scott Mahan stated that review of the bony structure revealed an old, healing fracture of left posterior rib #6 in the midclavicular line. The remaining bony structures revealed no significant abnormalities.

On November 25th, baby Alan was treated with a sedative, potassium, fluid, heparin, sodium bicarbonate, neuromuscular blocker, and antihistamine. The list of medications is presented in Table 11. This second sodium bicarbonate infusion was started at 8:00 AM as treatment for acidosis. However, the blood pH was 7.67 (highly alkaline) at 11:00 PM of the previous night and it was 7.61 at 3:40 AM on November 25th. At this time the baby was suffering from metabolic alkalosis, thus the treatment with bicarbonate was not justified. Metabolic alkalosis causes hypoxia by increasing the binding of oxygen with hemoglobin and preventing the release of oxygen to the tissues [7, 25, 26, 27].

At 8:00 AM, the baby was also given heparin by infusion similar to the dose given on November 24th. This treatment was not justified at all, because heparin at high therapeutic dosage should not be given to any patient suffering from bleeding and hypotension [17]. Baby Alan had a bleeding gastric ulcer, subdural hemorrhage, bleeding in the brain, and hypotension. The platelet count prior to the administration of heparin on November 24th was 571,000/μL of blood, dropping to 397,000/μL (30.5% reduction) at 5:45 AM on November 25, 1997 (at about 15 hours following the start of the first heparin infusion). Heparin increases the tendency of platelets to aggregate and form a clot. Blood analysis values of November 24th through November 27th are presented in Table 12.

In the first twenty-four hours after admission, the baby received 525.8 mL of fluid by IV; 10 mL by nasogastric tube (NG); 60 mL red blood cells; and 130 mL plasmanate. His total intake was 725.8 mL. However, his 24-hour output was 786 mL (756 mL urine and 30 mL from NG). The net output was 60.2 mL.

The baby was given a sedative, red blood cells, and plasmanate from November 26th through 28th. He was also given antidiuretic hormone on November 28th (Table 11). The results of the blood analysis are presented in Table 12 and Table 13. On November 26th, his serum glucose dropped to a normal level of 95 mg/dL from 397 mg/dL (76% reduction) on November 24, 1997. Also, on November 26th the LDH, alkaline phosphate, and SGPT levels dropped by 70%, 47%, and 19% respectively from their levels on November 24. On November 26, 1997, the total white blood cell count was reduced by 35% from the level on November 24th. This clearly indicates that the baby had liver and pancreas bacterial infections and his infections were resolved because of the treatment with antibiotics (Tables 6 and 7).

The baby was declared brain dead on November 27, 1997, at about 75 hours following the hospital admission. Autopsy was performed on November 29, 1997. Prior to autopsy, his heart, liver, pancreas, and a portion of his intestine were taken by Translife for transplantation. He was also given a megadose of heparin (22,950 IU) prior to and during organ harvesting to prevent the formation of blood clots in organs.

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Table 11. Medications given to baby Alan in Florida Hospital on November 25-29, 1997

Date/Time	Medications	Dose	Route	Class/type
11/25/1997				
2:55 AM	Plasmanate	12.5 cc/kg (50 mL)	IV	Protein
6:15 AM	Norcuron	0.3 mg/hr	IV	Neuromuscular blocking agent
6:15 AM	Versed	0.5 mg/hr	IV	Sedative
6:15 AM	Cimetidine	45 mg	NG	Treat gastric and duodenal Ulcer
7:45 AM	Versed	0.5 mg/hr	IV	Sedative
8:00 AM	Norcuron	0.3 mg/hr	IV	Neuromuscular blocking agent
8:00 AM	**Bicarbonate**	**5 cc/hr (1 mEq/cc)**	**IV**	**Treat Acidosis**
8:00 AM	**Heparin 1:1**	**2 cc/hr**	**IV**	**Anticoagulant**
8:45 AM	½ NS +KCl	20 mEq KCl/20 cc (500 mL)	IV	Potassium + fluid
10:40 AM	1/2 NS+KCl	40 mEq KCl/20 cc	IV	Potassium + fluid
4:00 PM	Versed	0.5 mg	IV	Sedative
5:30 PM	1/2 NS+KCl	55 mEq KCl/L	IV	Potassium + fluid
5:30 PM	Lasix	0.8 mg	IV	Diuretic
6:00 PM	Versed	0.5 mg	IV	Sedative
6:00 PM	Versed	0.5 mg/hr	IV	Sedative
6:45 PM	Lasix	5 mg	IV	Diuretic
9:20 PM	Cimitidine	45 mg	IV	Treat gastric and duodenal Ulcer
11:00 PM	1/2NS+KCl	40 mEq KCl/20 cc	IV	Potassium + fluid
11:00 PM	Plasmanate	90 cc	IV	Protein
11/26/1997				
6:00 AM	Versed	0.5mg/hr		Sedative
11/28/1997				
5:00 PM	80 cc RBC			Treat anemia
8:15 AM	**DDAVP**	**0.5 µg**	**IV**	**Antidiuretic hormone**
8:25 PM	Plasmanate	50 cc	IV	Protein
11/29/1997	Plasmanate	50 cc	IV	Protein

Table 12. Blood and Serum Analysis results for Alan Ream Yurko on November 24-27, 1997

	Date (mm/dd) and Time									
Measurement	11/24 1209	11/24 1515	11/25 0545	11/25 1645	11/25 2200	11/26 0500	11/27 0553	11/27 1515	Normal Range	Units
Glucose	337	397	141	131	121	95	92	89	70-110	mg/dL
Urea Nitrogen	6	9	11	15	17	13	14	6	5-25	mg/dL
Creatinine	0.5	0.4	0.2	0.2	0.5	0.3	0.2	0.2	0.6-1.2	mg/dL
Bilirubin Total	0.6	0.3	1	-	-	0.6	0.7	0.6	0.1-1.5	mg/dL
Direct Bilirubin		0.1	0.3	-	-	0.2	0.2	0.2	0-0.3	mg/dL
SGOT	207	98	167	-	-	167	117	65	14-50	IU/L
SGPT	121	156	331	-	-	154	106	55	25-41	IU/L
Alkaline phos	255	295	162	-	-	135	145	147	39-126	IU/L
LDH	2411	718	1096	-	-	733	773	672	110-210	IU/L
Protein	5.6	4.3	5.1	-	-	4.9	4.7	4.8	6.5-8.0	g/dL
Albumin	3.3	2.9	3.9	-	-	3.6	3.3	3.2	3.2-5.5	g/dL
Globulin	NM	1.4	1.2	-	-	1.3	1.4	1.6	1.9-3.9	g/dL
Cholesterol	85	61	34	-	-	54	80	82	100-200	mg/dL
Triglyceride	128	60	59	-	-	108	151	116	10-190	mg/dL
Uric Acid	4.7	5.5	3.3	-	-	3.7	1.7	2	2.0-8.0	mg/dL
Calcium	9.4	7.6	7.5	-	-	8.8	8.8	8.3	8.5-10.5	mg/dL
Phosphorous	9.8	7	4.9	-	-	4.1	4.3	4.4	5.0-9.5	mg/dL
Sodium	139	140	153	149	150	148	129	130	135-145	mEq/L
Potassium	4.9	5.2	2.3	2.7	3.6	4.3	5.1	4.2	3.5-5.0	mEq/L
Chloride	104	110	106	109	109	110	100	106	98-110	mEq/L
Magnesium	-	2.3	108	-	-	2	1.8	1.9	1.5-2.5	mg/dL
CO2	13	19	31	28	33	31	20	14	24-32	mEq/L
Anion Gap	22	16	18	-	-	11	14	14	5-15	mEq/L
WBC	20.9	8	11.2	14.1	-	13.6	11.6	-	3.0-12.0	$x10^3/\mu L$
RBC	2.61	2.14	3.01	2.88	-	2.67	4.11	-	4.16-5.7	$x10^6/\mu L$
Hemoglobin	7.8	6.3	9	8.6	-	7.9	12.2	-	12.1-17.3	g/dL
Hematocrit	25.3	19.3	26.5	25.5	-	23.8	36.5	-	36.5-52	%
MCV	96.9	90.1	88.1	88.5	-	89.2	88.7	-	82-99	FL
MCH	29.9	29.7	29.9	29.8	-	29.6	29.6	-	28-40	PG
MCHC	30.8	33	33.9	33.7	-	33.2	33.3	-	29-37	g/dL
Platelet	571	553	397	398	-	411	335	-	150-400	$x10^3/\mu L$
Neuto-Segm.	26	41.4	18	20	-	22	52.5	-	30-40	%
Lymphocyte	61	49.5	28	34	-	39	33.6	-	45-60	%
Neutro-Band	5	-	43	31	-	34	-	-	0-12	%
Monocyte	8	8.1	11	13	-	5	9.4	-	0-5	%
Eosinophil	0	0.6	0	0	-	0	4	-	0-5	%
Basophil	0	0.4	0	0	-	0	0.5	-	-	-
Aniso	1+		1+		-	-	-	-	-	-

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Table 13. Blood and Serum Analysis results for Alan Ream Yurko on 11/28/1997

	Time			
Measurement	**4:55 PM**	**7:45 PM**	**Range**	**Unit**
Glucose	-	92	70-110	mg/dL
Urea Nitrogen	-	5	5-25	mg/dL
Creatinine	-	0.3	0.6-1.2	mg/dL
Sodium	-	137	135-145	mEq/L
Potassium	-	3.7	3.5-5.0	mEq/L
Chloride	-	114	98-110	mEq/L
CO2	-	20	24-32	mEq/L
Anion Gap	-		5-15	mEq/L
WBC	11.8	-	3.0-12.0	$x10^3/\mu L$
RBC	3.54	-	4.16-5.7	$x10^6/\mu L$
Hemoglobin	10.5	-	12.1-17.3	g/dL
Hematocrit	31.6	-	36.5-52	%
MCV	89.3	-	82-99	FL
MCH	29.7	-	28-40	PG
MCHC	33.3	-	29-37	g/dL
Platelet	317	-	150-400	$x10^3/\mu L$
Neuto-Segm.	55.5	-	30-40	%
Lymphocyte	28.4	-	45-60	%
Monocyte	10.5	-	0-5	%
Eosinophil	5.2	-	0-5	%

4.3. Analysis of hospital events and clinical data

Francine stated that her baby Alan developed a high-pitched cry and his skin became warm to touch at about 10 or 11 days following receipt of his six vaccines listed in Table 5. Also, she observed the baby in a state of increasing lethargy with a declining feeding pattern [4, 16]. On the morning of November 24th, the father was alone at home with the baby and his 4-year old sister. The father observed that in rapid succession, the baby began wheezing, then spit up, then stopped breathing. While attempting mouth-to-mouth breathing and going (daughter in tow) to a neighbor's house to borrow a car, the father rushed the baby to the Princeton Hospital of Orlando where the baby was eventually resuscitated. Alan also stated that he did not shake his baby.

My review of the medical records of baby Alan obtained from Princeton and Florida hospitals and described above (Section 4) confirmed Francine and Alan Yurko's stories. Baby Alan's blood analysis results of November 24, 1997 (Table 8, Table 12) revealed that he was suffering from diabetes mellitus and complications of diabetes such as cardiac arrest, apnea, hypokalemia, metabolic and respiratory acidosis, and infections.

Baby Alan's serum glucose levels at 12:09 and 3:15 PM were 337 and 397 mg/dL, respectively. Normal serum glucose rage is 70-110 mg/dL. His blood pH was 7.18 at 12:09 PM and dropped to 7.1 at 2:40 PM (Table 8). His serum potassium level was 4.9 mEq/L at 12:09 PM and dropped to 2.3 mEq/L at 5:45 AM on November 25, 1997 following treatment with excessive amount of sodium bicarbonate (blood pH was 7.6-7.7). His hypokalemia was severe. He was treated with potassium solutions by IV infusion several times on November 24th-25th (Table 7, Table 11). Also, he had elevated white blood cell count (20, 900/μL), elevated LDH (1148% of normal), alkaline phosphatase (202% of normal), and SGOT (414% of normal). His anion gap was 22 mEq/L.

Furthermore, at the time of admission to Princeton Hospital, the baby had a gastric ulcer and his corneas were cloudy. Chest x-rays taken on November 24th showed lung infiltrate which is a sign of lung infection. The elevated white blood cell count (20, 900/μL) and temperature (105.8ºF or 41.0ºC at 6:00 PM) are other signs of bacterial infection.

In metabolic acidosis resulting from diabetes, potassium usually leaves the intracellular environment because the intracellular proteins bind with hydrogen, which leads to cardiac arrest and paralysis of the respiratory muscles. At this stage, serum potassium levels are usually normal or elevated, but after treatment with bicarbonate and elevation of pH to normal or above normal, the potassium leaves the blood and goes back inside the cells. This leads to hypokalemia, as we observed in this case. At time of admission, baby Alan had no muscle tone, no intestinal movement, and his abdomen was distended. Harrison's Principles of Internal Medicine states that in metabolic acidosis, initial serum potassium concentrations are normal to high, despite depletion of body stores, and potassium concentrations fall rapidly during therapy with sodium bicarbonate, predisposing the patient to cardiac arrhythmias and/or paralysis of the respiratory muscles [7:2060].

Baby Alan had all the symptoms and complications of diabetes as described in the medical literature such as metabolic acidosis, cardiac arrest (hyperkalemia), cardiac arrhythmias (due to hypokalemia), infections, fever, cerebral edema, and gastric ulcer [7, 8, 11]. In diabetic children, cerebral edema is a common cause of death and more frequent than in adults. Baby Alan had cerebral edema as stated in the autopsy report [28]. The Chief Medical Examiner reported: "the brain appears very edematous, shiny and fluffy. Differentiation of the cortex and medulla appears poor and the ventricles are slightly reduced in size. Cerebral edema is confirmed."

The Treating Physician also confirmed on November 24th at Princeton Hospital that the baby had a gastric ulcer. The Treating Physician stated that the child developed bleeding from the gastrostomy tube due to stress ulcer. The child was treated with cimetidine (histamine H2-receptor antagonist) in the hospital for his ulcer (Table 11). The presence of gastric ulcer can explain the inability of the baby to take his food at home in the days prior to his cardiac arrest on November 24th. In Florida Hospital, the baby was given 10 mL of liquid by a nasogastric tube (NG) on November 24th through November 25th, and 30 mL came back through the NG tube, as described above (Section 4.1).

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Furthermore, baby Alan had metabolic acidosis as indicated by low blood pH (7.1), high blood PCO_2 level (74 mm Hg), low blood bicarbonate level (17.9 mEq/L), and high anion gap (22 mEq/L). In diabetic patients, the metabolic acidosis and anion gap are almost totally accounted for by the elevated plasma levels of acetoacetate and beta-hydroxybutyrate, although other acids (e.g., lactate, free fatty acids, phosphates) contribute [7]. Baby Alan was treated with sodium bicarbonate to correct his blood acidosis. However, he was given an excessive amount of bicarbonate. His blood pH was 7.1 at 2:40 PM on November 24, 1997 and increased to 7.67 at 11:00 PM (Table 8). Additionally, he was again given bicarbonate by IV infusion at 8:00 AM on November 25th (Table 11) and his blood pH was 7.61 at 3:40 AM of the same day (Table 8).

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 is 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 (7:2073). Alkalinization increases the avidity of hemoglobin to bind oxygen, impairing the release of oxygen in peripheral tissues. The hemoglobin-oxygen dissociation curve is normal in diabetic ketoacidosis because of opposing effects of acidosis and deficiency of 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 to minimize possible detrimental side effects and to prevent metabolic alkalosis as circulating ketones are metabolized to bicarbonate with reversal of ketoacidosis. The key parameters to follow are the pH and the calculated anion gap.

It is very obvious that these vital treatment recommendations were not followed in baby Alan's case and that his treatment with an excessive amount of bicarbonate led to severe hypoxia and cerebral edema [25-27]. Furthermore, baby Alan suffered from hypoxia as a result of his severe anemia as shown by very low hemoglobin (7.8 g/dL), hematocrit (25.3%), and low RBC (2.61 x 10^6/μL). His apnea, cardiac arrest, and hypotension also resulted in hypoxia and general ischemia of the brain.

Dehydration, polyurea, weight loss, and wasting are symptoms and complications of diabetes mellitus. In the first twenty-four hours, baby Alan received 525.8 mL of fluid by IV; 10 mL by nasogastric tube (NG); 60 mL red blood cells; and 130 mL plasmanate. His total intake was 725.8 mL. However, his twenty-four hour output was 786 mL (756 mL urine and 30 mL from NG). The net output was 60.2 mL. He was dehydrated in spite of receiving adequate amount of fluid by IV infusion (Tables 6, 7 and 11).

Moreover, the baby was treated with antidiuretic hormone (DDAVP) on November 28th to prevent dehydration (Table 13). DDAVP is a synthetic analog of the natural pituitary hormone 8-arginine vasopressin (ADH), an antidiuretic hormone affecting renal conservation. On November 24th, the baby's weight was 10.05 pounds (4.57 kg); on November 29th, his weight was 9.0 lbs (4.08 kg). He lost 1.05 lbs (0.476 kg), 10% of his weight, in five days during his stay in the hospital despite treatment with relatively high volume of fluid IV and antidiuretic hormone. Also, his average serum creatinine value on November 24th was 0.45 mg/dL (75% of low normal value) and dropped to 0.2 mg/dL (33% of low normal) on November 27th (Table 12). Low creatinine is an indicator of low muscle mass and wasting disease.

The clinical data indicate that Alan's diabetes resulted from bacterial infection of the pancreas and may have been due to infections of other organs. 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 [7]. Stress hyperglycemia, usually associated with infections and other life-threatening illnesses, is due to release of glucagons and catecholamines [7:2061]. Bacterial and mycotic infections complicate the life of the diabetic in whom hyperglycemia is poorly controlled. Multiple abnormalities in the host response to microbial invasion have been described in such patients. Leukocyte functions are compromised and immune response is blunted [7].

Blood analysis performed on November 24th prior to Alan receiving treatment with antibiotics shows that his white blood cells were elevated (20,900/μL). Also, he had elevated serum glucose level of 337 mg/dL, LDH level of 2411 IU/L (1148% of normal), alkaline phosphatase level of 255 IU/L (202% of normal), and SGOT level of 207 IU/L (414% of normal). He also had elevated anion gap of 22 mEq/L (Table 12). The treatment with high therapeutic doses of three types of antibiotics on November 24th resulted in significant reduction in serum glucose, liver enzymes, and anion gap levels (Tables 6, 7, 12). On November 26th, the serum glucose level was 95 mg/dL (normal) with low values for the following: LDH, 733 IU/L (reduced by 70%); alkaline phosphatase, 135 IU/L (reduced by 47%); SGOT, 167 IU/L (reduced by 19%); and anion gap 11 mEq/L (50% reduction) as shown in Table 12.

On November 24th, baby Alan was treated with three types of antibiotic IVs to fight bacterial infections (Table 6 and 7). These included: 20 mg gentamicin (recommended dose 7.5 mg/kg/day); 300 mg rocephin (recommended dose 50-75 mg/kg/day); and 222 mg Claforan (recommended dose 50-180 mg/kg/day). Gentamicin sulfate is a water-soluble antibiotic of the aminoglycoside group. Intravenous administration of gentamicin is used to treat patients with bacterial septicemia or those in shock. Gentamicin is indicated in the treatment of serious infections caused by susceptible strains of Pseudomonas aeruginosa, Proteus species, Escherichia coli, Klebsiella-Enrtrobacter-Serratia species, Citrobacter species, and Staphylococcus species [17:2845].

Rocephin is a semisynthetic, broad-spectrum antibiotic. The bactericidal activity of rocephin results from inhibition of cell wall synthesis. It has a high degree of stability in the presence of beta-lactamases (both penicillinases and cephalosporinases) of gram-negative and gram-positive bacteria [17:2694]. Rocephin is usually used to treat the following systemic infections: (1) bacterial septicemia caused by Staphylococcus aureus, Streptococcus pneumonia, Escherichia coli, Haemohilus influenza or Klebsiella pneumonia, (2) meningitis caused by Haemophilus influenzae, Neisseria meningitides or Streptococcus pneumonia, (3) lower respiratory infections caused by Streptoccocus pneumonia, Staphyloccus aureus, Haemophilus

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influenza, Staphylococcus parainfluenza, Klebsiella pneumonia, Escherichia coli, Enterobacter aerogenes, Proteus mirabilis or Serratia marcescens.

Claforan (cefotaxime sodium) is a semisynthetic, broad spectrum cephalosporin antibiotic. The antibacterial activity of cefotaxime results from inhibition of cell-wall synthesis and it has in-vitro activity against a wide range of gram-positive and gram-negative organisms [17:1318]. Cefotaxime is indicated for the treatment of serious infections caused by susceptible strains of the designated microorganisms in these diseases: (1) lower respiratory infections, including pneumonia caused by Streptococcus pneumonia, Streptococcus pyogenes, Staphylococcus aureus, Escherichia coli, Klebsiella species, Haemophilus influenzae, Haemophilus parainfluenzae, Proteus mirabilis, Serratia marcescens, Enterobacter species, indole positive Proteus and Pseudomonas species; (2) central nervous system infections, e.g., meningitis and ventriculitis caused by Streptococcus pneumonia, Klebsiella pneumonia, and Escherichia coli; (3) bacteremia/septicemia caused by Escherichia coli, Klebsiella species, Serratia marcescens, Staphylococcus aureus, and Streptococcus species (including S. pneumonia).

Furthermore, at 2:45 PM, baby Alan was given heparin at a dose level of 2 cc per hour of 50% heparin sodium by intravenous infusion. Heparin is a heterogeneous group of straight-chain anionic mucopolysaccharides, called glycosaminoglycans, having anticoagulant properties [17]. 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. The biomarkers of heparin toxicity observed in baby Alan's case indicated that the baby received a high doses of heparin. I assumed the stock solution of heparin used in this case contained 1000 IU per mL as described in the PDR [17]. Each one mL of heparin sodium injection USP contains 1,000 units heparin sodium and 10 mg benzyl alcohol as a preservative. With the baby's weight at 4.57 kg, the resulting effective heparin dose was 219 IU/kg per hour. The PDR recommends 50 units/kg IV as initial dose for infants and children, and a maintenance dose of 100 unit/kg (IV, drip) every four hours, or 25 unit/kg per hour [17:3306].

A computerized tomography scan of the brain taken at 7:50 PM (at about five hours following the start of heparin infusion) showed a right subdural hematoma and intraparenchymal hemorrhage in the right frontal region of the cerebral hemisphere. Based on the dose of heparin infused to the baby (219 IU/kg per hour), the estimated total dose of heparin infused in five hours was 1095 IU/kg, which is about 8.8 times the recommended maintenance dose for infants of 125 IU/kg per five hours [17].

Hemorrhage can occur at virtually any site in patients receiving heparin. Patients suffering from anemia, any unexplained symptoms, and/or having low blood pressure are at the greatest risk of having serious hemorrhagic events after receiving a therapeutic dose of heparin. Alan had hypotension and his hematocrit was very low (25.3%). The normal range for hematocrit is 36.5-52%. In addition, the baby was treated with adenosine, a potent vasodilator in most vascular beds that causes significant hypotension (Tables 7 and 11).

Heparin sodium should be used with extreme caution in disease states where there is increased danger of hemorrhage. In addition to serious bleeding, heparin has been found to induce the formation of white clot due to the aggregation of platelets and to reduce the platelet count due to consumption. At 3:15 PM, at about 30 minutes post-heparin infusion, blood analysis showed increases in fibrinogen split product (160 μg/mL) and prothrombin time (14.6 seconds), which are 1600% and 115% of normal respectively. Platelet count prior to the administration of heparin on November 24th was 571,000/μL of blood, and dropped to 397,000/μL (30.5% reduction) at 5:45 AM on November 25, 1997 (at about 15 hours following the start of the first heparin infusion). Blood analysis values of November 24th through November 27th are presented in Table 12.

Alan was given heparin again at 8:00 AM on November 25th by IV infusion. This treatment was not at all justified because heparin at high therapeutic dosage should not be given to any patient suffering from bleeding, hypotension, and anemia [17]. Baby Alan had bleeding gastric ulcer, subdural hemorrhage, bleeding in the brain, and hypotension. One day later, on November 26th, the fibrinogen split product value and prothrombin time returned to normal. This indicates that the elevation of these values were associated with the heparin treatment.

Furthermore, the baby was suffering from metabolic alkalosis as a result of his treatment with excessive amounts of sodium bicarbonate and this condition causes hypoxia as described above. His blood pH and bicarbonate levels at 3:40 AM on November 25th were 7.61 and 30.4 mEq/L, respectively (Table 8). He was also given bicarbonate by IV infusion at 8:00 AM on November 25th at the time of his treatment with heparin (Table 11).

An ophthalmologist examined the baby's eyes and observed minimal bleeding in the retina of the right eye. Many risk factors existed in Alan's case in addition to the treatment with heparin that usually lead to retinal bleeding. These factors include the following: (1) diabetes—retinal hemorrhage, including hemorrhage in the inner retinal areas and superficial nerve fiber layer, and preretinal hemorrhage is commonly described in patients suffering from diabetes [7], (2) hypoxia as a result of severe anemia, apnea, hypotension, metabolic and respiratory acidosis, and metabolic alkalosis from the excessive use of bicarbonate, (3) probable corneal infection as indicated at the time of hospital admission on November 24th (his corneas were cloudy).

The Treating Physician examined Alan on November 24th and found no signs of injuries except a small reddish linear bruise under the right eye. The Treating Physician stated that the tympanic membranes were clear—no hemotympanum—and the mouth was free of injury externally. Moreover, examination of the thorax, both anterior and posterior, and examination of the extremities did not reveal any bruise or other injury. In addition, Dr. Scott Mahan reviewed the chest x-ray taken on November 24th and found no significant abnormalities in the bony structure except for an old healing fracture of left posterior rib #6 in the midclavicular line.

In conclusion, baby Alan's many health complications resulted from being diabetic and from the treatment received in the hospital on November 24-27, 1997. These complications

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included the following: hyperglycemia; metabolic acidosis and respiratory acidosis; dehydration; weight loss; cardiac arrest; apnea; metabolic alkalosis; hypokalemia; cardiac dysrhythmias; subdural hemorrhage and bleeding in the brain (a result of excessive treatment with heparin, hypotension, and severe hypoxia); systemic infections; retinal hemorrhage and corneal edema; liver damage (elevated liver enzymes, heart damage (LDH was very high); and anemia.

The Chief Medical Examiner and other physicians who testified in court that baby Alan died as a result of "Shaken Baby Syndrome" overlooked the clinical data described in this report and based their conclusions on a theory only. My review of the autopsy report and the testimonies of the state expert witnesses revealed that these witnesses did not take the time to review all relevant data. My review and analysis of the autopsy report and testimonies of witnesses are presented in the next two sections of this report (Sections 5 and 6).

5. Analysis of the medical examiner's autopsy report and his court testimony in the case of baby Alan

On November 27, 1997, approximately 75 hours after initial hospital admission, baby Alan was pronounced brain dead. Prior to autopsy on November 29th, Translife (a company specializing in donor organ removal and transport) took the baby's heart, liver, pancreas, and a portion of the intestine for organ transplant. Prior to and during the harvesting of these organs, baby Alan was given 22,950 IU of intravenous heparin to prevent the formation of blood clots in the organs. This amount of heparin is capable of keeping 1000 mL of blood liquid at room temperature. The estimated whole-blood volume in baby Alan's case is about 320-366 mL, which is 7-8% of his body weight of 4.57 kg.

The Chief Medical Examiner performed the autopsy on Alan Ream Yurko (case number: MEH-1064-97) at 10:15 AM on November 29, 1997 in Orlando [28]. The main objective of this autopsy was to establish the cause(s) of death. He stated that Baby Alan died because of bleeding in the brain resulting from shaking of the baby by his father, Alan Yurko. My review of the Chief Medical Examiner's autopsy report indicates that it lacks the accuracy and the expected minimum scientific detail to make it reliable and useful to answer the questions about the cause(s) of death. In addition, he did not provide the medical evidence in his report and his court testimony in February of 1999 to support his conclusion that baby Alan died of "Shaken Baby Syndrome." The following is a list of medical evidence that supports my assessment.

5.1. General appearance

> ***The Chief Medical Examiner stated,*** *"Alan's body is that of a 2 months old white male infant and appears to be of that age. The build and nourishment are average. The height of the body is 22 inches. The weight of the body is 9 lbs. The circumference of the head is 22 cm."*

The medical evidence related to this case contradicts the Chief Medical Examiner's statements that the baby had average build and nourishment at the time of autopsy, and that his head circumference was 22 cm. Baby Alan was born on September 16, 1997, and his age on November 29th was about 2 and a half months. His head circumference measurements on October 1 and November 11, 1997 were 33.3 and 37.5 cm, respectively (Table 4). It is very obvious that the Chief Medical Examiner's measurement of 22 cm for the circumference of Alan's head was wrong.

Furthermore, the baby had diabetes at the time of admission to Princeton Hospital on November 24th as described above in Section 4. Dehydration, polyurea, weight loss, and wasting are symptoms and complications of diabetes mellitus. His weight on November 24th was 10.05 pounds (4.57 kg), and during his stay in the hospital, despite treatment with a relatively high volume of fluid IV, he lost 1.05 lb (0.476 kg), or 10% of his weight, in five days. Alan received 725.8 mL of fluid and red blood cells during the first twenty-four hours in the hospital and his output during this period was 786 mL of fluid. Net output was 60.2 mL. Again, he was suffering from dehydration in spite of being treated with adequate amount of fluid by IV infusion in the hospital (Tables 6, 7, and 13).

Also, he was treated with antidiuretic hormone (DDAVP) on November 28th to prevent dehydration (Table 13). Additionally, his average serum creatinine value on November 24 was 0.45 mg/dL (75% of low normal value) and dropped to 0.2 mg/dL (33% of low normal) on November 27th (Table 12). Low creatinine is an indicator of low muscle mass and wasting disease. Moreover, Dr. Douglas Radford Shanklin found that the fatty tissue of baby Alan was mostly pink and granular, which is an abnormal metabolic state that is consistent with poor development [13].

5.2. Microscopic examination of the heart and liver function tests

> ***The Chief Medical Examiner stated on page 9 of his autopsy report,*** *"The myocardium shows no evidence of inflammation, interstitial or replacement type fibrosis. There is no necrosis of myocytes and no evidence of ischemic change. The microvasculature shows no areas of thickening or perivascular fibrosis. There are no atypical changes present. No inflammation is noted."*

The Chief Medical Examiner's description of the histology of Alan's heart as stated above, contradicts his statement in court in February of 1999 as well as his findings presented on page 5 of his autopsy report. He stated in court that Translife removed the heart with other organs prior to performing the autopsy [13]. He described in his report, "when the chest and the abdominal cavities are opened it is noted that the heart, the liver with gallbladder, spleen, pancreas, mesenteric lymph nodes and parts of the small intestine are surgically absent as a result of organ harvesting by Translife [28]." This indicates that he did not have the chance to examine the heart grossly and to take samples for pathological evaluation. He also confirmed this issue in his court testimony in February of 1999. He stated that the liver, spleen, pancreas, heart and partial small intestine were donated [13].

Moreover, blood analysis of November 24, 1997 showed that the baby had a very high LDH level of 2411 IU/L (1148%

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of normal) and this indicates damage in the cardiac muscle. The other indicators of cardiac problems are dysrhythmia, hypokalemia, and diabetes, which are observed in this case. Also, the serum levels of liver enzymes were elevated (Table 12). These findings contradict the statements made by the examiner in his court testimony that the heart and the liver were normal and did not contribute to the cause of illness and death.

5.3. Subdural hemorrhage, brain

> ***The Chief Medical Examiner stated,*** *"Subdural hemorrhage was seen prominently on the right cerebral hemisphere, and that this hemorrhage was in liquid as well as in clotted form. There was also subdural hemorrhage on the left cerebral hemisphere posteriorly, and this hemorrhage was relatively less prominent as compared to the right. The dura mater of the cortex of the cerebral hemispheres showed thickened and slightly clotted blood adherent to the dura mater. At places, the thickness of this clotted material was between 2-3 mm."*

The description of the nature of the clot and bleeding stated above indicate that the blood was released from the blood vessels in a continuous fashion during the five days prior to autopsy or more precisely, in three stages. The thickened clotted blood that adhered to the dura mater represents the first stage of blood release, the clotted blood represents the second stage, and the blood in the liquid form represents the third stage which is the most recent. Dr. Gary Steven Pearl, the state witness, examined the blood clot and observed the proliferation of fibroblasts in layers. Based on this observation he estimated the age of the oldest portion of the subdural hematoma to be two to five days [13]. I also examined the H & E stained tissue section of the meninges and observed the proliferation of fibroblasts in the blood clot in the subdural space and in the clot attached to the dura matter. I also observed fresh blood in the subdural space.

Furthermore, the CT brain scan taken at 7:50 PM on November 24th showed the subdural hematoma present only on the right side of the brain and no bleeding was seen on the left. This means that the bleeding on the left occurred after 7:50 pm on November 24th. These facts contradict the Chief Medical Examiner's conclusion that the hemorrhage occurred in minutes or even in a few seconds due to vigorous shaking of the head.

Also, the medical evidence indicates that the subdural hemorrhage resulted from damage to the blood vessel wall due to the excessive use of heparin and from severe hypoxia due to severe anemia, hypotension, apnea, and metabolic alkalosis induced by excessive treatment with sodium bicarbonate (Tables 6, 7, 11). The facts in the following list support my assessment:

1. The baby was treated with high doses of heparin on November 24th through 27th and prior and during harvesting the baby's organ for donation. Treatment with such doses of heparin usually leads to bleeding in healthy individuals and individuals already suffering from anemia, hypotension, and hypoxia.
2. The platelet count was reduced by 30.5% following the treatment with heparin due to clot formation induced by heparin.
3. The fibrinogen split product and prothrombin time were found to be elevated on November 24th, but returning to normal on November 26th. These changes coincide with the use of heparin on November 24th.
4. The blood vessels of the meninges were swollen as reported by Drs. Pearl and Shanklin in their court testimony. This indicates that these blood vessels were damaged as a result of hypoxia, metabolic changes, and/or inflammation [13].
5. The brain was edematous. Fluid was released from the blood vessels because of damage to their wall from hypoxia, inflammation, hypotension, and the excessive use of sodium bicarbonate.
6. Hemorrhage was also observed in the lungs and the subdura of the spinal cord. This shows that the damage in the blood vessels was not limited to the brain, and resulted from metabolic changes and inflammation that affected many sites.
7. The subdural hemorrhage observed in the left side of the brain on November 29th that was absent on November 24th at 7:50 PM, also indicates that the subdural bleeding and the bleeding in the brain occurred in the hospital.

5.4. Subdural hemorrhage, spinal cord

> ***The Chief Medical Examiner stated,*** *"When the spinal cord is traced downward through the lumbar and sacral regions it is noted that there is a small quantity of hemorrhage in the subdural space of the spinal cord representing the areas of the lower thoracic, lumber and sacral regions."*

The Chief Medical Examiner stated that the bleeding was present only in the subdural space of the lower thoracic, lumber, and the sacral regions of the spinal cord. There was no bleeding found in the cervical and upper thoracic portions of the spinal cord. The Chief Medical Examiner's observation indicates that the bleeding in these regions of the spinal cord occurred independently of the bleeding that occurred in the brain. The blood did not come down from the brain through the spinal canal. This fact is also supported by the observations of two pathologists: Dr. Shanklin observed a fresh hemorrhage (6-12 hours old) in the subdural space of the spinal cord and Dr. Pearl indicated that there was a spinal cord injury, that blood vessels were swollen and nerve cells damaged. I also examined the H & E stained tissue section of the spinal cord and found a fresh hemorrhage in the subdural space. The Chief Medical Examiner and other physicians examined the entire vertebral column of the baby and they did not find any injury caused by trauma. These observations indicate that the bleeding occurring in the subdural space resulted from damage in the blood vessels due to hypoxia and from the treatment with excessive doses of heparin.

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5.5. Bleeding in the brain

> *The Chief Medical Examiner stated, "Serial-cut sections of the brain did not show any internal hemorrhage in the brain parenchyma grossly. The two sections which were stained with H & E stain showed appearance of very minute parenchymal hemorrhages. The cerebellum showed normal appearance, and one section showed evidence of shearing type injury with multiple foci of minute hemorrhages. He also stated that the brain appeared very edematous, shiny and fluffy. Differentiation of the cortex and medulla appeared poor. The ventricles were slightly reduced in size and the cerebrospinal fluid appeared clear."*

The information described above indicates that the hemorrhage in the brain was very minor and only microscopic. It also shows that the brain was edematous. The edema fluid leaks into the extracellular space either through damaged capillary endothelial cells that have lost their barrier function or through newly formed capillaries that have not established barriers. Fluid in the brain increases the intracranial pressure (ICP) and this pressure causes brain damage. ICP causes either focal or diffuse flattening of the cortical gyri, sometimes associated with compressed or distorted ventricles. Relatively rigid dural folds form the flax cerebri and the tentorium cerebelli partitions in the cranial vault. Localized expansion of the brain causes it to be displaced in relation to these portions, producing brain herniations [11]. On section, the white matter may appear soft and gelatinous and the peripheral layer of gray matter is widened. The ventricles are usually compressed. Microscopically, there is considerable widening of the interfibrillar spaces of the brain which gives a loose appearance to the white and gray matter. Swelling of the neural and glial cells may also be present [11:87-88].

In baby Alan's case, the brain edema was severe and diffused. The ICP pressure reduced ventricle size and made the differentiation between the cortex and medulla appear poor. The ICP in this case is certainly capable of causing damage to the tiny blood vessels in the brain that were damaged by hypoxia. It seems that the Chief Medical Examiner overlooked these established medical facts and incorrectly stated that the minor bleeding observed in the brain was caused by shaking the baby.

5.6. Meningitis

Meningitis is an inflammation of the meninges and the subarachnoid space. Infectious meningitis can be broadly classified as acute pyogenic meningitis (usually bacterial), acute lymphocytic meningitis (generally viral), and chronic meningitis, which may be bacterial or fungal [11:1378]. The changes in tissues that are usually observed in cases of acute meningitis include: congestion of the blood vessels, swollen blood vessels, edema, presence of inflammatory cells in tissues (neutrophils and/or lymphocytes), hemorrhage, and degeneration and necrosis of brain cells. These changes were observed in baby Alan's brain as described by the state and the defense expert witnesses [13] and they confirm that Alan had meningitis. I also examined H & E stained tissue section of the meninges and observed the infiltration of the middle arachnoid membrane with lymphocytes and macrophages.

Dr. Douglas Shanklin examined the meninges, finding the blood vessels distended and the meninges tremendously thickened, perhaps to eight or ten times normal. Also present were hundreds of inflammatory cells (acute and chronic inflammatory cells). He also observed damaged nerve cells in the brain and inflammatory cells in the walls of the blood vessels. Dr. Gary Pearl also observed swollen blood vessels and chronic inflammatory cells in the meninges. In addition, he found damaged neurons in the cerebellar dentate nucleus. Also, cerebral edema was confirmed by the Chief Medical Examiner.

The changes in tissues described above, with the presence of fever (105.8ºF or 41.0ºC) and an elevated white blood cell count (20,900/μL) that were observed on November 24th (Table 12), indicate that the baby suffered from acute meningitis. However, the severity of the acute inflammation in tissue was reduced by the treatment with high therapeutic doses of antibiotics. On November 24th, baby Alan was treated with three types of antibiotics to fight bacterial infections. These included 20 mg gentamicin, 300 mg rocephin, and 222 mg Claforan (Tables 6 and 7). Treatment with the antibiotics reduced blood white blood cell count from 20,900 to 13,600/μL, and body temperature from 105.8ºF (41ºC) to 99.8ºF (37.7ºC). The antibiotics also caused a significant reduction in number of neutrophils in the inflamed tissues.

It seems that the Chief Medical Examiner overlooked the medical facts described above when he stated that baby Alan did not suffer from meningitis. He stated, "I examined the meninges in this case and found no evidence of meningitis." However, there is no description of a microscopic examination of the meninges in his report. Also, he did not examine the cerebrospinal fluid (CSF) at the time of autopsy to check for the presence of inflammatory cells. He stated in court that he did not examine the CSF because it was mixed with blood [13]. However, he stated in his autopsy report that the CSF fluid was clear. He reported that serial cut sections of the brain did not show any internal hemorrhage in the brain parenchyma grossly. "The ventricles are slightly reduced in size and the cerebrospinal fluid appears clear [28]."

5.7. Diffuse axonal injury

The Chief Medical Examiner stated that baby Alan died as a result of vigorous shaking which caused diffuse axonal injury. He also claimed that diffuse axonal injury is a characteristic lesion of Shaken Baby Syndrome. I have two problems with the Chief Medical Examiner's conclusions: (1) the Chief Medical Examiner did not present any evidence in his autopsy report or in court that he found an axonal injury in this case; and (2) axonal injuries indistinguishable from those observed in cases of head trauma can occur as a result of edema, hypoxia, hypoglycemia, cardiac arrest, and other causes. In this case, the child was suffering from brain edema, hypoxia, and cardiac arrest, and no head injury due to trauma was found. However, the Chief Medical Examiner overlooked these medical facts and based his conclusions on a theory only. Below are data that support my assessment.

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(1) The Chief Medical Examiner did not provide any evidence to show that he found diffuse axonal injury in Alan's brain. Below are Mr. Barrett's (defense attorney) questions related to the axonal injury, and the Chief Medical Examiner's answers. These are taken from the Chief Medical Examiner's court testimony in February of 1999 related to this case [13].

> ***Barrett:*** *Are there any of the slides we could look at to show us this diffuse axonal injury?*
> ***Chief Medical Examiner:*** *Well, diffuse axonal injury is nothing but minute petechial hemorrhage and these we can not show you.*
>
> ***Barrett:*** *These are injuries to the axons which are parts of the nerves, correct?*
> ***Chief Medical Examiner:*** *Right.*
>
> ***Barrett:*** *Are there any slides you have that you examined that you could show us that will show us this diffuse axonal injuries you said you saw in this case?*
> ***Chief Medical Examiner:*** *No.*
>
> ***Barrett:*** *And again, nowhere in your report do you mention anything, about diffuse axonal injury, correct?*
> ***Chief Medical Examiner:*** *Correct.*

(2) Below is a description of the findings of six studies that show axonal injury present in the brain in cases of edema, hypoxia, cardiac arrest, which are observed in Alan's case. As noted, axonal injury due to brain trauma cannot be differentiated from axonal injury resulting from other causes. Therefore, all the causes that lead to an axonal injury should be considered prior to stating that this axonal injury was caused by a shaking force, especially in cases with no evidence of trauma.

Study 1: Extensive neurohistological examination was undertaken in 13 patients in whom coma was attributed to hypoglycemia. The study revealed varying degrees of widely distributed neuronal necrosis. In five of these cases there was also evidence that the intracranial pressure had been high with internal herniation. It was concluded that a significant amount of axonal injury found in these 13 cases can be attributed to hypoglycemia per se, although the amount and distribution of the axonal damage is altered in the presence of increased intracranial pressure (ICP). However, in some cases axonal damage is seen in the absence of an elevated intracranial pressure and in one case its distribution closely mimicked that seen in microscopical diffuse traumatic axonal injury. This further demonstrates that not all axonal pathology is caused by trauma [29].

Study 2: The brains of 17 individuals who died of cardiorespiratory arrest, and 12 of status epilepticus were evaluated microscopically to check for axonal damage. Axonal damage was seen in 9 of 17 and 7 of 12 of the cases, respectively. In most of these cases, there was also evidence of elevated ICP. It is concluded that the great majority of axonal damage identified in cases dying after cardiac arrest and status epilepticus can be attributed to increased ICP and the vascular complications of internal herniation. However, in some cases axonal damage was seen in the absence of an elevated ICP, although its amount and distribution were different from diffuse axonal injury [30].

Study 3: Sections from 28 brains showing evidence of cerebral hypoxia with no history of head injury, four with a history of head trauma but no evidence of hypoxic change, and eight with a history of head trauma and hypoxic change were evaluated by immunohistochemistry staining. Axonal damage was found in seven of 8 cases of head injury and hypoxic changes and 12 of 28 cases of hypoxia without history of head injury. The role of hypoxia, increased ICP, edema, shift effects, and ventilatory support in the formation of axonal bulbs should be considered. The presence of axonal bulbs cannot necessarily be attributed to shearing forces alone [31].

Study 4: Brain tissue sections from 67 individuals who died due to trauma-induced focal cortical hemorrhage without dural involvement and 51 cases of non-traumatic death due to cerebral hypoxia/ischemia were evaluated by immunohistochemical staining to determine the reliability of axonal injury (AI) as a marker of traumatic insult. Investigations of the pons in theses cases revealed that cases of non-traumatic death due to cerebral hypoxia/ischemia (n = 51) demonstrated AI with the same frequency as in the trauma group, although the expression tended to be less pronounced. The investigations were based primarily upon immunohistochemical demonstration of antibodies targeted to beta-amyloid precursor protein (beta-APP) in the pons as a marker of AI. The results of this study confirm that beta-APP expression in the pons is a reliable indicator of AI, but does not discriminate between (a) injuries caused by traumatic strain or shearing mechanisms and (b) secondary damage due to cerebral hypoxia/ischemia or edema. Therefore, positive differentiation of the type of biomechanical event based on this criterion alone is not possible [32].

Study 5: Beta-amyloid precursor protein (beta-APP) was used to detect axonal injury (AI) in the brain of individuals who died of nonmissile closed-head injury (n = 119), gunshot injury (n = 30), cerebral ischemia/hypoxia (n = 51), brain death caused by mechanical trauma (n =14), and nonmechanical injury (n = 18). AI was observed in 65% to 100% of cases of closed-head injury, fatal cerebral ischemia/hypoxia, and brain death, with a survival time of more than 3 hours. A statistically significant difference between traumatically and nontraumatically induced (nondisruptive) AI was not found [33].

Study 6: Brain tissues of 14 children who lacked skull fracture and allegedly died of Shaken Baby Syndrome (SBS) and 7 children who died of non-traumatic hypoxic ischemic encephalopathy (HIE) were evaluated using immunohistochemical stains. Swollen axons were present in 11 of 14 cases of SBS and in 6 of 7 cases of HIE [34].

5.8. Retinal hemorrhage

The Treating Physician examined baby Alan at the time of the baby's admission to Princeton Hospital on November 24th and found that Alan's corneas were cloudy. Dr. Douglas Shanklin evaluated the H & E stained tissue section of the right

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eye microscopically and observed the presence of numerous inflammatory cells that are consistent with a long-standing chronic inflammation. Dr. Shanklin, Pearl and the Chief Medical Examiner examined the H & E stained tissue sections of the retina of the eyes microscopically and observed very minor bleeding in the retina of the right eye only. Dr. Pearl stated that the bleeding in the retina was not significant. It did not impact his evaluation of this case [13].

My evaluation of baby Alan's case revealed the presence of many risk factors in addition to the heparin that usually lead to bleeding in the retina. These factors include the following: (1) diabetes, as explained in this report (Section 4), and retinal hemorrhage (inner retina, superficial nerve fiber layer, and preretinal hemorrhage) is commonly described in patients suffering from diabetes [7]; (2) hypoxia as a result of severe anemia; apnea; hypotension; metabolic and respiratory acidosis; and metabolic alkalosis (from the excessive treatment with sodium bicarbonate in the hospital). Hypoxia causes damage in the small blood vessels wall that leads to bleeding. (3) Probable infection in the right eye as indicated by the corneal edema (his corneas were cloudy) and the presence of inflammatory cells in the retina of the right eye as described above.

The Chief Medical Examiner presented the minor bleeding in the retina of the right eye as evidence in court to support his claim that baby Alan died as a result of vigorous shaking. It is very hard to believe that the Chief Medical Examiner overlooked the medical evidence described above that provides explanation for the factual causes of the minor bleeding observed in the retina of right eye. His duty as a medical examiner is to evaluate the medical evidence that explains the causes of injuries in this case. I believe that he did not follow the standard medical protocol in this case as required by his job and the law.

5.9 Pneumonia and lung hemorrhage

Dr. James Edward Hannah, radiologist, evaluated baby Alan's chest x-ray taken on November 24, 1997 and observed that the lungs were infiltrated. This means that the lungs were filled with fluids, inflammatory cells, and debris. These are signs of pneumonia. In addition, the presence of fever (105.8ºF or 41.0ºC) and the elevated white blood cell counts (20,900/μL) observed on November 24th (Table 12) indicated that the baby suffered from acute bacterial infection.

On November 29th, the Chief Medical Examiner examined the lungs grossly and found that both lungs were congested and contained irregular areas of hemorrhagic appearance. Serial cutting sections of both lungs showed irregular areas of hemorrhages. He also examined the H & E stained tissue sections of the lungs microscopically and observed the presence of red blood cells and clumps of inflammatory cells in the alveolar sacs. The inflammatory cell infiltrates are scattered throughout one section. He stated that this picture appears to be somewhat similar to interstitial pneumonitis. Dr. Douglas Shanklin also examined the H & E stained tissue sections of the lungs and observed inflammatory cells (white blood cells) present in the alveoli and in the structure of the lung. He also found the bronchioles filled with inflammatory cells. He identified this condition as pneumonia and said that the infection was much older than 75 hours. Furthermore, I examined the H & E stained tissue section of the lung and observed thickening of the interstitial septa, bronchioles filled with inflammatory cells, and multifocal areas of fresh hemorrhage.

The histopathology and the radiology findings described above, and the elevated body temperature (105.8ºF or 41.0ºC) and white blood cell count (20,900/μL) indicate that the baby suffered from acute pneumonia. However, the severity of the acute inflammation in tissue was reduced by the treatment with high therapeutic doses of antibiotics. On November 24th, baby Alan was treated with three types of antibiotics IV to fight bacterial infections that included 20 mg gentamicin, 300 mg rocephin, and 222 mg Claforan (Tables 6, 7). The treatment with antibiotics reduced the blood white blood cell count from 20, 900 to 13, 600 /μL and the body temperature from 105.8ºF to 99.8ºF (or 41.0ºC to 37.7ºC). The treatment with antibiotics also caused a significant reduction in the number of neutrophils in the inflamed tissue. It misled the Chief Medical Examiner to believe that the lesions in the lungs on November 24th were minor and not significant.

The pathological changes observed in the lungs have two significant clinical points:. (1) Alan had acute pneumonia, which caused hypoxia and respiratory acidosis; and (2) the hemorrhage in the lungs indicates that the baby was suffering from a bleeding condition that affected many organs, and that this bleeding was not caused by trauma. Bleeding was also observed in the subdural area of the spinal cord, and the examination of the vertebral column revealed no injuries due to trauma, as described in Section 5.4 of this report.

These clinical findings support my conclusions that the bleeding in tissues in this case was caused by treatment with excessive doses of heparin and sodium bicarbonate, and by hypoxia.

5.10 Rib fracture

Dr. Robert Scott Mahan and Dr. James Edward Hannah, radiologists, read the chest x-rays of baby Alan taken on November 24 and 25, 1997 and found only one fractured rib (rib #6). They gave the time for this fracture between weeks and several months. The Chief Medical Examiner stated that he observed during his autopsy irregular swelling of the left ribs #5, 6, 7 and 10; that these sites appeared as globular masses of cartilaginous tissue, and that cut sections of these masses showed normal appearance of the cartilage. He further stated that these masses probably resulted from healing fractures of the ribs and that these fractures occurred between one and four weeks. He also stated that x-rays were taken and confirmed the presence and positions of these healing fractures and that multiple sections were taken for histopathological study [28]. However, the Chief Medical Examiner did not present in his report or in court any information describing his histology and radiology findings of these fractured ribs. He showed only photographs of these ribs in his court testimony.

The Chief Medical Examiner further stated that the cut sections of these masses showed normal appearance of cartilage. The calluses of healing bone usually contain calcium and not simply normal cartilage. This indicates that the masses of cartilage observed by the Chief Medical Examiner in ribs #5, 7, and 10 do not represent healing fractures of these ribs. This assess-

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ment is supported by the fact that two radiologists read the chest x-rays taken on November 24th/25th and saw only one rib fracture (rib #6). It is also supported by the Chief Medical Examiner's statement in court describing the process of healing in a rib fracture. He stated that the healing process in a rib usually starts with initial swelling of the rib and then gradual calcium deposition in the fracture sites [13]. I am puzzled by the fact that the Chief Medical Examiner did not show the x-ray film of these fractured ribs in court, but showed only photographs. He stated that he took the x-rays and samples for pathology to confirm the presence and positions of these healing fractures. It seems reasonable that he be asked to explain his reason for not presenting the evidence that supports his claim.

Review of the medical literature reveals cases in which rib fractures that occurred during labor were missed during initial examination of the baby. Below is brief description of these cases.

Case 1: A large weight (3912 g) for gestational age female neonate was delivered vaginally with the use of vacuum extraction. The neonate was breathing quietly, with no respiratory tract distress. A chest radiograph was obtained and showed minimally displaced fractures of ribs 4, through 8 posteriorly on the right side. The lungs, heart, and other skeletal structures were normal [35].

Case 2: A large weight (4205 g) for gestational age, term male neonate was delivered vaginally with the use of vacuum extraction to assist delivery. A chest radiograph showed nondisplaced fractures of ribs 6 through 8 posteriorly on the right side. The baby had normal lungs and mediastinum [35].

Case 3: A 37-year-old diabetic woman spontaneously went into labor at 38 weeks of gestation. She delivered a 3300g baby with the assistance of vacuum extraction. Physical examination did not detect any abnormality. At about 9 hours after delivery, the nurse noticed the child having rapid respirations. Examination by a resident physician revealed mild respiratory distress with tachypnea and tachycardia. Crepitus was palpable over the left posterolateral chest. No skin changes suggestive of trauma were found. Chest x-ray examination revealed five fractured ribs over the left posterolateral chest area. There is no evidence of pneumothorax or other skeletal trauma. Over the next 36 hours the child experienced progressively less tachypnea and gradual disappearance of the crepitus. Full skeletal survey failed to show evidence of osteogenesis imperfecta or any other abnormality of bone mineralization [36].

Case 4: Chest radiograph was taken of a 4905-gm female delivered by midforceps after right shoulder dystocia. On the 11th day, a prominent mass was noted in the right midclavicular region. Radiograph reveals a right midclavicular fracture with slight superior angulation and incidental fractures of left ribs #5 and 6.

Moreover, Fanaroff et al. explained the mechanism of rib injuries during labor as follows: rib injury is initiated when the anterior shoulder is impacted behind the symphysis pubis, with the other shoulder attempting to descend into the posterior compartment of the pelvis. This results in compression forces on the fetal arm and thorax, leading to spontaneous rib fractures on the same side as the posterior shoulder [10]. They also stated that the specific clinical manifestations of ribs injuries are often absent making diagnosis difficult [10].

The four cases with rib fracture described above were born at term or close to term and had better health than baby Alan. Alan was born five weeks premature and had respiratory distress and jaundice. His mother was suffering from gestational diabetes and had chronic oligohydramnios. Skeletal and facial deformities in a fetus are some of the risks associated with oligohydramnios pregnancy. Furthermore, pregnancy in diabetics is usually associated with a higher incidence of congenital anomalies (6-12% vs. 2-3% in non diabetics) [7]. These data point out the high possibility that Alan's rib #6 was fractured during labor.

The prosecutor brought the issue that the rib fracture did not occur during birth, because it was not seen in the x-ray films taken on September 16-18, 1997. Alan was born on September 16, 1997. A rib fracture occurring during labor would have been two days old on September 18th and it takes at least 7 days for the calluses to form and to show on the x-rays. Cumming reported that when a fracture is discovered in a newborn infant, it is important to decide whether it occurred at birth or after birth. Calcification around the fracture site gives a useful estimate of the age of the fracture. We reviewed films of 23 patients with fractures resulting from delivery. These fractures occurred at three different sites: the clavicle, the humerus, and the femur. Calcification could be seen as early as seven days after birth and was absent for as long as 11 days after birth [37].

5.11 Superficial bruise and contusions

The Treating Physician examined baby Alan at the time of admission to the Princeton Hospital on November 24, 1997 to check for injuries resulting from trauma. He found only a small, reddish, linear bruise under the right eye, as described in Section 4.1 of this report. Dr. Robert Gold, pediatric ophthalmologist, also examined the baby on November 24th and observed only a slight bruise under the right lower eyelid. No other injuries to suggest trauma were found by other physicians and nurses who examined baby Alan during his five days in the hospital. Furthermore, on November 29th, the Chief Medical Examiner examined the body during his autopsy to check for injuries caused by trauma and found no evidence of injuries caused by trauma except the small bruise under the right lower eyelid described above and two minor contusions on the right and the left temporal areas.

The Chief Medical Examiner stated in court that the bruise under the right eye was about five days old and the minor contusions about 24 hours old. This means that these contusions occurred no later than November 28th and that they happened in the hospital. The baby was admitted on November 24, 1997. Also, the baby's mother explained in court that the bruise under the right eyelid described above was caused by an octagon baby bottle. The baby's four year-old sister was giving the bottle to her father and accidentally hit the baby in the eye area [13].

In court, the Chief Medical Examiner showed three photographs of the bruise under the eyelid and the two minor contusions, and he spent time describing these minor superficial inju-

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ries. These actions on the Chief Medical Examiner's part are quite troubling because he knew very well that these minor injuries had nothing to do with the causes of baby Alan's illness and death. He stated that the two minor contusions occurred at about 24 hours prior to autopsy. This means that they occurred in the hospital and have no impact on this case. Furthermore, he did not ask the parents of the baby for an explanation of the minor bruise under the right eyelid. His presentation of the three photographs of these minor injuries in the court did not serve any medical objectives, but certainly confused the jury members by making them think that a physical force was used. He mentioned that these injuries were caused by blunt force. I believe that the Chief Medical Examiner's approach is not scientifically and professionally justified, and that he should be asked to explain his actions.

5.13 Conclusions

The Chief Medical Examiner conducted the medicolegal examination of the body of Alan Ream Yurko Pursuant to Florida Statutes Chapter 406 and 732.9185. The main purpose of his examination was to discover the cause(s) of injuries and death. This task can be accomplished only by evaluating all medical evidence related to the case, as presented in this report (Sections 2 through 5). I believe that the medical examiner did not meet his obligation as required by his job and the law as shown by the evidence described above in Section 5. Below is a summary of the medical evidence showing that the Chief Medical Examiner's actions and conclusions are not supported by medical facts, and that he did not meet his obligation as medical examiner in this case.

1. The Chief Medical Examiner did not review the case history of baby Alan's mother during her pregnancy with Alan. She suffered from several medical problems (gestational diabetes, oligohydramnios, anemia, infections). Her illness caused health problems in the fetus as described in Section 3 of this report, which placed the baby at greater risk to develop adverse reactions to vaccines. Alan developed an infection and diabetes after receiving six vaccines, which led to his cardiac arrest on November 24, 1997.

2. The Chief Medical Examiner did not review the case history of baby Alan from birth to the time of his cardiac arrest on November 24, 1997. Alan was born five weeks premature and suffered from Respiratory Distress Syndrome, jaundice, hypoglycemia, anemia, and growth retardation (Section 3). These illnesses caused him to be at greater risk for developing adverse reactions to vaccines. Alan developed an infection and diabetes which led to his cardiac arrest on November 24, 1997

3. The Chief Medical Examiner did not evaluate the impact of vaccines given to Alan on his health (Table 5). The studies presented in Section 3 of this report clearly show that these vaccines caused very serious adverse reactions in premature infants such as apnea, cardiac problem, and respiratory infections. Baby Alan developed diabetes which was the result of an infection and complications of vaccination. This led to his cardiac arrest on November 24, 1997.

4. The Chief Medical Examiner overlooked the evidence that showed baby Alan had diabetes on November 24, 1997, which led to his cardiac arrest and apnea due to hypokalemia. Also, the level of serum LDH was high and indicates Alan suffered from cardiomyopathy.

5. The Chief Medical Examiner overlooked the evidence that showed Alan had bacterial infections as indicated by elevated white blood cell count, body temperature, and his response to treatment with antibiotics.

6. The Chief Medical Examiner overlooked the evidence that indicated Alan was treated with high doses of three types of antibiotics, and his infections responded well to this treatment (Table 6, 7). Also, he did not consider the influence of this treatment on the pathological changes in tissues and on his autopsy findings on November 29, 1997.

7. The Chief Medical Examiner overlooked the evidence that showed Alan was treated with excessive doses of sodium bicarbonate and heparin that caused hypoxia and bleeding in the brain, spinal cord, and lungs. Also, the baby suffered from anemia and hypotension which are risk factors for bleeding in individuals treated with heparin.

8. The Chief Medical Examiner overlooked the evidence that baby Alan had bleeding in the brain, spinal cord, and lungs. This indicates that his problem was caused by heparin, metabolic changes, and cardiovascular disturbance.

9. The Chief Medical Examiner did not consider the clinical data that showed Alan suffered from anemia, clotting problems, and infections.

10. The Chief Medical Examiner stated that the liver, heart, and organs other than the brain did not contribute to the cause of illness and death. The clinical data presented in this report shows that the baby suffered from diabetes and had problems in liver and heart.

11. The Chief Medical Examiner stated that baby Alan had an average build and nourishment, but the clinical data presented in this report shows that the baby was losing weight and suffering from dehydration.

12. The Chief Medical Examiner's measurement for the head circumference of 22 cm on November 29th was obviously wrong. It was 37.5 cm at eighteen days prior to this date (Table 4).

13. The Chief Medical Examiner described the histology of the heart in his autopsy report; but the evidence showed that the heart was donated prior to autopsy and therefore he did not have the chance to examine it. This indicates that his work is suffering from very serious problems. The indication is that he mixed cases up with each other. The level of serum LDH, hypokalemia, and dysrhythmia indicate that the heart was not normal.

14. The Chief Medical Examiner overlooked the evidence that showed the subdural hemorrhage was found only on the right side of the brain on November 24, 1997. Finding bleeding on the left side on November 29th indicates that the bleeding did not happen in minutes or a few seconds, as The Chief Medical Examiner claimed in his court testimony in February of 1999.

15. The Chief Medical Examiner overlooked the facts that the brain was edematous and that edema increased the intracranial pressure and caused axonal injury and brain damage.

16. The Chief Medical Examiner claimed that he observed an axonal injury in the brain but he did not provide any evidence that supported his claim. There is no description of an

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axonal injury presented in his report or in court. He said that we could not show you the axonal injury in the brain! He needs to be asked to explain his reason.

17. The Chief Medical Examiner stated in court that the cerebrospinal fluid (CSF) was mixed with blood, but the information presented in his report shows that the CSF was clear. He reported that serial cut sections of the brain did not show any internal hemorrhage in the brain parenchyma grossly. The ventricles were slightly reduced in size and the cerebrospinal fluid appeared clear [28].

18. The Chief Medical Examiner stated that Alan had no meningitis, but the evidence presented in this report showed otherwise (Sections 4 and 5). Alan suffered from meningitis, as indicated by the presence of swelling blood vessels, congestion, edema, and infiltration of tissue with inflammatory cells.

19. The Chief Medical Examiner claimed that axonal injury is a characteristic lesion of Shaken Baby Syndrome, but the published literature described in Section 5 of this report shows that axonal injuries occurred in cases of brain edema, hypoxia, and cardiac arrest.

20. The Chief Medical Examiner presented the minor bleeding in the retinal of the right eye as evidence that baby Alan died as a result of "Shaken Baby Syndrome," and he did not investigate the factual causes that led to bleeding in the retina as presented in Section 5.

21. The Chief Medical Examiner did not provide x-ray findings to prove that Alan had fractures in ribs #5, 7, and 10. Also he did not search the medical literature to find out if rib fractures can occur during labor as presented in this report (Section 5.10).

22. The Chief Medical Examiner showed photographs in court of a minor contusion in the temporal areas of the head that occurred in the hospital at about one day prior to autopsy, and had no relation to the cause of death in this case. I believe that he did this to influence the jury's thinking that physical force was used in this case.

23. The Chief Medical Examiner showed a photograph in court of a minor bruise under the right eyelid which has no relation to the cause of death in this case. He did not make any attempt to speak with the baby's parents or to seek their explanation for the cause of this insignificant minor injury. I believe that he was acting desperately to find any injury caused by trauma in this case to influence the jury's thinking that physical force was used in this case.

6. Review of Alan R. Yurko's jury trial, analysis of the expert witnesses' testimonies, and the State's claim

Alan R. Yurko's jury trial took place February 22 to 24, 1999 in the state of Florida [13]. The prosecutor provided four major witnesses testifying for the state, and (following the defense witness) two of these were called for repeat appearances before the jury. Against these four, the defense provided a single witness. The state witnesses included (1) the Chief Medical Examiner; (2) Dr. Gary Pearl, a consultant neuropathologist (testified twice); (3) the Treating Physician; and (4) the General Pediatrician (testified twice). The defense witness was Dr. Douglas Radford Shanklin, a pathologist. In addition, two radiologists, Dr. James Edward Hannah and Dr. Robert Scott Mahan, testified in relation to the fractured rib #6; and Dr. Robert Gold, ophthalmologist, gave testimony in relation to the minor bleeding in the retina.

Three state witnesses (the Chief Medical Examiner, the Treating Physician, and the General Pediatrician) stated that baby Alan died as the result of Shaken Baby Syndrome. However, none of them provided medical evidence to prove their case, and their testimonies were based only on a theory. Dr. Pearl stated that the injuries in the brain and spinal cord were acute injuries and did not start at birth or after birth. He did not say that these injuries were caused by Shaken Baby Syndrome. His findings are discussed in Section 6.3, below.

In the previous Section 5 of this report is a review and analysis of the medical examiner autopsy report and his testimony in court relating to this case. They clearly show that the Chief Medical Examiner did not review all the medical evidence, and he did not prove his case. In addition, he described the histology of the heart, even though the heart had been donated for transplantation prior to his examination. Also, in court he stated that the cerebrospinal fluid was mixed with blood, but in his report he stated that the CSF was clear. In this section, I present my review and analysis of the testimonies of the other three state witnesses. This presentation shows that these witnesses also did not review all the evidence and did not provide the medical evidence to prove their case. I also present my review of the defense witness. He made important contributions to this case.

6.1. Review of the Treating Physician's testimony

The Treating Physician stated in court that baby Alan died as a result of "Shaken Baby Syndrome." His theory was that shaking the head caused bleeding, and that the blood pushed on the brain causing an increase in pressure. The pressure on the brain stem caused unconsciousness and cessation of breathing. The Treating Physician presented his conclusion about the cause of death without making any attempt to review Francine's medical record during her pregnancy with Alan, the baby's medical records from birth until his hospitalization on November 24, 1997, the adverse reactions to vaccines and medications given to the baby, and the autopsy findings. I explained these issues and their impact on Alan's health in Sections 2 through 5 of this report. It is very clear that the Treating Physician's conclusion about the cause of death was based on a theory and not on facts, because he did not evaluate the medical evidence related to this case.

Furthermore, the Treating Physician presented in court only selected items of his clinical findings related to baby Alan's five days in the Princeton and Florida hospitals. He did not reveal that, at the time of his admission to Princeton Hospital, Alan had diabetes and complications of diabetes, such as gastric ulcer, metabolic acidosis, elevated anion gap, dehydration, anemia, elevated serum liver enzymes and LDH (Table 12). Furthermore, the Treating Physician did not reveal to the court that he treated baby Alan with a long list of medications that included three types of antibiotics, Tylenol, Motrin, heparin, antidiuretic hormones, potassium, and others (Tables 6, 7, 11). The clinical findings during Alan's hospitalization on November 24th through 29th, along with an analysis of the clinical

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events are presented in Section 4 of this report. Below is a brief summary of these clinical findings to show that the Treating Physician was aware that baby Alan had diabetes, hypokalemia, metabolic acidosis, infections, and dehydration, because he reviewed the data and treated him for these conditions. Unfortunately, his treatment with excessive doses of sodium bicarbonate and heparin caused severe hypoxia and severe bleeding in the brain, lungs, and spinal cord.

The Treating Physician examined baby Alan at Princeton Hospital shortly after his admission. He saw the baby at about 12:30 PM on November 24, 1997 in the emergency room after the emergency department physicians resuscitated him. The baby arrived at Princeton with cardiac arrest and apnea. The Treating Physician's initial evaluation revealed that baby Alan was flaccid, his abdomen was soft and somewhat distended, and there was no bowel sound. The baby developed bleeding from the gastrostomy tube due to a gastric ulcer. His corneas were somewhat cloudy. The Treating Physician also reviewed the result of the first laboratory work done at Princeton. The first blood gas was done at 12:09 PM showing a pH of 7.179, a PCO_2 of 74 mm Hg, and a PO_2 of 585 mm Hg (Table 8). Other laboratory work drawn at 12:09 PM showed a glucose of 337 mg/dL, LDH of 2411 IU/L, SGOT of 207 IU/L, SGPT of 121 IU/L, a CO_2 of 13 mEq/L, anion gap of 22 mEq/L, and potassium of 4.9 mEq/L. The white blood cell count was 20,900 per μL, hematocrit 25.3%, hemoglobin concentration 7.8 g/dL, and the platelet count was 571,000 per μL (Table 12).

These tests clearly showed that the baby suffered from diabetes, metabolic and respiratory acidosis, bacterial infections, anemia, and liver and heart damage. In Princeton Hospital, the Treating Physician treated the baby with sodium bicarbonate to control acidosis; three types of antibiotics—rocephin, gentamicin, and Claforan (cefotaxime sodium)—to control bacterial infection; and saline to treat dehydration (Table 6). He also treated Alan in Florida Hospital on November 24 through November 28, 1997. He gave the baby antibiotics, Tylenol®, Motrin®, sodium bicarbonate, potassium chloride, heparin, dopamine, adenosine, muscle relaxant, antidiuretic hormone, and other medications (Tables 7 and 11). The baby responded very well to antibiotics. His temperature, white blood cell count, and blood glucose levels returned to normal following two days of treatment (Tables 8, 12, 13).

On November 26th, his serum glucose level dropped to a normal level of 95 mg/dL from the level of 397 mg/dL (76% reduction) on November 24, 1997. Also, on November 26th, the LDH, alkaline phosphate, and SGPT levels dropped by 70%, 47%, and 19% respectively from their levels on November 24. On 11/26, the total white blood cell count was reduced by 35% from its level on November 24th. This clearly indicates that the baby had liver and pancreas bacterial infections, and that his infections were resolved because of the treatment with antibiotics (Tables 6, 7).

Unfortunately, the Treating Physician treated Alan with heparin at 2:45 PM on November 24th, at an estimated dose level of 2 cc (500 IU/mL) per hour by intravenous infusion. The baby's weight was 4.57 kg and the estimated heparin dose was 219 IU/kg per hour. Based on this dose, the estimated total dose of heparin infused up to the time of the CT scan (five hours) was 1095 IU/kg, which is about 8.8 times the recommended maintenance dose for infants of 125 IU/kg per five hours, recommended by the PDR [17]. Hemorrhage can occur virtually at any site in patients receiving heparin. Patients suffering from anemia, any unexplained symptoms, and/or having low blood pressure are at the greatest risk of having serious hemorrhagic events following a therapeutic dose of heparin. Alan had hypotension, and his hematocrit was very low (25.3%). In addition, the baby was treated with adenosine, which is a potent vasodilator in most vascular beds, causing significant hypotension (Tables 7, 11). Heparin also induces the formation of white clot due to the aggregation of platelets. At 3:15 PM, at about 30 minutes post heparin infusion, blood analysis showed increased prothrombin time and fibrinogen split product (Table 9). The treatment with heparin explains the bleeding seen on the CT scan taken at 7:50 PM on November 24th and the fall in the platelet count seen on November 25th (Table 12).

Furthermore, the bleeding in the brain was made worse by Alan's second treatment with heparin at 8:00 AM on November 25th. This treatment was not justified at all, because heparin at a high therapeutic dose should not be given to any patient suffering from bleeding and hypotension [17]. Baby Alan had a bleeding gastric ulcer, subdural hemorrhage, bleeding in the brain, and hypotension. The platelet count prior to the administration of heparin on November 24th was 571,000/μL of blood and dropped to 397,000/μL (30.5% reduction) at 5:45 AM on November 25, 1997 (at about 15 hours following the start of the first heparin infusion). Heparin increases the tendency of the platelets to aggregate and form a clot. Blood analysis values for November 24th through November 27th are presented in Table 12. Alan also suffered from disseminated intravascular coagulation (DIC) as a result of his treatment with heparin. At 3:15 PM, at about 30 minutes post heparin infusion, blood analysis showed increases in fibrinogen split product (160 μg/mL) and prothrombin time (14.6 seconds). These values are 1600 % and 115% of normal respectively, and they returned to normal on November 26th following the cessation of the treatment with heparin.

To make matters even worse, the Treating Physician treated the baby with excessive amount of sodium bicarbonate by IV to treat acidosis. This treatment caused severe metabolic alkalosis, and the blood pH reached a very critical high level of 7.7. This treatment caused severe hypoxia by preventing the hemoglobin from releasing oxygen to the tissues; it caused reduced potassium level in the blood, and caused cerebral edema, as explained in Sections 4 of this report. Alan's blood pH was 7.1 and serum potassium level was 4.9 mEq/L on November 24th. The potassium dropped to critical low of 2.3 mEq/L at 5:45 AM on November 25th following the treatment with excessive amount of sodium bicarbonate. The blood pH was at critically high levels of 7.6-7.7.

Dehydration, polyurea, weight loss, and wasting are symptoms and complications of diabetes mellitus. In the first twenty-four hours, baby Alan's input was 725.8 mL, while his output during this period was 786 mL. Net output was 60.2 mL. On November 24th, Alan weighed 10.05 pounds (4.57 kg), and on November 29th, 9.0 lb (4.08 kg). He lost 1.05 lb (or 0.476 kg), 10% of his weight, in five days during his hospital stay despite treatment with relatively high volume of fluid IV. Furthermore,

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his average serum creatinine on November 24th was 0.45 mg/dL (75% of low normal value) and dropped to 0.2 mg/dL (33% of low normal) on November 27th (Table 12). Low creatinine is an indicator of low muscle mass and wasting disease.

In addition, the baby was treated with an antidiuretic hormone (DDAVP) on November 28th to prevent dehydration (Table 13). DDAVP is a synthetic analog of the natural pituitary hormone 8-arginine vasopressin (ADH), an antidiuretic hormone affecting renal conservation. This fact shows that he was responding to medication in spite of being declared brain dead on November 27, 1997. Also, he was able to maintain his body temperature at about normal levels following his treatment with antibiotics. The treatment with antibiotics reduced the blood white blood cell count from 20,900 to 13,600/μL and body temperature from 105.8ºF (41.0ºC) to 99.8ºF (37.7ºC). This indicates that his fever was caused by bacterial infections and not by brain injury as the Treating Physician claimed.

The Treating Physician and Florida Hospital contacted the Orange County Sheriff's Office and the Child Protective Office on November 24, 1997 and filed a report of child abuse based on the assumption that baby Alan was injured as a result of abuse by his father. Alan R. Yurko was arrested by officers from the Orange County Sheriff's Office on November 26, 1997 while his son Alan was still alive in Florida Hospital. I found that the actions of the Treating Physician and Florida Hospital are not supported by any medical facts. Their actions are tragic for Alan's family and dangerous for the entire society!

The Treating Physician assumed that Mr. Yurko was guilty of child abuse, yet his own examination of baby Alan revealed no injuries caused by trauma. Also, he did not wait for the concluding results of the autopsy. In addition, he did not review the adverse reactions of medications that he gave to the baby on November 24th to see if the heparin and other medications had something to do with bleeding in the brain. Then he gave more heparin and sodium bicarbonate the next morning which caused more bleeding. On November 24th the subdural bleeding was on the right side of the brain only, and on November 29th the bleeding was on the right and left sides of the brain. The Treating Physician gave excessive amount of heparin and sodium bicarbonate which can cause bleeding in any child who has hypotension, anemia, and tissue inflammation similar to baby Alan's case.

6.2 Review of the General Pediatrician's testimony

The General Pediatrician is a consultant with the child protection team based in the children's hospital in Orlando. He examined baby Alan and reviewed his chart on November 25th for about 30 to 45 minutes in Florida Hospital, and declared that the baby was injured as a result of child abuse. He gave his conclusion about the cause of injuries without review of Francine's medical records while pregnant with Alan, the baby's medical records from birth until his hospitalization on November 24, 1997, the adverse reactions to the vaccines and medications given to baby Alan, or the autopsy findings. These issues and their impact on this case are explained in Sections 2 through 5 of this report. Below are the questions presented to the General pediatrician in court by the State of Florida and defense attorneys and his answers to these questions [13]. They clearly show that he presented his conclusion based on a theory only and not based on the medical evidence.

1. Examination and diagnosis:

> ***The state:*** *Where did you first examine the child?*
> ***General Pediatrician:*** *The one and only one time I examined him was in pediatric intensive care unit at Florida South on November 1997.*
>
> ***The state:*** *And once you examined him and reviewed his medical chart, did you have a diagnosis?*
> ***General Pediatrician:*** *Yes*
>
> ***The state:*** *What was that?*
> ***General Pediatrician:*** *The child had received inflicted trauma that was seen as both brain injuries as well as injuries to several ribs that is characteristic of an abusive situation.*
>
> ***The state:*** *As part of your examination of this child, would you rule out any natural disease and causes in making your diagnosis?*
> ***General Pediatrician:*** *You always like to try to put a nice face on it and you look as hard as you can to make sense of it, but I was not able to find any natural diseases other than trauma to cause these injuries in this child.*
>
> ***Defense attorney:*** *You talk about a few minutes, examining the child, a few minutes looking at the x-rays, a few minutes reading the report. Totally, how much time did you spend examining this case?*
> ***General Pediatrician:*** *Probably there or at the hospital between a half hour and 45 minutes.*
>
> ***Defense attorney:*** *Did you make any attempts to get this child's prior medical records, prenatal record, anything like that.*
> ***General Pediatrician:*** *No sir, I did not.*
>
> ***Defense attorney:*** *Did you look up the medical Examiner's report?*
> ***General Pediatrician:*** *I have been made aware of it. I do not have it with me. I do not remember reviewing it very, very carefully.*

2. Diffuse axonal injury:

> ***Defense attorney:*** *In your report you talk about diffuse axonal injury; is that correct?*
> ***General Pediatrician:*** *Yes, Sir, that is the current theories regarding these children when there is significant bleeding in the brain as we saw in this case.*
>
> ***Defense attorney:*** *We are not talking about theories. In your examination of this child you actually did not see diffuse axonal injury?*

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General Pediatrician: No Sir, I did not.

3. Retinal hemorrhage:

Defense attorney: *Do you know how much hemorrhage was found in the child's retina in this case?*
General Pediatrician: *No, I do not.*

Defense attorney: *Do you know whether or not there was retinal hemorrhage in both eyes or one eye?*
General Pediatrician: *I am not sure which one or who did it.*

4. Rib fracture:

The state: *Are there fractures that can be the result of birth trauma?*
General Pediatrician: *Yes*

The state: *And what type of fractures would that be?*
General Pediatrician: *The most common fractures that we see is fractures of the clavicle which is the collar bone right here.*

The state: *Within a reasonable degree of medical certainty, rib fractures are considered to be abuse?*
General Pediatrician: *Yes*

The General Pediatrician's answers to the questions in court said it all. He spent only 30 to 45 minutes on the entire case. He did not see axonal injury. He did not know about the bleeding in the retina. He did not evaluate the child's and mother's medical records. He gave his conclusion about the cause of injury on November 25th when the baby was still alive. He said that the child had several rib fractures, but the medical records showed the baby had only a fracture in rib #6. Furthermore, my review of the medical literature revealed that multiple rib fractures do occur during labor. I presented the findings of four studies that show the occurrence of rib fractures during labor in Section 5.10 of this report. Also, I presented a study that showed fracture of long bones during labor. The General Pediatrician stated that the child's injuries were caused by trauma, but the examination by the Treating Physician on November 24th revealed no injuries due to trauma on the head or the body of the baby, except for a minor bruise under the right eyelid.

It is very hard to imagine that the General Pediatrician testified in a court on this very serious matter based solely on a theory; without evaluating the medical evidence and the related published medical literature. His testimony put an innocent father in prison for life and destroyed the family's structure. I believe that his unscientific and unjustifiable actions resulted in a tragedy which puts our society in danger. His actions should be investigated.

As previously explained in this report (Sections 4 and 5), the bleeding in the brain and other tissues in baby Alan resulted from his treatment with excessive amounts of heparin and sodium bicarbonate in the hospital. I reached my conclusions after spending more than two hundred and fifty hours evaluating the medical evidence and the related published medical information. Baby Alan's case and similar cases are very complicated medical matters as shown by the information presented in this report. They should be evaluated by using differential diagnosis prior to giving any conclusion. I am certain that spending only 30 or 45 minutes on this case, as the General Pediatrician did, could not lead to a valid conclusion. Physicians should take all facts into consideration when they are faced with a complicated task such as this one.

6.3 Review of Dr. Gary Steven Pearl's testimony

Dr. Pearl is a consultant neuropathologist working with the Medical Examiner's office. He examined the brain, spinal cord, and the eyes of baby Alan grossly on December 19, 1997 and also prepared slides. His pathology findings on the brain, spinal cord, and the right eye do not support the medical examiner's and other physicians' claims that the bleeding in the brain occurred in minutes or in a few seconds. Dr. Pearl examined the hematoma in the subdural space of the brain and spinal cord and observed the proliferation of fibroblasts in layers, clotted blood with no fibrosis, and fresh blood. He estimated the age of the oldest portion of the subdural hematoma to be two to five days [13]. This means that the blood was released in at least three stages as described in Section 5.4 of this report and this contradicts the theory of the other state witnesses that the bleeding in the brain occurred in minutes or in a few seconds.

Dr. Pearl also stated that baby Alan suffered from disseminated intravascular coagulation (DIC), which led to the hemorrhage in the subdural space and that the DIC was induced by brain injury. I agree with Dr. Pearl that the baby suffered from DIC, as shown clinically (Tables 9 and 12); but the reason for the DIC was the treatment with excessive doses of heparin as explained in this report (Sections 4 and 5). Dr. Pearl missed the role of heparin in the bleeding in the brain and spinal cord and in the formation of DIC because he did not review the baby's records and treatment chart during his five days in the hospital.

Furthermore, Dr. Pearl observed swollen blood vessels and acute degeneration of nerve cells in the brain and spinal cord and stated that these are signs of acute injuries. They occurred as a response to a brain injury. I agree with Dr. Pearl that these lesions are signs of acute injuries occurring within 2-5 days prior to autopsy, but these lesions were induced by severe hypoxia caused by excessive treatment with sodium bicarbonate on November 24th and 25th, by severe anemia, apnea, cardiac arrest, and hypotension, as explain in this report (Sections 4 and 5). The treatment with bicarbonate caused severe metabolic alkalosis. The blood pH was 7.7. This prevents the hemoglobin from releasing oxygen to the tissues and it caused hypoxia and cerebral edema in this case. Brain edema was confirmed at autopsy. Dr. Pearl missed the influence of these factors on the formation of the lesions in the brain and the spinal cord because he did not review Alan's medical records.

Dr. Pearl also observed meningeal inflammation and called it hemogenic meningitis (inflammation of the meninges resulting from the presence of blood). He based his conclusion on the quantity and type of inflammatory cells present in the inflamed tissue. This lesion lacked the presence of neutrophils. I believe that Dr. Pearl's interpretation of the tissue changes is incomplete because he did not review the baby's medical records. The baby was treated with high therapeutic doses of three types of

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antibiotics and this treatment reduced the severity of the acute inflammation observed at the time of autopsy. The swollen blood vessels, the presence of edema in the brain, fever (105.8ºF or 41.0ºC) and the elevated white blood cell count (20,900/μL) observed on November 24th (Table 12) indicate that the baby suffered from acute meningitis. However, the severity of the acute inflammation in the tissue was reduced by the treatment with high therapeutic doses of gentamicin, rocephin, and Claforan (cefotaxime sodium) on November 24th (Tables 6 and 7). The treatment with antibiotics reduced the blood white blood cell counts from 20,900 to 13,600/μL and body temperature from 105.8ºF (41.0ºC) to 99.8ºF (37.7ºC).

Also, Dr. Pearl did not present evidence in court that showed diffuse axonal injury. In addition, the Chief Medical Examiner's autopsy report makes no mention of Dr. Pearl's pathology findings on the brain, spinal cord, and eyes. Other state witnesses also did not present any evidence in court that showed diffuse axonal injury. However, they claimed that diffuse axonal injury is characteristic of "Shaken Baby Syndrome" (SBS). Hemorrhage in the retina is another claimed characteristic marker for SBS. Dr. Pearl stated that the bleeding in the right retina was very minor and did not impact his opinion in this case. Below are the defense attorney's questions presented to Dr. Pearl in court regarding the axonal injury and the retinal bleeding, and his answers to these questions.

1. Diffuse axonal injury:

> ***Defense attorney:*** *Now, I believe you testified that there were some slides that shows the diffuse axonal injuries?*
> ***Pearl:*** *That is correct.*
>
> ***Defense attorney:*** *Do you have those slides?*
> ***Pearl:*** *I do not have any of the slides.*
>
> ***Defense attorney:*** *But there were slides that were done that have shown the diffuse axonal injury?*
> ***Pearl:*** *There is a glass slide that does. I did not photograph that.*

2. Bleeding in the retina of the right eye:

> ***Defense attorney:*** *Was there hemorrhage in the retina or behind the retina?*
> *Pearl: There was a small hemorrhage.*
>
> ***Defense attorney:*** *Minute?*
> ***Pearl:*** *Minute.*
>
> ***Defense attorney:*** *Was that significant?*
> ***Pearl:*** *That wasn't significant to me. Really did not impact on my evaluation of the case.*

Furthermore, Dr. Pearl did not review Francine's medical records during her pregnancy with Alan, the baby's medical records from birth until his hospitalization on November 24, 1997, or the adverse reactions of vaccines and medications given to baby Alan. This limited his ability to interpret his pathology findings and to provide valid conclusions about the causes of injury and death.

6.4 Review of Dr. Douglas R. Shanklin's testimony

Dr. Shanklin, the defense witness evaluated the H & E stained tissue sections of the brain and other organs and stated that baby Alan died from natural causes. I agree with Dr. Shanklin that baby Alan suffered from health problems at birth and following birth. These included respiratory distress syndrome, jaundice, and growth retardation. However, these problems did not cause his cardiac arrest or the bleeding in the brain and other tissues observed in the autopsy. Alan's cardiac arrest was caused by hypokalemia developed as a result of metabolic and respiratory acidosis due to diabetes and pneumonia. Alan's health problems at birth and following birth increased his susceptibility to develop adverse reactions to vaccines. The six vaccines that he received on November 11, 1997 caused his infections and diabetes as explained in Sections 3 and 5 of this report. The bleeding in the brain and other tissues was caused by the excessive treatment with heparin and sodium bicarbonate given in the hospital, as explained previously.

Dr. Shanklin made very important contributions to this case. He stated that baby Alan's kidneys were not fully developed. This finding may explain the mother's problem with oligohydramnios as explained in Section 3 of this report. He also stated that the baby suffered from pneumonia and meningitis in the brain region and the spinal cord. His findings may explain the susceptibility of these regions to bleeding caused by the treatment with heparin and sodium bicarbonate. The inflammation in these regions affected the integrity of the blood vessels and caused predisposition to the leakage of fluid and blood induced by treatment with excessive doses of heparin and sodium bicarbonate. In addition, he described old neurological injuries in the brain and spinal cord. I believe that the high levels of bilirubin observed in the first week following birth caused these injuries (Table3). I addressed this issue in Section 3.

6.5 Analysis of the state's claim

During Alan R. Yurko's jury trial, the prosecutor, Ms. Wilkinson, presented only one theory—that Baby Alan died as a result of "Shaken Baby Syndrome" (SBS), and Mr. Yurko, the father of the child, did it. My review of the medical evidence and the trial transcript revealed that the prosecutor did not prove her case that the injuries were caused by trauma and that Mr. Yurko abused his child. However, the prosecutor achieved her goal of convicting Mr. Yurko of a horrible crime he did not commit. He received a life sentence plus 10 years. I believe that the prosecutor used unfair practices and violated Mr. Yurko's right of getting a fair trial. Below is a list of the prosecutor's unfair tactics and evidence that shows the state did not prove its case.

1. The prosecutor presented trauma as the only possibility for the cause of injuries in this case; but the Treating Physician examined the baby on November 24th, and his examination did not reveal any injury caused by trauma, except for the minor bruise under the right eyelid. In addition, Mr. Yurko never

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stated that he abused his child, and no one observed him hurting baby Alan.

2. The prosecutor did not discuss the adverse reactions of vaccines given to baby Alan (Table 5). The studies presented in Section 3 of this report clearly show that these vaccines caused severe adverse reactions, such as apnea, cardiac problem, and respiratory infections, in premature infants. Baby Alan developed diabetes resulting from an infection and complications of his vaccination. This led to his cardiac arrest on November 24, 1997.

3. The prosecutor did not discuss the adverse reactions of medications given to Alan on November 24th through November 29th. It is very obvious that heparin causes bleeding at the doses given.

4. The prosecutor stated that the baby developed subdural bleeding in the brain, retinal bleeding, and diffuse axonal injury as a result of SBS. However, the autopsy and the pathology findings clearly show that the subdural bleeding did not occur in a few minutes as the SBS theory claimed. In addition, none of the state witnesses presented evidence in court that they found diffused axonal injury. Also, the bleeding in the retina of the right eye was very minor. Even Dr. Pearl stated that this injury was very minute and had no impact on his evaluation.

5. The prosecutor did not ask the Chief Medical Examiner to provide the x-ray findings to prove that Alan had fractures in ribs #5, 7, and 10. He stated in his autopsy report that he used an x-ray to confirm his findings. In addition, the prosecutor held the x-ray findings taken on September 16, 17, and 18 as proof that the rib fractures did not happen during labor. It takes at least 7 days for the calluses to form, and on September 18, 1997, the baby was 3 days old. However, the prosecutor did not accept the x-ray findings of November 24th and 25th that the baby had only rib #6 fracture. She stated that the fractures are better seen at autopsy, but she overlooked the fact that the Chief Medical Examiner stated in his report that he took the x-ray to confirm the rib fractures. It seems that the prosecutor was trying to select certain evidence to support her theory that the father was abusing his son and caused multiple rib fractures. Her actions showed that she did not conduct her business in a fair and impartial manner.

6. The prosecutor allowed the Chief Medical Examiner to present in court as evidence of trauma two photographs of minor contusions in the temporal areas of the head that occurred in the hospital about one day prior to autopsy. The medical examiner's main objective should be discussing the causes of injuries that caused death in this case. I believe he used these photos to influence the jury's thinking that physical force was used, and the prosecutor should not have allowed this.

7. None of the state witnesses evaluated all of the evidence relevant to this case, such as prenatal records and the baby's medical records from birth until the time of his cardiac arrest, and the prosecutor did not question the validity of their testimonies.

7. Conclusions and Recommendations

Baby Alan Ream Yurko suffered from several serious heath problems at birth and following birth, such as respiratory distress syndrome, hypoglycemia, jaundice, growth retardation, and bacterial infections. His serum bilirubin level was 17.4 mg/dL at 3 days post-birth, which is capable of causing encephalopathy. Also, his risk of developing encephalopathy and of suffering from hypoxia was increased by the treatment with antibiotics that bind with albumin and release bilirubin. In addition, baby Alan had high risk of developing congenital deformity of organs and the skeleton because his mother suffered from gestational diabetes and oligohydramnios during the pregnancy. His growth rate in the first month of life was poor, but he showed a good improvement in his growth rate during the second month.

Unfortunately, his pediatrician gave him six vaccines at two months of age and sent him home without monitoring or medical supervision. It has been reported that premature children, such as baby Alan, who were vaccinated prior to 70 days of age showed very high risk of developing serious health problems, such as apnea, bradycardia, and oxygen desaturation, that required medical intervention. Alan developed fever, reduction in food intake, lethargy, and became irritable a few days following vaccination. The medical evidence indicates that his vaccination induced these symptoms.

At thirteen days post vaccination, the baby had cardiac arrest and apnea. The clinical tests revealed that he was suffering from diabetes mellitus and the complications of diabetes, such as bacterial infections, gastric ulcer, metabolic acidosis, hypokalemia, dehydration, anemia, weight loss, loss of muscle tone, and cardiac arrest. The six vaccines received on November 11, 1997 were the likely cause of his infections and diabetes (Table 5). His cardiac arrest and apnea on November 24th, resulted from hypokalemia which was a result of metabolic and respiratory acidosis. It appears that his diabetes was induced by bacterial infections, because his blood sugar level of 397 mg/dL on November 24th returned to normal two days following IV antibiotics. He also had pneumonia, meningitis, eye infection, and liver and heart damage. His liver serum enzymes and LDH levels were also reduced significantly following the treatment with antibiotics, and this indicates that he had liver, and maybe heart, bacterial infections (Table 12).

Unfortunately, at Florida Hospital baby Alan was treated on November 24-25, 1997 with high doses of sodium bicarbonate that caused metabolic alkalosis (pH 7.7) and severe hypoxia. He was also treated with high doses of heparin that caused bleeding in tissues. Heparin caused severe subdural bleeding in the brain and spinal cord, minor bleeding in the brain and the retina of the right eye, and bleeding in the lungs. It also caused disseminated intravascular coagulation (DIC). I am very surprised to see that the Treating Physician treated Alan with high doses of sodium bicarbonate and heparin at 8:00 AM on November 25th despite the fact that he was suffering from severe metabolic alkalosis (pH 7.61-7.7) and bleeding in the brain and subdural space (Tables 8 and 11). Treatments with high doses of sodium bicarbonate and heparin were not medically justified.

The treatment with bicarbonate for individuals with diabetes should stop at pH 7.2, and this treatment carries high risk of causing cerebral edema, as happened in this case. Heparin should not be given to patients suffering from bleeding, hypotension, and anemia, because of the high risk of bleeding in such patients associated with heparin. Baby Alan had hypotension, anemia, hypoxia, bacterial infections in several organs,

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bleeding gastric ulcer, and bleeding in several organs. It seems that the Treating Physician overlooked the adverse reactions of sodium bicarbonate and heparin, as well as the recommendations presented in the PDR and medical textbooks concerning the treatment with these agents.

The radiology findings of November 24 and 25, 1997 showed that Alan had only rib #6 fracture. Rib fractures have been observed to occur during labor even in mature babies. Also, oligohydramnios can cause positional skeletal deformity. The medical evidence indicates that the fracture of rib #6 most likely happened during labor. The claim of the Chief Medical Examiner that the baby had additional ribs fractures is not valid, because he did not provide any evidence to support his claim, such as x-ray results and films. In addition, there were no calluses observed in the cut sections of the swollen cartilage at autopsy.

The Chief Medical Examiner's autopsy report and his court testimony related to this case suffer from accuracy problems and contain very serious contradictions. They are certainly not reliable medical evidence to be used to answer questions about the cause(s) of death. He stated that the heart was normal and described the histology of the heart, but in the same report, he mentioned that the heart was donated prior to his examination. This indicates that the Chief Medical Examiner was describing the heart of a different child and got his cases mixed up. This is very serious, and his work should be investigated. Also, he stated in his report that the cerebrospinal fluid was clear; whereas, in court he said that the CSF fluid was mixed with blood. This is another serious problem found in the Chief Medical Examiner's work.

In addition, the Chief Medical Examiner said in court that the baby did not have meningitis, but his report does not contain any description of the histology of the meninges to show that he examined them. The other two pathologists who evaluated the slides of the meninges observed acute changes that indicate meningeal inflammation. Furthermore, he reported that the baby's head circumference was 22 cm, and that is obviously wrong. It was 37.5 cm eighteen days prior to the autopsy date.

Furthermore, the Chief Medical Examiner did not provide the medical evidence in his report and in court to support his conclusion that baby Alan died as a result of "Shaken Baby Syndrome." The Chief Medical Examiner stated that the subdural bleeding in the brain occurred in a few minutes or even in a few seconds due to vigorous shaking. However, the autopsy and the pathology findings showed that the bleeding occurred during 2-5 days and at least in three stages. The bleeding also occurred in the spinal cord and the lungs, which has nothing to do with the SBS theory, but it indicates that the bleeding resulted from cardiovascular problems. In addition, he did not show any evidence that the baby had diffuse axonal injury in the brain. No description of axonal injury was presented in his report, and he did not show a single slide describing such a lesion during the trial. The other three state witnesses also did not show any slide or picture of an axonal injury.

Furthermore, I find the Chief Medical Examiner's conclusion about the cause of death invalid, because he did not review the baby's prenatal records, the mother's records during her pregnancy with Alan, or adverse reactions of vaccines and medications given to the baby. He missed the facts that the baby had diabetes, hypokalemia, anemia, metabolic alkalosis, bleeding as a result of heparin treatment, and hypoxia and cerebral edema due to the treatment with bicarbonate.

In addition, my review of the medical literature revealed that axonal injury in the brain and spinal cord can occur in cases of cardiac arrest, edema, hypoglycemia, and from other causes; and it is not necessarily a characteristic lesion of injuries caused by trauma. Also, treatment with high doses of heparin and sodium bicarbonate can induce bleeding in cases of hypotension, anemia, and inflammation in tissues, as happened in this case. Heparin and sodium bicarbonate are commonly-used agents to treat clotting disorder and acidosis. Therefore, cases of SBS should be investigated to rule out the contribution of therapeutic agents and other factors in the pathogenesis of brain lesions. I believe that cases of individuals who were convicted of killing babies by SBS based on the axonal injury, subdural bleeding, and eye bleeding should be reexamined.

Also, I believe that the Chief Medical Examiner's action of presenting in court two photographs of minor contusions that occurred in the hospital, which were unrelated to the cause of death, is scientifically and professionally unjustified. I believe that he did it to influence the thinking of the jury that physical force had been used on the baby.

My review of the evidence indicates that the Treating Physician's testimony in court is unsupported by science and medical facts. He did not review the medical evidence related to this case such as prenatal record, the baby medical record, and the pathology findings. In addition, he did not review related published literature describing the adverse reactions to vaccines and medications given in this case. Also he did not reveal to the court that the baby was treated with three types of antibiotics to fight infections, bicarbonate and heparin; and that the baby suffered from diabetes, dehydration, hypokalemia and complications of diabetes. I believe that withholding of these vital data from the court resulted in a negative outcome of the trial, and this issue should also be investigated.

The Treating Physician's treatment of baby Alan with high doses of bicarbonate and heparin caused serious injuries and death and should be investigated. In addition, this treatment produces bleeding in the brain and other tissues and axonal injuries, which are considered by the proponent of SBS as characteristic markers for it. It led to the false conviction and the imprisonment of Mr. Yurko.

I believe that the General Pediatrician's testimony in court was also unsupported by medical evidence and science, based on the facts that he did not review the medical evidence that related to this case such as prenatal record, the baby medical record and the related published literature describing the adverse reactions to vaccines and medications given, and the pathology findings. He spent only 30-45 minutes on this case, and this certainly did not give him the expertise to testify on this complicated medical case.

Alan Yurko and his family suffered two tragedies as a result of problems with our current medical system regarding the vaccination of premature babies, the treatment given to baby Alan in the hospital, and the approaches of the state witnesses in evaluating the evidence in this case. The first tragedy is the loss of baby Alan because of adverse reactions to vaccines and the excessive doses of heparin and sodium bicarbonate given in the

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hospital. It seems that his pediatrician was unfamiliar with the adverse reactions of vaccines in premature children. He gave him six vaccines and sent him home. The Treating Physician also overlooked the adverse potentials of heparin and bicarbonate. These issues should be addressed by the state and medical authorities, which might save babies from death due to adverse reactions to vaccines and treatment with the wrong medications.

The Yurkos' second tragedy was the conviction of Mr. Yurko with a horrible crime he did not commit. He was convicted because the state's four expert witnesses did not take the time to review the evidence and the related published literature. Nor did they sort out the facts, so that their testimonies were based on a theory only. The prosecutor contributed to the problem by focusing on only one theory. She also allowed the Chief Medical Examiner to present evidence that had no connection to the case such, as the photographs describing the two contusions on the baby's head.

I believe that the state of Florida has the responsibility to review the evidence presented in this report. It shows that Mr. Yurko is innocent, and they should take speedy action to free him from prison. The objective of the state and the medical system should be determining the factual causes that led to the illness and death of a child and to prevent such problems from happening to other children. Accusing innocent parents of abusing and killing their children based on unsupported theory, as it happened in this case, will not prevent the death of other children by vaccines and incorrect medications. But it certainly puts innocent people in prison and causes suffering. It also costs the taxpayers huge sum of money to pay for trials and legal fees.

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