

See discussions, stats, and author profiles for this publication at: <https://www.researchgate.net/publication/237525747>

# Did Bryant Arroyo kill baby Jordan Anthony Shenk, as alleged by the Commonwealth of Pennsylvania?

**Article** in *Medical Veritas The Journal of Medical Truth* · April 2005

DOI: 10.1588/medver.2005.02.00046

---

CITATIONS

0

---

READS

87

**1 author:**



**Mohammed Al-Bayati**

Toxi-Health International

**50** PUBLICATIONS **480** CITATIONS

SEE PROFILE

# Did Bryant Arroyo kill baby Jordan Anthony Shenk, as alleged by the Commonwealth Of Pennsylvania?

**Mohammed Ali Al-Bayati, PhD, DABT, DABVT**

Toxicologist & Pathologist

Toxi-Health International

150 Bloom Dr.

Dixon, CA 95620 USA

Phone: +1 707 678 4484 Fax: +1 707 678 8505

Email: maalbayati@toxi-health.com Website: <http://www.toxi-health.com>

Submitted: January 29, 2005 Accepted: March 5, 2005

---

## Abstract

Bryant Arroyo was arrested in Lancaster County, Pennsylvania on September 26, 1994 in connection with baby Jordan Anthony Shenk's death. On May 10, 1995, he was convicted of first-degree murder and sentenced to life imprisonment without parole. The medical examiner testified at Arroyo's preliminary hearing and trial that Jordan was killed by blunt trauma to the chest and abdomen, and that the manner of death was homicide. My investigation of this case clearly showed that Jordan died as a result of serious illnesses that led to his cardiac arrest and bleedings on September 25, 1994. He had brain disease (spongiosis of the cerebral cortex and white matter, and focal Purkinje cell dropout in the cerebellum); aspiration pneumonitis; sepsis; inflammation of the liver, gallbladder, and the mesentery; thymus atrophy; and weight loss. These lesions and symptoms have been reported in children with propionic acidemia and other metabolic problems involving branched amino acids. The baby's symptoms and lesions indicate that he probably suffered from genetic illness that led to the development of propionic acidemia and his death. The government and the medical authority in the State of Pennsylvania should evaluate the medical evidence that shows Bryant Arroyo was falsely accused and unjustly convicted of killing Jordan Anthony Shenk because the factual causes of illness and death in this case were not revealed to the jury. I believe that Bryant should be released from prison immediately and compensated for his pain and suffering and time wrongly spent in prison.  
© Copyright 2005, Pearlblossom Private School, Inc.—Publishing Division. All rights reserved.

*Keywords:* propionic acidemia, homicide, trauma

---

## Summary of the Case

Jordan's mother left home at about 0005 on September 25, 1994 and left her boyfriend, Bryant Arroyo watching her four-year-old son and her baby, Jordan Anthony Shenk, asleep in his crib upstairs. Jordan was eight-and-a-half months old, and Bryant was 22 years old. Jordan's mother and her three children had lived with Bryant since June of 1994.

Jordan's mother returned home about two hours later, at approximately 0200. At about 0240 Bryant went upstairs to check on Jordan and found him unconscious and lifeless in his crib. He was not breathing. Bryant carried the baby downstairs and yelled to the mother to call 911. He put the baby on a chair and performed cardiopulmonary resuscitation (CPR).

The police and paramedics arrived a few minutes after they received the call. They also performed CPR on Jordan to revive him, but they were unsuccessful. The paramedics transported Jordan to Lancaster General Hospital at 0311, where he was pronounced dead at 0340. He was treated with epinephrine, atropine, sodium bicarbonate, and oxygen by the paramedics and in the hospital. He was also given 410 ml of fluid IV by the medical staff.

The paramedics examined Jordan and observed eleven brownish/purplish circular marks on his lower chest and upper abdomen, which were approximately 1/8 of an inch in diameter. Jordan's mother stated that she observed some bruising on the chest wall resulting from Bryant's attempts to perform CPR.

The paramedics and the physician at the emergency room also noted an old erythematous mark (1/2 x 5 cm) on the baby's

left cheek. As stated by Jordan's mother, this was a burn mark caused by an iron about two weeks prior to the baby's cardiac arrest. The paramedics and the emergency physician did not notice any other mark or injury on Jordan's body.

Dr. Wayne K. Ross, forensic pathologist of Lancaster County, Pennsylvania, performed the autopsy on Jordan at about 0800 on 25 September 1994 [Autopsy No: A94-347 (LC94-265)]. Prior to autopsy, full body x-rays were performed and showed no evidence of acute or healing trauma. Dr. Boal, pediatric radiologist, also reviewed these x-rays and did not observe any significant abnormalities. In addition, Dr. Ross examined Jordan's body and observed no evidence of injuries caused by trauma in the scalp, mouth, back, and upper and lower extremities.

Randy Miller, Assistant District Attorney, Officer Roland L. Buch, and Detective Allen W. Leed from the Manheim Township Police Department were present with Dr. Ross and observed the autopsy. Dr. Ross took tissue samples from selected organs and lesions at the time of autopsy and submitted them to Harrisburg Hospital to process them for histological evaluation.

After completing his autopsy on 25 September, Dr. Ross went to Manheim Township Police Department and met with officers and detectives. He informed them that Jordan died from complications of blunt trauma to the chest and abdomen and that the manner of death was homicide. He also provided the same information to Mr. Miller in the district attorney's office.

Bryant Arroyo was arrested in Lancaster County, Pennsylvania on 26 September, 1994 in connection with Jordan's death. On 10 May 1995, he was convicted of first-degree murder and

sentenced to life imprisonment without parole. Dr. Ross testified at Bryant's preliminary hearing and trial that Jordan was killed by blunt trauma to the chest and abdomen, and that the manner of death was homicide.

Moreover, Detective Larry Mathias and Detective Solt from the Manheim Township Police Department alleged that Bryant confessed to them that he killed Jordan because he had an anger problem, was angry with his cousin at the time, and took it out on the baby. The Commonwealth presented to the jury an eight-page confession typed out by Det. Solt.

Bryant was not allowed to testify in his trial, and his public-defender lawyers did not retain a medical expert to review Dr. Ross's pathology findings and to testify for the defense. Bryant denied the allegation that he killed Jordan or confessed to the police. In addition, Bryant had nineteen character witnesses—including Jordan's mother and grandmother—who testified and/or wrote affidavits on his behalf and rejected the police's claim that Bryant had an anger problem.

Nancy Garcia, President of Families United for Justice in Springfield, MA contacted me and asked me to review the medical evidence in Bryant's case and to provide an opinion concerning the factual causes that led to Jordan's death. Nancy is Bryant's representative. Bryant and his mother also contacted me and asked me to evaluate the case.

I evaluated the medical evidence, trial documents, testimonies of witnesses, police reports, and medical literature pertinent to Jordan's case. I used differential diagnosis to identify the possible causes that led to the bleedings and death, and the synergistic actions among these causes. I describe my findings in Sections 1 through 5 of this report. Section 6 contains my conclusions and recommendations.

Jordan was born on 6 January 1994 by caesarian section at 41 weeks of gestation. His birth weight was 5 pounds, 15 ounces. He was taken to his pediatrician several times for regular checkup between 24 January and 12 August 1994. His last visit to his pediatrician was 43-days prior to his cardiac arrest on 25 September.

Jordan's weight on 12 August was 15 pounds, 10 ounces, which is identical to his weight on 25 September (15 lb and 10 ounces) after he received 410 ml of fluid IV (weighing about one pound) following his cardiac arrest. These data indicate that Jordan lost about one pound during the forty-three days prior to his cardiac arrest. In addition, Jordan's thymus weight at autopsy was 13 g, which is about 52% of normal, indicating thymus atrophy and immune depression. Jordan's mother and his babysitter reported that he had a cold during the week prior to his cardiac arrest.

As noted, Dr. Ross performed an autopsy on Jordan's body on 25 September 1994 and concluded that Jordan was killed by blunt trauma to the chest and abdomen and that the manner of the death was homicide. However, the medical evidence described in Section 3 of this report clearly shows that Jordan died as a result of serious acute and chronic illnesses. He suffered from spongiosis of the brain (cerebral cortex and white matter); focal Purkinje cell dropout in the cerebellum; aspiration pneumonitis; weight loss; thymus atrophy; inflammation of the liver, gallbladder, and mesentery; sepsis; metabolic disease; and internal bleedings.

The presence of these significant pathological abnormalities in the brain, lungs, and other organs explain the sequence of events that led to Jordan's cardiac arrest on 25 September. The brain lesions caused neurological, gastric, and esophageal reflex problems that led to aspiration pneumonitis. This situation caused sepsis, thrombocytopenia, inflammation in liver and other organs, deficiency of blood clotting factors and internal bleedings, lung bleedings and edema, asphyxia, and cardiac arrest.

The objectives of Dr. Ross's investigation were to find the factual causes and the manner of death. My investigation revealed that Dr. Ross did not achieve these objectives, because he did not follow standard medical procedures in his investigation. He also rushed to judgment in providing the cause and the manner of death to the Manheim Township Police and the assistant district attorney. In addition, he did not reveal important medical evidence to the court that explains the factual causes of bleedings and death. The following is a list of clinical medical data that support my conclusions.

1) Dr. Ross overlooked the fact that Jordan was suffering from weight loss during the forty-three-day prior to his cardiac arrest. As stated above, the baby lost about one pound. Jordan also suffered from thymus atrophy. These data indicate that the baby suffered from chronic illness.

2) Dr. Ross examined Jordan's body at about 0800 on 25 September, observing sixteen circular, red and brown minor contusions of various sizes, 1/8-1/2 inch in diameter, on the lower anterior chest wall and upper abdomen. He assumed that these bruises were present when the paramedics picked the baby up at his house, and that these bruises were caused by blunt trauma inflicted intentionally prior to the baby's death. I find Dr. Ross's assumptions scientifically invalid based on the following medical facts:

a. The paramedics examined Jordan prior to transporting him to Lancaster General Hospital at 0305 and they found only eleven brownish/purplish circular bruises and marks on his lower chest and upper abdomen. The size of the marks was approximately 1/8 inch in diameter. These data indicate that five bruises or marks of the sixteen bruises and marks reported by Dr. Ross were formed after the baby was transported by the paramedics. In addition, the sizes of these bruises and marks increased by 400% from the time the baby was transported by the paramedics at 0305 until the time of autopsy at 0800.

b. Dr. Ross examined the H & E stained tissue sections from the bruises and marks on Jordan's lower chest and the upper abdomen. He did not find a significant white blood cell infiltration in these areas. Also, in the abdominal tissue, he saw only congested blood vessels without hemorrhage. These observations contradict Dr. Ross's conclusions that the bruises in the chest area occurred when the baby was alive and that the baby had bruises on the upper abdomen.

c. Various types of injuries in the chest and abdomen have been reported in patients receiving cardiopulmonary resuscitation (CPR), and Jordan's mother stated that the bruises on the baby's chest were caused by CPR.

d. Jordan had spongiosis of the brain, aspiration pneumonitis (leading to sepsis), and inflammation of the liver. Thrombocy-

topenia has been reported in children who have sepsis and brain spongiosis. Thrombocytopenia and inflammation of liver increase susceptibility to bruising and bleedings.

3) Dr. Ross examined the H and E stained tissue sections of Jordan's brain microscopically and found serious, chronic brain disease—namely spongiosis of the cerebral cortex and white matter, and focal Purkinje cell dropout in the cerebellum. But Dr. Ross did not consider this an important discovery. Furthermore, he did not reveal these findings to the court when he testified in October of 1994 and May of 1995. These lesions are observed in babies with propionic acidemia (PA) and other metabolic problems associated with branched amino acids. PA is a genetically related (recessive gene) metabolic problem. It also causes aspiration pneumonitis, thymus atrophy, sepsis, bleedings, and weight loss—all observed in Jordan's case.

4) Dr. Ross stated that Jordan's thymus weight was 13 grams. He was eight-and-a-half months old at the time of autopsy. The average thymus weight (g) in a white infant male at six to nine months of age is reported to be 25 g., making Jordan's thymus weight about 52% of normal. These data indicate that Jordan suffered from thymus atrophy. Reportedly, babies who had PA and spongiosis of the brain also had thymus atrophy and serious infections, as observed in this case.

5) Dr. Ross examined the H & E stained lung tissue sections microscopically and found aspiration pneumonitis. However, he did not consider this an important discovery in his evaluation. He also did not reveal this crucial information in court when he testified in October of 1994 and May of 1995.

Aspiration pneumonitis is usually caused by a foreign substance (food and/or secretions) and microorganisms entering the lung causing inflammation. It has occurred in children with poor airway reflexes or gastroesophageal reflux, or both. Jordan had serious neurological problems that caused poor airway and gastroesophageal reflexes, namely spongiosis of the cerebral cortex and white matter, and focal Purkinje cell dropout in the cerebellum.

Inflammation of the lungs due to the aspiration of foreign substance and bacteria can explain the bleedings and edema of the lungs observed by Dr. Ross. In the earliest stage of pneumococcal pneumonia, protein-rich edema fluid containing numerous organisms usually fills the alveoli. Marked congestion of the capillaries is followed by a massive outpouring of polymorphonuclear leukocytes and accompanied by intra-alveolar hemorrhage. On gross examination, the lungs of patients who die of streptococcal pneumonia are heavy and display bloody edema, which is identical to Dr. Ross's observation in Jordan's case.

6) Dr. Ross examined the H & E stained tissue sections of Jordan's liver and found that the liver was totally infiltrated, primarily with polymorphonuclear cells (PMN) but with macrophages as well. The presence of the PMN and macrophages in the liver indicates inflammation and that the inflammation was about 24 hours old or more. The PMN usually enter the inflammatory site within 6 to 12 hours after initial injury, while macrophages enter later, usually after 24 hours. Bacteria were the likely cause of the liver inflammation. Jordan had aspiration

pneumonitis, and bacteria can travel via blood and infect other organs. Dr. Ross did not take a blood sample or abdominal fluid to culture them for bacterial growth in order to exclude bacteria as a cause in this case.

7) Dr. Ross examined Jordan's gallbladder at autopsy and found early necrosis associated with edema in the gallbladder wall. He also examined the H and E stained tissue sections of the gallbladder and found a blood clot, autolysis of epithelium, and subepithelial hemorrhages with early infiltration of PMN.

It takes three to four hours for necrosis in the gallbladder to become apparent to the naked eye. It takes longer to observe cell autolysis, which is caused by enzymes activated and released from the lysosomes of dead cells. Dead cells evoke an inflammatory reaction that brings leukocytes to the area. The autolysis observed in Jordan's case occurred prior to death, because the wall of the gallbladder was infiltrated by white blood cells [23,24]. It is not medically possible for all these events to occur in less than two hours. In addition, the inflammation and necrosis in Jordan's gallbladder were likely caused by microorganisms, because the baby was suffering from aspiration pneumonitis, liver inflammation, thymus atrophy, and immune depression.

8) Dr. Ross examined the H & E stained tissue sections of the mesentery and the intestines, and observed fresh hemorrhage with inflammation in the mesentery. The mesentery was also infiltrated with PMN and macrophages. The presence of the PMN and macrophages in the mesenteric tissue indicates that Jordan's mesentery was inflamed and that the inflammation was about 24 hours old or more, as explained above. In addition, bacteria were the likely cause of the inflammation of the mesentery, because of the aspiration pneumonitis, inflammation of the liver and other organs, and thymus atrophy as explained above.

9) Dr. Ross examined Jordan's gastrointestinal tract, liver, and gallbladder grossly and microscopically. He observed fresh bleedings in liver, wall of gallbladder, multiple areas of the mesentery, pericolonic region, serosa of small intestine, and ascending colon. He also collected 200 ml of bloody fluid from the abdominal cavity. The bleedings in these sites arose from the synergistic actions of several factors. These include: a) bacterial infections; b) probable thrombocytopenia due to sepsis and metabolic problems; c) inflammation of the liver, causing deficiency of clotting factors as explained in Section 3; d) severe damage of liver, gastrointestinal tract, and other internal organs, possibly caused by cardiopulmonary resuscitation CPR, as has been reported in medical literature.

10) Without taking blood sample and/or sample from the abdominal fluid to do bacterial culture, Dr. Ross assumed that the inflammation in the liver, gallbladder, and mesentery were caused by trauma. Bacterial inflammation of the lung (aspiration pneumonitis) can lead to the inflammation of other organs.

Detectives Mathias and Solt alleged that Bryant Arroyo freely confessed to them after 1600 on 26 September 1994 that he killed Jordan. Mathias and Solt claimed that Bryant confessed to them when they told him that the medical examiner

informed them that Jordan's injuries were intentional and not caused accidentally by somebody performing CPR. The officers told Bryant that they believed he was responsible for causing Jordan's injuries.

The police alleged that Bryant told them he went upstairs to Jordan's room after 0030 on 25 September; that he picked the baby up with his left hand under the baby's arm and started punching the baby; that he then switched the baby over to hold him with his right hand and punched the baby with his left hand; that he then laid the baby down on his older brother's bed and punched him some more. It was alleged that Bryant punched the baby in the chest and stomach area a few times, and that he then put the baby back in his bed and laid him face down.

Bryant has claimed that he did not confess to the police that he killed Jordan, but that the police altered his statement of 26 September describing Jordan's cardiac arrest incident and the way he did CPR to rescue the baby. Bryant's alleged confession is an eight-page typed-document produced by Det. Solt with a computer and a word-processing program. There are no copies of Bryant's alleged confession handwritten by Bryant or the detectives to compare with the computer-generated document. Det. Solt discarded all interview notes.

I found that Bryant's alleged confession described above does not dovetail with the medical evidence pertinent to this case, or with the sequence of events that led to the baby's cardiac arrest and death. Below is a list of medical evidence and facts that raise doubt about the police's claim that Bryant confessed to them he killed Jordan.

1) Jordan's weight on 25 September was about 15 lb. The police alleged that Bryant picked Jordan up by one arm with a single hand. Then, using his other hand, Bryant punched the baby with a strong force in the chest and the abdomen. Jordan had serious neurological problems, aspiration pneumonitis, liver inflammation, and sepsis. Reportedly, babies who had these health problems also had thrombocytopenia and bleeding problems. I do not believe it is possible to manhandle a healthy 15 lb baby in the manner described by the police, not to mention a baby with the bleeding problems Jordan had, without leaving bruises and marks.

The paramedics, emergency physician, and the medical examiner examined Jordan on 25 September and did not see any evidence of trauma on his the upper and lower extremities. The medical examiner reported that Jordan's upper arms, forearms, and hands are symmetrically developed and free of evidence of trauma, edema, and other significant natural diseases. In addition, prior to autopsy, full body x-rays were performed and showed no evidence of acute or healing trauma.

2) It was alleged that Bryant punched Jordan repeatedly with his hands in the upper abdominal region. Punching a baby with bleeding problems repeatedly, with a strong force, as described in Bryant's alleged confession and by the medical examiner, will certainly causes serious bruises. Dr. Ross examined the H & E stained tissue section from the marks on the skin on the abdominal area and observed only congested blood vessels with no obvious hemorrhage or inflammatory reactions. These findings do not support the allegations that the baby was hit in the

abdominal region with strong force (or with any force significant enough to cause injury) as described in Bryant's alleged confession.

3) The sizes of the bruises on the lower chest area increased by 400% from the time the paramedics picked up the baby from his house at 0305 until the time of autopsy at 0800 (Section 3). These data and the evidence described in Section 3 suggest that these minor bruises (1/8 inch) were caused when people performed CPR on Jordan.

4) Jordan suffered from spongiosis of the cerebrum and Purkinje cell dropout in the cerebellar cortex; aspiration pneumonitis; thymus atrophy; inflammation of the liver, gallbladder and mesentery; and severe weight loss. Babies with these health problems also reportedly suffered from thrombocytopenia and bleedings (Section 3). These are the factual causes of Jordan's cardiac arrest and bleedings, and not blunt trauma as alleged by the medical examiner and the police.

5) The events described in Bryant's alleged confession dealing with the manner of death in Jordan's case dovetail only with the medical examiner's theory that Jordan was killed by repeated blows to the chest and abdomen. The medical examiner's theory is not supported by the medical facts. In addition, the medical examiner shared his theory with the police on 25 September before any microscopic examinations had been done.

6) Bryant was interviewed three times by the police prior to 1600 on 26 September, and on each occasion he stated that he did not hurt Jordan. He found him unconscious in his crib and performed CPR to revive him. Bryant's story was consistent on the three occasions, and it dovetails with the medical facts, whereas the allegation by police does not.

7) As noted earlier, the police alleged that Bryant confessed to them that he had an anger problem and that he killed Jordan to let his anger out. They alleged that Bryant was angry with his cousin on 24 September. Bryant had nineteen character witnesses who knew him well—including Jordan's grandmother, mother (who had lived with Bryant for four months), and Bryant's cousin—who testified and/or wrote affidavits on his behalf stating that he is a very calm person and they never saw him getting angry, and rejecting the police's claim that Bryant had an anger problem.

The medical evidence and facts described above clearly contradict the statements made by Detectives Mathias and Solt that Bryant confessed to them that he had an anger problem and killed Jordan because of it. These facts also indicate that Bryant did not provide the statements cited in his alleged confession describing the methods and the manner of Jordan's death. I believe that the State of Pennsylvania and the court need to re-examine the medical data presented in this report and ask Detectives Mathias and Solt to explain their positions.

Furthermore, the medical evidence in this case clearly contradicts Dr. Wayne K. Ross's claim that Jordan died from blunt trauma to the chest and abdomen and that the manner of death was homicide. The medical evidence clearly shows that Bryant was falsely accused. He was unjustly convicted because the

factual causes of illness and death were not revealed to the jury by the medical examiner and police.

I hope that the government and the medical authority of the State of Pennsylvania will evaluate the medical evidence presented in this report and open an investigation to correct the injustice. I believe that Bryant Arroyo should be released from prison immediately and should be compensated for pain and suffering and for the time spent in prison.

In addition, the State of Pennsylvania should inform Jordan's family about the factual causes of the baby's illness and death. The medical evidence clearly shows that he died from a genetically related metabolic problem, propionic acidemia. The gene involved is recessive. Jordan's family should be alerted to test their other children and future children for this fatal genetic defect to prevent similar tragedy.

### 1. Jordan's Medical History Prior to His Cardiac Arrest

Jordan Anthony Shenk was born on January 6, 1994 by caesarian section at 41 weeks of gestation. His birth weight was 5 pounds, 15 ounces. He was taken to his doctor for a regular check up several times between 24 January and 12 August 1994. He received several vaccines between 9 January and 20 June 1994, as shown in Table 1. These include hepatitis B, diphtheria/tetanus/pertussis (DTP), polio, and hemophilus B conjugate vaccines [1].

Jordan suffered a fatal cardiac arrest on 25 September 1994 at the age of 8 months and 19 days. His body weight at autopsy was 15 pounds, 10 ounces [2], which was the same as his weight on 12 August 1994 (Table 2). These data suggest that Jordan did not gain any weight during the forty-three-days prior to his cardiac arrest.

However, Jordan was given more than 400 ml of fluid (about one pound) IV following his cardiac arrest on 25 September by the medical staff (Tables 3, 4). Subtracting the weight of fluid given from the weight measured at autopsy gives an actual body weight of 14 pounds and 10 ounces on 25 September, or about one pound less than the weight measured forty-three days earlier. Also, Jordan gained only one pound in the forty-nine-days prior to 12 August 1994 (Table 2). These data indicate that he had serious health problems, especially during the forty-three-days prior to his cardiac arrest.

Jordan's babysitter and mother reported that Jordan had a cold during the week prior to his cardiac arrest and had a runny nose for most of the week. The babysitter noticed that Jordan's nose and the part underneath his nose and over his upper lip looked sore. Jordan's mother also stated that the baby had discharge coming out of his left eye due to infection. Also, Jordan had a reddish-purple area near his left eye as a result of the infection. The mother said that the baby was fussy on the night prior to his cardiac arrest. Furthermore, the baby had a mark (1/8 x 2 inch) on his left jaw and cheek as a result of a burn from an iron. This incident happened by accident at the babysitter's house about two weeks prior to Jordan's cardiac arrest [3-6].

**Table 1. Jordan's immunization history**

| Date       | Vaccine type  |
|------------|---|
| 01/09/1994 | Hepatitis B   |
| 02/07/1994 | Hepatitis B   |
| 03/07/1994 | Diphtheria/Tetanus/Pertussis (DTP)<br>Polio Hemophilus B conjugate  |
| 05/23/1994 | Diphtheria/Tetanus/Pertussis (DTP)<br>Polio, Hemophilus B conjugate |
| 06/20/1994 | Hepatitis B   |

**Table 2. Jordan's weight, height, and head circumference measurements**

| Date <sup>a</sup>     | Age (months) | Weight        | Height (inches) | Head Circumference (inches) |
|-----------------------|--------------|---------------|-----------------|-----------------------------|
| 01/24/94              | 0.5          | 6 lb & 15 oz  | 19.50           | 14.00                       |
| 03/04/94              | 2.0          | 10 lb & 2 oz  | 21.50           | ---                         |
| 05/16/94              | 4.4          | 12 lb & 2 oz  | 23.25           | 16.00                       |
| 06/20/94              | 5.5          | 14 lb & 10 oz | 25.00           | 16.75                       |
| 08/12/94              | 7.3          | 15 lb & 10 oz | 26.00           | 17.25                       |
| 09/25/94 <sup>b</sup> | 8.5          | 15 lb & 10 oz | 26.25           | 18.00                       |

<sup>a</sup>Jordan was born on January 6, 1994 and his birth weight was 5 lbs & 15 oz.

<sup>b</sup>Measurements were obtained at the time of autopsy on September 25, 1994 [2]

### 2. Jordan's cardiac arrest and treatment received

#### 2.1 Description of events before and after Bryant found the baby with cardiac arrest

Jordan's babysitter left Jordan asleep in his crib upstairs at about 11:00 PM on 24 September 1994, after his mother and Bryant arrived home at about 10:30 PM. Jordan's mother left the house at about 0005 on 25 September leaving Jordan sleeping in his crib and Bryant and her 4-year-old son in the house. Prior to leaving the house, Jordan's mother checked on him. She saw him sleeping in his bed and did not notice anything unusual [3,7].

Jordan's mother returned home at about 0200 on 25 September and found her four-year-old son and Bryant on the sofa downstairs. Her son was sleeping and Bryant was a half asleep. At about 0240, Bryant went upstairs to check on Jordan and found him unconscious and lifeless in his crib. He was not breathing. Bryant carried the baby downstairs and yelled to the baby's mother to call 911. He put the baby on a chair and he did chest compressions [8].

Officer John Wettlafer was the first responder to arrive at the house. He arrived at 0254 and saw Jordan was on a kitchen chair and that Bryant was doing CPR. Wettlafer put the baby on the floor and continued CPR [5,8,9]. Table 3 contains a list of events and times concerning Jordan's cardiac arrest incident.

#### 2.2 Examination of Jordan by the paramedics and treatment given

At 0256 on September 25th, two paramedics arrived at the scene and saw officer John Wettlafer performing CPR on Jordan. One of the paramedics took over the CPR. The baby was lifeless, asystolic, and without respiration and pulse. He was

unresponsive to all stimuli. His lips were cyanotic and his left eye was reddened and cyanotic.

The paramedics intubated the child and placed an intraosseous line (IO) in his upper tibia on the right leg. Initially, epinephrine was administered down the endotracheal tube and then epinephrine was administered via the IO line [5,6,10]. Treatments given to the baby by the paramedics and in the hospital are listed in Table 4.

The paramedics examined Jordan and observed eleven brownish/purplish circular marks on his lower chest and upper abdomen, which were approximately 1/8 of an inch in diameter. Jordan's mother stated that she observed some bruising on the chest wall resulting from Bryant's attempts to perform CPR. The baby was transported by ambulance to Lancaster General Hospital at 0311 (Table 3). The paramedics also performed CPR on their way to the hospital but were unable to revive the baby [5,6].

**Table 3. Timetable of events occurring prior to Jordan's cardiac arrest until the time of the autopsy**

| Date/Time          | Description of Events   |
|--------------------|---|
| 09/24/1994<br>2300 | Jordan's babysitter left Jordan sleeping in his bed after his mother and Bryant arrived home.                           |
| 09/24/1994<br>0005 | Jordan's mother left home leaving the baby sleeping in his bed and Bryant in the apartment watching her 4-year-old son. |
| 0200               | Jordan's mother returned home.  |
| 0240               | Bryant checked Jordan in his bed and found him lifeless and not breathing.  |
| 0245               | Jordan's mother called 911 asking for help.   |
| 0248               | Emergency Teams left the station.   |
| 0254               | Officer John Wettlafer arrived at the scene.  |
| 0256               | Emergency Teams arrived at the scene.   |
| 0311               | Jordan was transported to the hospital by an ambulance.   |
| 0315               | Jordan arrived at the hospital.   |
| 0340               | Jordan was pronounced dead in the hospital.   |
| 0800               | Dr. Wayne K. Ross started the autopsy on Jordan's body.   |

**Table 4. Treatment given to Jordan on 25 September following his cardiac arrest**

| Time      | Treatment   |
|-----------|---|
| 0258-0340 | 10% O <sub>2</sub>  |
| 0305      | 2 cc 1/1000 epinephrine given via ET  |
| 0311      | 1.0 cc atropine   |
| 0312      | 1.0 cc 1/10,000 epinephrine intraosseous (I.O);<br>1.0 cc atropine; 1.0 cc 1/10,000 epinephrine (I.O) |
| 0258-0315 | 150 ml of fluid IV  |
| 0315-0340 | repeated doses of adrenaline (I.O) atropine,<br>sodium bicarbonate, 250 ml of fluid IV                |

### 2.3 Examination of Jordan at the Lancaster General Hospital and treatment given

Jordan was examined in the hospital by the emergency physician and appeared lifeless. The baby was intubated and bagged via air bag valve mask. He had full and equal bilateral breath sounds on both sides. His pupils were dilated and unresponsive to light. CPR was also performed in the hospital [6].

The emergency physician observed an erythematous mark (1/2 x 5 cm) on the baby's left cheek. This was a burn mark caused by an iron about two weeks prior to the baby's cardiac arrest as stated by Jordan's mother. Furthermore, the physician noted annular bruises on the lower anterior chest wall and upper abdominal wall. These bruises were about a half centimeter (1/5 inch) in diameter and of various sizes, some smaller than others. Jordan's mother stated that these bruises were from attempts by Bryant to do CPR. The physician did not see any obvious deformity to the extremities and other injuries on the baby's body [6].

The treatment given to Jordan in the hospital consisted of CPR, bag valve aeration of the lungs through the endotracheal tube, adrenaline, bicarbonate, atropine, and IV fluid (Table 4). Repeated doses of adrenaline were given at increasing strength through the intraosseous line, with no response. The total IV fluid given to the baby by the paramedics and the hospital was 400 ml. Bicarbonate was also administered as well as atropine, but despite all these medications, the baby remained asystolic. Resuscitative efforts were stopped, and the baby was pronounced dead at 0340 on 25 September 1994. The medical examiner performed an autopsy on Jordan at about 0800 on 25 September 1994.

### 3. Review of The Medical Examiner's autopsy and pathology report, and interpretation of results

Jordan Anthony Shenk was pronounced dead at 0340 on September 25, 1994 in the Lancaster General Hospital. Dr. Wayne K. Ross, forensic pathologist of Lancaster County, Pennsylvania performed the autopsy at about 0800 [Autopsy No: A94-347 (LC94-265)]. The objectives of Dr. Ross's autopsy were to identify the cause and the manner of death. Assistant District Attorney Randy Miller, Officer Roland L. Buch, and Detective Allen W. Leed from the Manheim Township Police Department were present with Dr. Ross and observed the autopsy.

Prior to autopsy, full-body x-rays were performed on Jordan, showing no evidence of acute or healing trauma. Dr. Boal, pediatric radiologist, also reviewed these x-rays and did not observe any significant abnormalities. In addition, Dr. Ross examined Jordan's body and observed no evidence of injuries caused by trauma in the scalp, mouth, back, and upper and lower extremities [2].

Dr. Ross took tissue samples from selected organs and lesions at the time of autopsy. He submitted them to Harrisburg Hospital to process them for histological evaluation. Dr. Ross examined the H & E stained tissue sections of the samples that he received from Harrisburg Hospital at a later date. He found significant abnormalities in several organs as described below in Section 3.1-9 [2].

Dr. Ross went to Manheim Township Police Department on 25 September after he finished his autopsy on Jordan and met with officers and Detectives. He informed them that Jordan died due to complications from blunt trauma to the chest and abdomen and that the manner of death was homicide. He also provided the same information to Mr. Miller in the district attorney's office [9,11,12].

Dr. Ross stated in his autopsy report [2], “After autopsy and review of the history, it is my opinion that the cause of death is complications of blunt chest and abdominal trauma. The manner of death is homicide. The blunt chest and abdominal trauma is due to a severe, repetitive beating. Multiple bruises are seen on/in the skin. In addition, multiple hemorrhages on the liver, in the liver, in the gallbladder, and in the mesentery of the bowel as well as in the outer portions of the bowel itself. The beating is most probably due to being repetitively hit with knuckles from a fist, but could be from holding the abdomen with severe pressure, kicking with toes, or the beatings from the hands.”

I am very surprised to learn that Dr. Ross declared the cause and the manner of death in this case prior to examining the tissues microscopically to see if Jordan was suffering from acute and/or chronic illnesses that led to his cardiac arrest and bleedings. In addition, he did not perform differential diagnosis to consider all possible causes of the bleedings and inflammation in tissues observed in this case.

My review of Dr. Ross’ autopsy and pathology report and other medical evidence pertinent to this case revealed that Jordan suffered from chronic and acute health problems that led to cardiac arrest and bleedings. He had serious brain disease (spongiosis of the cerebral cortex and white matter and focal Purkinje cell dropout in the cerebellum); aspiration pneumonitis; sepsis; inflammation of the liver, gallbladder, and the mesentery; thymus atrophy; and weight loss [2].

Dr. Ross examined Jordan’s H & E stained tissue sections microscopically and found brain spongiosis; aspiration pneumonitis; and inflammation of the liver, mesentery, and gallbladder [2]. However, he did not consider these serious illnesses in his evaluation of the cause of bleedings and death. In addition, Dr. Ross testified in court in October of 1994 and May of 1995 concerning Jordan’s case, but did not reveal to the court that the baby had brain prognosis, Purkinje cell dropout in the cerebellum, and aspiration pneumonitis [11,12]. These are the factual causes of death.

My investigation of the medical literature revealed that babies who had propionic acidemia and other metabolic problems involving branched amino acids developed brain spongiosis, aspiration pneumonia, thymus atrophy, sepsis, and weight loss, as did Jordan. These babies also had severe thrombocytopenia that led to bleedings. Also, severe liver inflammation usually caused reduction in the synthesis of clotting factors, with enhanced bleedings, as also happened in Jordan’s case.

Furthermore, the literature reveals that bruises on skin and damage to lungs, liver, and gastrointestinal tract have been reported in children and adults who received cardiac pulmonary resuscitation (CPR). Bryant, a police officer, and the paramedics performed CPR on Jordan. Jordan’s mother stated that Bryant caused bruises on her son’s chest when he performed CPR. The medical evidence below shows that CPR was the likely cause of the bruises and marks observed on Jordan’s lower chest and upper abdomen.

The paramedics observed eleven bruises on the lower chest and upper abdomen. The largest one was 1/8 inch in diameter. However, Dr. Ross counted sixteen bruises in these areas, with the largest being 1/2 inch in diameter—an increase of 400%. Also, five new bruises appeared on Jordan’ lower chest area following the time that the baby was transported from his house

by the paramedics. Dr. Ross did not consider these medical data in his evaluation, which clearly indicate that CPR and medical intervention were the probable causes of the bruises observed on the baby’s skin on the chest and abdomen.

Below are detailed descriptions of Dr. Ross’ autopsy and histopathology findings and the pertinent medical evidence. These data clearly (1) invalidate Dr. Ross’s conclusions that Jordan died as a result of blunt trauma to the chest and abdomen and that the manner of death was homicide; (2) show that Dr. Ross did not follow standard medical procedures to perform the required medical tests and to interpret medical data correctly; (3) show that Dr. Ross rushed to judgment in accusing Bryant in Jordan’s death. Furthermore, these data explain the factual causes of death and bleedings in this case.

### 3.1. Spongiosis of the cerebrum, Purkinje cell dropout in cerebellum, and brain edema

Dr. Ross examined Jordan’s brain grossly at the time of autopsy on September 25th and observed severe cerebral edema. The weight of the brain was 900 gm. His microscopic examination of the H & E stained sections of the brain at a later date revealed serious chronic brain lesions (spongiosis of the cerebral cortex and white matter, and focal Purkinje cell dropout in the cerebellum) [2]. These lesions indicate a serious, chronic neurological problem. However, Dr. Ross did not consider these lesions in his evaluation of the cause of death. In addition, he did not reveal these crucial medical findings in court when he testified in October of 1994 and May of 1995 [11,12]. Below are the questions that were presented to Dr. Ross in court by the defense lawyer, and Dr. Ross’s answers.

Defense lawyer: Did you notice anything unusual about anything else within the body?

Dr. Ross. No [11:42].

Defense lawyer: Dr. Ross, have you prepared an autopsy report in this matter?

Dr. Ross. Yes.

Defense lawyer: And have you testified from that report today?

Dr. Ross: Yes, I have [11:51].

Dr. Ross’ answers in court clearly contradict the histology findings described in his autopsy report: that Jordan had spongiosis of the cerebrum and focal Purkinje cell dropout in the cerebellum [2:10]. These lesions indicate serious metabolic and neurological problems that led to weight loss; thymus atrophy; aspiration pneumonitis; bleedings in the lungs and other tissues; sepsis; inflammation of the liver, gallbladder, and mesentery; and cardiac arrest.

Spongiosis of the brain, pulmonary infection, atrophy of the thymus, thrombocytopenia, and loss of weight have been described in children who suffered from propionic acidemia and other metabolic diseases as shown by the published medical studies described below.

Propionic acidemia (PA) is a genetic disorder of branched amino acid and odd-chain fatty acid metabolism. It is caused by an inhibition of the activity of propionyl coenzyme A carboxylase and the accumulation of propionyl-CoA. The main sources of propionyl-CoA are isoleucine, valine, methionine, and cho-

lesterol. Odd-chain and branched odd-chain fatty acids are also significant contributors. The propionyl-CoA is known to inhibit several key enzymes (such as pyruvate dehydrogenase) as well as fatty acid oxidation. It also inhibits N-acetylglutamate, which might account for impaired ureogenesis and hyperammonia [13,14,15].

The clinical features of PA typically begin shortly after birth, but in some cases present in young adulthood. The symptoms and lesions of this disorder may include dehydration, lethargy, nausea, vomiting, generalized hypotonia and muscle weakness, thrombocytopenia, neutropenia, intercurrent infections, and neurological problems [14–17]. The symptoms of PA with onset in late infancy or early childhood are usually identical to those of the neonatal-onset disorder.

The onset of acute PA may be precipitated by upper respiratory tract or gastrointestinal infections, or possibly severe constipation. Many children who survive severe or prolonged metabolic disturbances sustain brain damage. Seizures are common in patients with propionic acidemia [16]. In neonates and older children, the neuropathologic findings in propionic acidemia may include spongiosis of the cerebrum (white matter and/or gray matter) and Purkinje cell dropout in the cerebellar cortex [13–16,18,19]. Dr. Ross observed these serious lesions in Jordan's brain.

The diagnosis of PA is readily achieved by urine gas chromatography/mass spectrometry (GC/MS), by tandem mass spectrometry (MS/MS), or by enzyme analysis of fibroblasts. Dr. Ross did not perform these clinical tests to exclude propionic acidemia from his findings.

The following are descriptions of published studies describing cases of children who suffered from PA. These children developed symptoms and lesions in brain and other tissues identical to those observed in Jordan's case. Some of these children also had bleedings resulting from thrombocytopenia.

1) Asconape et al. described pathologic changes in the brain of a five-and-a-half-month-old male infant who died of PA. These changes included spongy degeneration of the white matter and marked diffuse atrophy with early loss of the external granular layer of the cerebellum [19].

2) Feliz, Witt, and Harris reported a case of a 4-year-old girl with PA from infancy who died from multiorganism bronchopneumonia after a protracted clinical course. She was neutropenic (white blood cell count  $0.9 \times 10^3/\mu\text{L}$ ; normal range,  $4.3\text{--}10.8 \times 10^3/\mu\text{L}$ ) and thrombocytopenic (platelet count  $0.9 \times 10^3/\mu\text{L}$ ; normal range,  $150\text{--}300 \times 10^3/\mu\text{L}$ ). Their postmortem neuropathology examination revealed widespread gray matter vacuolization. The cerebellar cortex showed a patchy dropout of Purkinje cells and a proliferation of Bergmann glia [14].

3) Steinman et al. found spongiform changes in the white matter of the cerebrum in an infant who died as result of PA [18].

4) Ozand et al. studied twenty-five cases of neonates and infants with PA, and most of these children had acute or chronic encephalopathy with or without acidosis [15]. In fourteen patients, PA presented acutely with acidosis, hyperammonemia, and thrombocytopenia. Five infants presented encephalopathy

with acidosis, and four infants showed a progressive encephalopathy without acidosis. In addition, three infants showed immune deficiency. The prognosis in PA remained grave despite rigorous treatment. Only seven of the 25 PA patients recovered to have a normal lifestyle, while eight patients expired [15].

The clinical picture of PA in these patients included severe hypotonia with diminished reflexes and vomiting. During acute metabolic crisis, PA usually, but not always, causes acidosis, accumulation of ketone bodies and lactic acid in body fluids, hyperammonemia in the range of  $200\text{--}800 \mu\text{M}$ , and at times hypoglycemia. Characteristically, organic acids and organic acid esters are excreted in the urine [15].

Severe thrombocytopenia is the hallmark of the metabolic crisis. Severe and sustained thrombocytopenia is almost pathognomonic of propionic acidemia. Intracranial hemorrhage was observed in four patients with PA, and in three of them the intracranial bleeding caused death. Thrombocytopenia is encountered in other organic acidurias. Isovaleric acidemia can also cause severe thrombocytopenia during a metabolic event [15].

The immune suppressive action of PA can be so severe that the thymus gland may become atrophic. A thymic biopsy was obtained from a patient with PA, which showed depletion of lymphocytes from thymic cortex, with no differentiation between cortex and medulla, and with numerous epithelioid cells. Also the numbers of Hassall's corpuscles are reduced [15].

Repeated infections might be the only presenting symptom of the disease, with other signs of PA being less apparent or initially absent. The acute attacks are accompanied by mild neutropenia and by recurrent severe infections. Postmortem blood culture of one patient grew *Klebsiella pneumoniae* and coagulase-positive *Staphylococcus aureus*. In a second patient, *Pseudomonas aeruginosa* and *Staphylococcus aureus* were isolated from blood culture and her symptoms were attributed to sepsis. In a third patient, blood culture grew coagulase-negative *Staphylococcus aureus* [15].

5) Hamilton et al. reported neuropathologic findings in two patients with PA who were diagnosed in infancy and survived 35 months and 9 years, respectively [13]. Examination of the brain of the 35-month-old boy showed vascular and parenchymal mineralization, focal pallor and spongy change, and foci of acute neuronal injury.

The 9-year-old girl was in good metabolic control when she died. She was found at autopsy to have acute hemorrhagic lesions in the caudate, putamen, and globus pallidus bilaterally and in the left ventral thalamus. There was also focal neuronal loss. Vascular proliferation and swollen endothelial cells were seen in the basal ganglia, thalamus and substantia nigra of the brain. Electron microscopy showed swelling of endothelial cells with viable adjacent brain parenchyma. The endothelial changes suggest a breakdown of the blood-brain barrier [13].

6) Nyhan et al. evaluated two children with propionic acidemia who suffered from prominent neurological disease without the life-threatening episodes of ketoacidosis. Their prominent features were hypotonia followed by spastic quadriplegia, and both had seizures. One patient was mildly mentally retarded but

grew normally physically. The other had profound mental retardation and failure to thrive [20].

7) Akman et al. evaluated a neonate with propionic acidemia who had prominent neurological problems but without ketoacidosis. The baby had a serum ammonia level of 3,500 micrograms/dL. He developed *Candida albicans*, peritonitis, and sepsis, and died of cardiorespiratory failure [21].

### 3.2 Jordan's weight loss.

At autopsy on 25 September 1994, Dr. Ross reported Jordan's weight as 15 lb, 10 oz. However, Jordan's weight on 12 August was also 15 lb, 10 oz. These data indicate that the baby did not gain any weight during the 43-day-prior to his cardiac arrest. Jordan's clinical chart showed that he was given about 410 ml of fluid IV, which weighs about a pound, by the paramedics and in the hospital following his cardiac arrest on 25 September. These data indicate that the baby lost about one pound during the forty-three days prior to his cardiac arrest.

The average body weight and the 10th percentile weight for infants at nine months of age in the USA were reported as 19.6 lb (8.91 kg) and 17.82 (8.1 kg), respectively, [22]. It seems that Jordan's weight at the time of autopsy was about 2 to 3 lb less than babies of that age who have body weight at the 10th percentile. These data indicate that Jordan was suffering from serious weight loss.

### 3.3 Jordan's thymus atrophy

Dr. Ross stated that Jordan's thymus weight was 13 g and that its external and sectioned surfaces were unremarkable [2]. The average thymus weight (g) in a white male infant at six to nine months of age was found to be 25 g [22]. Jordan's age at the time of autopsy was eight-and-a-half months. It seems that Jordan's thymus weight was about 52% of normal. In addition, at Jordan's age, thymus weight is usually equal to the spleen weight. Jordan's spleen weight was 33 grams. These data indicate that Jordan suffered from thymus atrophy and contradict Dr. Ross's claim that Jordan had normal thymus size.

Furthermore, Dr. Ross did not take tissue samples from Jordan's thymus for histological evaluation. His conclusion that Jordan's thymus was normal is not scientifically valid. Reportedly, babies who suffered from spongiosis of the brain and propionic acidemia (PA) also had thymus atrophy, as observed in Jordan's case. A thymus biopsy obtained from a patient with PA showed depletion of lymphocytes from the thymic cortex with no differentiation between cortex and medulla. It also showed numerous epithelioid cells and clear reduction in the numbers of Hassall's corpuscles [15].

### 3.4 Jordan's aspiration pneumonitis

Dr. Ross examined Jordan's lungs grossly at the time of the autopsy on 25 September 1994. He stated that the lung parenchyma showed diffuse and severe pulmonary edema and congestion. He also observed petechial hemorrhages in lung parenchyma and hemorrhages in the alveoli in multiple areas. The total lung weight was 128 grams [2].

Furthermore, Dr. Ross examined the H & E stained tissue sections of Jordan's lungs microscopically at a later date. He found that the baby had aspiration pneumonitis, bleedings, and pulmonary edema [2:10]. Dr. Ross testified in court in October of 1994 and May of 1995 concerning the cause of Jordan's death. However, he did not reveal to the court that the baby had aspiration pneumonitis [11,12]. Below are the questions that were presented to Dr. Ross in court by the defense lawyer and his answers [11:42–51].

Defense lawyer: Did you notice anything unusual about anything else within the body?

Dr. Ross: No.

Defense Lawyer: Lungs?

Dr. Ross: There is bleeding inside the lungs.

Defense lawyer: Dr. Ross, have you prepared an autopsy report in this matter?

Dr. Ross: Yes.

Defense lawyer: And have you testified from that report today?

Dr. Ross: Yes, I have.

Aspiration pneumonitis is mentioned in Dr. Ross' autopsy report [2:10] but he did not mention it in court. It is usually caused by a foreign substance (food and/or secretions) and microorganisms entering the lung causing inflammation. The severity of the lung's injury after an aspiration incident is determined by pH of the aspirated material and presence of pathogenic bacteria. Very low pH or extremely high pH will cause a significant inflammatory response. Children at highest risk are children with poor airway reflexes or gastroesophageal reflux, or both [23:1213]. Jordan had serious neurological problems that caused poor airway and gastroesophageal reflexes, namely spongiosis of the cerebral cortex and white matter and focal Purkinje cell dropout in the cerebellum [2:10].

Inflammation of the lungs due to the aspiration of foreign substance and bacteria can explain Jordan's bleedings and edema of the lungs. *Streptococcus pneumoniae* is normally present in the nasopharynx. Frequently, this pneumonia follows viral infections of the upper respiratory tract. The bronchial secretions stimulated by the viral infection provide a hospitable environment for the proliferation of *S. pneumoniae* organisms. The thin, watery secretions carry the organisms into the alveoli, thereby initiating an inflammatory response [24].

Jordan's mother and his babysitter stated that Jordan had a cold during the week preceding his cardiac arrest. Jordan's cold and his neurological problems are important factors in causing the inflammation of the lungs by bacteria. In the earliest stage of pneumococcal pneumonia, protein-rich edema fluid containing numerous organisms usually fills the alveoli. The lesions include marked congestion of the capillaries, massive infiltration of polymorphonuclear leukocytes, and intra-alveolar hemorrhage. On gross examination, the lungs of patients who die of streptococcal pneumonia are heavy and display bloody edema [24:567,569]. Dr. Ross observed these lesions in Jordan's lungs [2].

Aspiration pneumonitis can also be caused by microorganisms other than *S. pneumoniae*, such as fusobacteria and other *Bacteroides* species. Pneumonia due to anaerobes is an especially likely outcome if the aspirated material is large in volume

or contains virulent components of the anaerobic microbial flora or foreign bodies, such as aspirated food or necrotic tissue. Impairment of the cough reflex increases the risk of pneumonia, as does mucociliary or alveolar macrophage dysfunction [24:569;25:1776]. Schmidt reviewed the data of 98 children for foreign body aspiration. In this data review, 78% of the children were younger than 2 years. Predominant clinical features were fever (46%), pneumonia (39%), and coughing (29%) [26].

I am very surprised to learn that Dr. Ross used a theory to explain the causes of bleeding in Jordan's lungs rather than citing aspiration pneumonitis. Dr. Ross stated that when you hit the abdomen or the chest, you produce various waves—shock and shear waves, which were responsible for Jordan's bleeding in the lungs [12:95]. It is very obvious to me that aspiration pneumonitis was the actual cause of the bleeding and edema.

Furthermore, aspiration pneumonitis can also cause asphyxia, as was described in Jordan's case. Delmonte and Capelozzi observed signs of asphyxia in thirty-five victims who died as a result of aspiration pneumonitis. Autopsies revealed that the lungs of these victims had congestion, septal hemorrhage, and foreign bodies, as observed in Jordan [27].

### 3.5. Causes of the marks observed on Jordan's lower chest and upper abdomen

Dr. Ross examined Jordan's body at about 0800 on 25 September 1994. He observed sixteen brownish/purplish circular contusions of various sizes (1/8-1/2 inch in diameter) on the lower anterior chest wall and upper abdomen [2]. He alleged that these were bruises caused by repetitive beatings of the chest and abdomen by knuckles and/or fists or by kicking [11:9]. He stated that the injuries observed in Jordan were equivalent to those caused by the impact of a car moving at 20-26 miles per hour.

Furthermore, Dr. Ross excluded cardiopulmonary resuscitation (CPR) as the cause of injuries. He stated that Jordan's injuries were severe and showed inflammatory reactions as indicated by the presences of white blood cells. Dr. Ross concluded that Jordan died within hours after receiving the injuries, and this allowed enough time to cause an inflammatory response [11,12].

My review of the medical evidence in this case revealed that Dr. Ross based his conclusions on a theory and not on medical facts. The following is a list of medical facts that invalidate Dr. Ross's conclusions. They also clearly show that CPR was the likely cause of the marks observed on Jordan's lower chest and upper abdomen.

1) The paramedics examined Jordan prior to transporting him to Lancaster General Hospital at 0305 on 25 September and observed only eleven brownish/purplish circular marks on his lower chest and upper abdomen. The sizes of the marks were approximately 1/8 inch in diameter. In addition, the physician who examined Jordan at the hospital's emergency room estimated the sizes of the marks to be about a half centimeter in diameter (1/5 inch). However, Dr. Ross stated that he found 16 bruises and that the sizes of these bruises were 1/8-1/12 inch in diameter.

These observations indicate that five of the sixteen bruises observed by Dr. Ross occurred after the baby was transported to the hospital. Also, the sizes of the bruises increased by 400% from the time the paramedics transported the baby at 0305 until the time of autopsy at 0800.

2) Dr. Ross examined the H & E stained tissue sections of the bruises on Jordan's chest, including muscle, and found fresh hemorrhage without the presence of a significant white blood cell infiltration. These observations contradict Dr. Ross's conclusion that the bruises on the chest occurred when the baby was alive because the injury triggered inflammatory response.

3) Dr. Ross examined the H & E stained tissue section from the marks on Jordan's upper abdomen and observed only congested blood vessels with no obvious hemorrhage or inflammatory reactions. These findings contradict Dr. Ross statements that he found bruises on the upper abdomen, that these marks were caused by hitting the baby with a strong force, and that the marks occurred prior to the baby's death.

4) Jordan's mother stated that the bruises on Jordan's chest were caused by Bryant's attempts to do CPR.

5) Jordan had brain spongiosis and aspiration pneumonitis. Severe thrombocytopenia has been reported in children with sepsis and brain spongiosis. Patients with thrombocytopenia are susceptible to bruising and bleedings.

6) Various types of injuries in the chest and abdomen areas have been reported in patients receiving CPR. These injuries are more severe than those reported in Jordan's case. For example, complications caused by cardiopulmonary resuscitation (CPR) were reviewed by analyzing the autopsy protocols of 25 patients who died after receiving standard CPR. Rib fractures were detected in 28%, and sternal fractures in 16% [28].

In addition, Sokolove reported a case of a 61-year-old woman in whom cardiopulmonary resuscitation (CPR) was unsuccessful. While the patient was initially resuscitated from the primary cardiac arrest, with evidence of neurologic recovery, she ultimately succumbed to injuries resulting directly from CPR. Autopsy revealed multiple rib fractures, a sternal fracture, pulmonary laceration, and cardiac rupture [29]. Also, costochondritis resulting from resuscitation was described in another patient [30].

### 3.6 Causes of the inflammation and bleedings in Jordan's liver

Dr. Ross examined Jordan's liver at autopsy on 25 September 1994 and observed subcapsular hemorrhage, bruises, and bleedings in multiple areas of the left and right lobes of the liver. In addition, he examined the H & E stained tissue sections of the liver at a later date and found edema, fresh hemorrhage, and blood clotting. In addition, the entire liver was infiltrated primarily with polymorphonuclear cells, but macrophages also noted [2].

Dr. Ross concluded that the injuries observed in Jordan's liver were caused by blunt trauma to the abdomen. He also excluded CPR as a factor in causing these injuries. Furthermore,

Dr. Ross stated that the type of injuries observed in Jordan's liver usually occurred in people hit by a car moving at approximately 26 miles per hour [12:236]. I found Dr. Ross' conclusions invalid, based on the following medical facts:

1) The presence of the polymorphonuclear neutrophils (PMN) and macrophages throughout Jordan's liver indicates an inflammation that started about 24 hours prior to death. PMN usually enter the inflammatory site in 6 to 12 hours after the initial injury, while macrophages enter the site at the later time, usually after 24 hours. [23:220;24:49].

2) Bacterial infection was the likely cause of the inflammation observed in Jordan's liver, because he had aspiration pneumonitis. Bacteria can travel via blood to infect the liver and other organs. In addition, Jordan had thymus atrophy, which predisposed him to infection.

3) The bleedings and bruises observed in Jordan's liver likely resulted from the synergistic actions of the following factors:

a. Jordan had sepsis and brain spongiosis, which were probably caused by propionic acidemia. Reportedly, babies who suffered from these illnesses also developed thrombocytopenia and bleedings;

b. Inflammation of the liver usually causes deficiency of clotting factors. Blood clotting factors, except factor VIII, are made exclusively in hepatocytes. The serum half-lives of the clotting factors range from 6 hours for factor VII to 5 days for fibrinogen [25:1714];

c. Severe liver damage has been reported in people receiving CPR. For example, Adler et al. treated two patients with laceration of the liver precipitated by cardiopulmonary resuscitation (CPR) and recognized ante mortem [31]. These patients had excruciating abdominal pain and bled massively, which led to the development of hypovolemic shock. The authors stated that when severe abdominal pain associated with hypovolemic shock occurs in a patient who has received CPR, hepatic laceration, hematoma, or both, should be considered [31].

In addition, Druwe et al. reported a patient with an acute myocardial infarction who had to be resuscitated due to recurring ventricular fibrillation. Fourteen hours later, clinical signs of hemoperitoneum developed and the diagnosis of liver rupture was made [32]. Also Reinartz reported a case of a combined manifold rupture of the liver and spleen occurring after the individual received cardiopulmonary resuscitation [33].

4) Dr. Ross examined the H & E stained tissue section from the marks on the skin in the abdominal area and observed only congested blood vessels with no obvious hemorrhage or inflammatory reactions. These findings contradict Dr. Ross's statement that the baby was hit by a strong force equivalent to a car driving 26 miles per hour, or by any significant force that causes internal bleedings.

### 3.7. Causes of the inflammation and necrosis in Jordan's gallbladder

Dr. Ross examined Jordan's gallbladder at autopsy and observed hemorrhage in the perigallbladder region and on the wall inside the gallbladder. He also found early necrosis associated

with edema in the gallbladder wall. In addition, he examined the H & E stained tissue sections of the gallbladder microscopically and observed blood clot, autolysis of the epithelium, and subepithelial hemorrhages with early infiltration by polymorphonuclear cells. Dr. Ross concluded that the lesions in Jordan's gallbladder were caused by blunt trauma and that it occurred within 20 minutes to two hours prior to the baby's death. I found these conclusions by Dr. Ross's scientifically invalid.

It takes about three to four hours for the gallbladder necrosis to become apparent to the naked eye. In addition, the autolysis of epithelium occurred after the death of cells and the release of the lysosomal enzymes. The autolysis observed in this case occurred prior to the death of the child because the wall of the gallbladder was infiltrated by white blood cells. Dead cells usually evoke an inflammatory reaction that brings leukocytes to the area [23,24].

Furthermore, infection with microorganisms was the likely cause of the inflammation and the necrosis of Jordan's gallbladder, because the baby had aspiration pneumonitis and liver inflammation. Microorganisms can travel via blood to infect the gallbladder and other organs. The baby also had thymus atrophy, which predisposed him to infections. In addition, the bleeding in Jordan's gallbladder likely resulted from the synergistic actions of several factors, as described above in the liver section. Briefly, these include thrombocytopenia, sepsis, metabolic problems, and liver inflammation that led to reduction in the synthesis of clotting factors.

### 3.8 Causes of the bleedings in Jordan's intestine and mesentery

Dr. Ross examined Jordan's gastrointestinal tract and abdominal cavity at autopsy and observed fresh bleedings in multiple areas of the mesentery, pericolic region, serosa of small intestine, and ascending colon. He collected 200 ml of bloody fluid from the abdominal cavity. At a later date, Dr. Ross examined the H & E stained tissue sections of the mesentery and intestines and found a fresh hemorrhage with inflammation in the mesentery and fresh hemorrhage and edema in the wall of the ascending colon. The mesentery was infiltrated primarily with polymorphonuclear cells, but some macrophages were noted also. No significant abnormalities were observed in the small intestine.

Dr. Ross concluded that the injuries of the mesentery and the intestine in Jordan's case were caused by blunt trauma applied to the upper abdomen and the lower anterior chest. He also stated that the injuries observed in Jordan's case usually occur in people hit by a car moving at approximately a 26 miles per hour, and that these injuries cannot be caused by somebody performing CPR [12:236]. The following is a list of medical facts that invalidate Dr. Ross's conclusions concerning the cause of injuries observed in Jordan's intestine and mesentery.

1) The presence of the polymorphonuclear neutrophils (PMN) and macrophages in the mesenteric tissue indicates that Jordan's mesentery was inflamed and that the inflammation was about 24 hours old or older, as shown by the presence of macrophages. Macrophages usually enter the site of inflammation after 24 hours [23,24].

2) Bacterial infection was the likely cause of the inflammation of the mesentery in Jordan's case because he had aspiration pneumonitis and inflammation of the liver and gallbladder. Microorganisms can travel from infected organs via blood to the mesentery and other organs. Jordan's immune function was also depressed because of the thymus atrophy.

3) The bleedings in the mesentery and the intestines arose from the synergistic actions of several factors, as described in the liver section (3.6). These include thrombocytopenia, sepsis, liver inflammation, and CPR. Severe damage of gastrointestinal tract has been reported in people receiving CPR. For example, Aguilar reviewed 16 patients with documented gastric trauma resulting from cardiopulmonary resuscitation [34].

In addition, Waldman evaluated a case of a child in which conventional CPR was augmented with interposed abdominal compressions. In this case, intraperitoneal visceral injury was noticed in the form of blood within the stomach and small intestine and parenchymal hemorrhage within the pancreas [35]. In addition, McGrath reported a case of fatal gastrointestinal hemorrhage from gastric mucosal lacerations related to closed-chest cardiac compression of CPR [36]. Also, previous autopsy series suggest that gastroesophageal lacerations may occur in 12 percent of cases not surviving CPR [36].

Also, three cases of gastric trauma following cardiopulmonary resuscitation were reported by Hulewicz [37]. In two cases, full-thickness lacerations were observed in the gastric wall, resulting in pneumoperitoneum. In the third case, gastric hemorrhage resulting from gastric mucosal lacerations was described. Reiger et al. also reported two patients who developed gastric rupture after successful standard CPR [38]. Furthermore, McDonnell et al. observed at autopsy two patients in whom clinically significant gastrointestinal bleeding occurred from gastric mucosal tears following closed-chest cardiac massage [39].

In addition, Bedell and Fulton studied autopsy findings of 130 patients who died after attempted CPR. Twenty-one percent of these patients had at least one CPR-related complication. Patients resuscitated in the wards were more likely to have a complication than those treated in the intensive care unit. This suggests that more proficient technique in CPR may reduce morbidity from this procedure [40].

4) As previously stated in this report, Dr. Ross examined the H & E stained tissue section from the marks on the skin in the abdominal area, observing only congested blood vessels and no obvious hemorrhage or inflammatory reactions. These findings contradict Dr. Ross's statement that the baby was hit by a strong force equivalent to a car moving 26 miles per hour.

### 3.9 Likely causes of Jordan's petechial hemorrhages

Dr. Ross examined Jordan's body at autopsy and observed petechial hemorrhage at various sites of his body. These sites include upper and lower lips, gums, distal end of tongue, sclera, eyelids, mucosa of the trachea, and the thymus.

Petechiae in tissues have been observed in patients in whom cardiopulmonary resuscitation has reestablished blood flow. The increase in the blood pressure in small vessels already

damaged by hypoxia can result in the formation of petechiae [41,42]. Jordan had anoxia because he was not breathing. In addition to CPR, Jordan was given 410 mL of fluid, which increased his blood volume by 100%. Jordan's weight was about 7 kg. His total blood volume was about 420 mL (60 mL per Kg). Injecting a relatively large volume of fluid in leaky blood vessels can cause petechiae.

Maxeiner and Winkhofer analyzed 474 autopsies. Resuscitation efforts (RES) were done in 144 cases (31%). Nineteen percent of these victims presented with petechial bleedings, predominantly in the conjunctivae, compared to only 11% in the non-RES group. The analysis revealed an influence of the following factors in the development of petechial bleedings: cause of death, age of the person, and the body mass of the person [42].

Furthermore, the frequency and density of intrathoracic and subconjunctival petechiae were studied in 250 cases of sudden infant death syndrome (SIDS) and 69 controls. Intrathoracic petechiae were found significantly more frequently in the SIDS group (91.2% SIDS; 42% controls;  $p < 0.001$ ) and were present at a higher density ( $p < 0.001$ ). In addition, subpericardial and thymic petechiae were detected at high density in older SIDS infants [43].

### 4. Problems with the methodology Dr. Ross used in the investigation of this case

Dr. Wayne K. Ross performed the autopsy on Jordan's body on 25 September 1994 and concluded that he was killed by blunt trauma to the chest and abdomen and that the manner of the death was homicide. The medical evidence presented in Section 3 of this report clearly shows that Jordan died as a result of serious acute and chronic illnesses. Jordan suffered from spongiosis of the brain; aspiration pneumonitis; weight loss; thymus atrophy; inflammation of the liver, gallbladder, and mesentery; sepsis; and metabolic disease.

The objectives of Dr. Ross's investigation were to find the factual causes of death and bleedings and the manner of death. My investigation revealed that Dr. Ross did not achieve his objectives, because he did not follow standard medical procedures in investigating the causes of death. He also rushed to judgment in providing the cause and manner of death to the Manheim Township Police and the assistant district attorney [2, 9,11,12]. Furthermore, he did not reveal important clinical medical evidence to the court that explain the factual causes of death and bleedings [11,12]. The following is a list of medical facts that support my conclusions.

1) Dr. Ross overlooked Jordan's weight loss during the forty-three days prior to his cardiac arrest. The baby's weight on 12 August 1994 was 15 lb, 10 ounces. His weight on 25 September, following his injection with 410 ml of fluid (weighing about one pound) remained at 15 lb, 10 ounces. These data indicate that Jordan lost about one pound during the forty-three-day period and was probably suffering from chronic illness.

2) Dr. Ross examined Jordan's body at about 0800 on 25 September and observed sixteen brownish/purplish circular contusions at various sizes (1/8-1/2 inch in diameter) on the lower

anterior chest wall and upper abdomen. He assumed that these were present when the paramedics picked the baby up from his house and that they were bruises caused by blunt trauma inflicted intentionally prior to the baby's death. I found that Dr. Ross's assumptions are not supported by scientific facts. My conclusion is supported by the following medical facts:

a. The paramedics examined Jordan prior to transporting him to Lancaster General Hospital at 0305 and found only eleven brownish/purplish circular marks on his lower chest and upper abdomen. The size of the marks was approximately 1/8 inch in diameter. These data indicated that five bruises or marks of the sixteen reported by Dr. Ross were formed after the baby was transported by the paramedics. In addition, the size of the marks increased by 400% from the time the paramedics transported the baby at 0305 until the time of autopsy at 0800;

b. Dr. Ross examined the H & E stained tissue sections from the bruises and marks on Jordan's lower chest and upper abdomen. He did not find a significant white blood cell infiltration in either area. Also, he saw only congested blood vessels without hemorrhage in the abdominal marks. These observations contradict Dr. Ross's conclusions that the bruises in the chest area occurred while the baby was alive and that the baby had bruises on the upper abdomen.

c. Various types of injuries in the chest area have been reported in patients receiving cardiopulmonary resuscitation (CPR), and Jordan's mother stated that the bruises on the baby's chest were caused by CPR;

d. Jordan had brain spongiosis and aspiration pneumonitis. Thrombocytopenia has been reported in children who have sepsis and brain spongiosis. Patients with thrombocytopenia are susceptible to bruising and bleeding.

3) Dr. Ross examined the H and E stained tissue sections of Jordan's brain microscopically and found serious chronic brain disease. Jordan had spongiosis of the cerebral cortex and white matter, and focal Purkinje cell dropout in the cerebellum; but Dr. Ross did not take this important discovery into account in his evaluation of this case. Furthermore, he did not reveal these findings to the court when he testified in October of 1994 and May of 1995. Reportedly, these lesions usually occur in babies with propionic acidemia and other metabolic problems associated with branch amino acids. Propionic acidemia also causes aspiration pneumonitis, thymus atrophy, sepsis, bleedings, and weight loss, as observed in Jordan's case.

4) Dr. Ross reported that Jordan's thymus weight was 13 grams. Jordan was eight-and-a-half-months old at the time of death/autopsy. The average thymus weight (g) in a white, infant male at six to nine months of age is 25 g; thus, Jordan's thymus weight was about 52% of normal. These data indicate that Jordan's thymus was atrophic. Reportedly, babies who had brain spongiosis and propionic acidemia also suffered from thymus atrophy and serious infections, as observed in Jordan's case.

5) Dr. Ross examined the H & E stained tissue sections of Jordan's lungs microscopically and found aspiration pneumonitis. However, he did not take this important discovery into account in his evaluation of the case. He also did not reveal the information in court when he testified in October of 1994 and May of 1995.

Aspiration pneumonitis is caused by a foreign substance (food and/or secretions) and microorganisms entering the lung and causing inflammation. It occurs in children with poor airway reflexes or gastroesophageal reflux, or both. Jordan had serious neurological problems that led to poor airway and gastroesophageal reflexes, namely spongiosis of the cerebral cortex and white matter and focal Purkinje cell dropout in the cerebellum.

Inflammation of the lungs due to the aspiration of foreign substance and bacteria can explain the bleedings and edema observed in Jordan's lungs. In the earliest stage of pneumococcal pneumonia, protein-rich edema containing numerous organisms usually fills the alveoli. Marked congestion of the capillaries is followed by a massive outpouring of polymorphonuclear leukocytes, accompanied by intra-alveolar hemorrhage. On gross examination, the lungs of patients who die of streptococcal pneumonia are heavy and display bloody edema.

6) Dr. Ross examined the H & E stained tissue sections of Jordan's liver and found that the entire liver was infiltrated primarily with polymorphonuclear cells (PMN), but some macrophages also. The presence of the PMN and macrophages indicates that Jordan's liver inflammation was about 24 hours old or older. The PMN usually enters the inflammatory site in 6 to 12 hours after initial injury, and macrophages enter the site later, usually after 24 hours. Bacteria were the likely cause of the liver inflammation. Dr. Ross did not take blood samples or abdominal fluid to culture them for bacterial growth. Jordan had aspiration pneumonitis, and bacteria can travel via blood and infect other organs.

7) Dr. Ross examined Jordan's gallbladder at autopsy and found early necrosis associated with edema in the gallbladder wall. He also examined the H & E stained tissue sections of the gallbladder and found blood clotting, autolysis of epithelium, and subepithelial hemorrhages with early infiltration by polymorphonuclear cells. It takes about three to four hours for the necrosis in gallbladder to become apparent to the naked eye. Enzymes are activated and released from the lysosomes of the dead cells, causing autolysis.

The autolysis observed in Jordan's case occurred prior to death, because the wall of the gallbladder was infiltrated by white blood cells. Dead cells evoke an inflammatory reaction that brings leukocytes to the area. The gallbladder inflammation and necrosis were likely caused by microorganisms, because the baby had aspiration pneumonitis and liver inflammation. Microorganisms can travel via blood to infect gallbladder and other organs via blood. Jordan's thymus atrophy also predisposed him to infection.

8) Dr. Ross examined the H & E stained tissue sections of the mesentery and the intestines and observed fresh hemorrhage with inflammation in the mesentery. The mesentery was infiltrated with polymorphonuclear cells (PMN) and macrophages. The presence of the PMN and macrophages indicates that the inflammation in Jordan's mesentery was about 24 hours old or older, as explained above. In addition, the inflammation was likely caused by microorganisms because of the aspiration pneumonitis and bacterial liver inflammation, as explained above.

9) Dr. Ross examined Jordan's gastrointestinal tract, liver, and gallbladder grossly and microscopically and observed fresh bleedings in liver, wall of gallbladder, multiple areas of the mesentery, pericolonic region, serosa of small intestine, and ascending colon. He also collected 200 ml of bloody fluid from the abdominal cavity. The bleedings in these sites resulted from the synergistic actions of several factors: (a) bacterial infections; (b) probable thrombocytopenia due to sepsis and metabolic problem; (c) liver inflammation, which causes deficiency of clotting factors as explained in Section 3; (d) severe damage of liver, gastrointestinal tract, and other internal organs has been reported in people receiving cardiopulmonary resuscitation CPR.

### 5. Analysis of Bryant's alleged confession that he killed Jordan: did Bryant really confess?

Detective Larry Mathias from the Manheim Township Police Department interviewed Bryant Arroyo in Lancaster General Hospital at 0720 on 25 September 1994, concerning Jordan's cardiac arrest [3]. Bryant stated that after Jordan's mother returned home at 0200 on 25 September, he went upstairs to check on the baby. He found him unconscious in his crib and not breathing. He carried the baby downstairs running and yelling for Jordan's mother to call 911. Bryant put the baby on a chair and began cardiopulmonary resuscitation (CPR), even though he had no formal training to do CPR. He told Mathias that he loved the baby and never hurt him [3,12].

Furthermore, Detective Raymond E. Solt interviewed Bryant at 0920 on 25 September at Bryant's house, where Jordan suffered cardiac arrest. Bryant also stated to Detective Solt that he found the baby not breathing upstairs, brought him downstairs, and performed CPR to revive him. He loved Jordan and had taken good care of him [12]. In addition, the police interviewed Bryant for the third time after 1030 on 26 September at the Manheim Township Police Department. Bryant consistently repeated the information previously given to the police in the two interviews noted above [3]. Briefly, he found Jordan lifeless in his crib and performed CPR to revive him.

However, Detectives Mathias and Solt alleged that Bryant Arroyo freely confessed to them after 1600 on 26 September that he killed Jordan [11,12,44]. Solt and Mathias claimed that Bryant confessed when they told him that the medical examiner had informed them that Jordan's injuries were inflicted intentionally, and that these injuries were not caused by CPR. Also they told Bryant that they believed he was responsible for causing Jordan's injuries [3].

The Detectives alleged that Bryant told them that he went upstairs to Jordan's room after 0030 on 25 September; that he picked the baby up with his left hand under the baby's arm and started punching; that he then switched the baby over to hold him with his right hand and punched with his left; that he laid the baby down on his older brother's bed and punched him some more. It was alleged that Bryant punched the baby in the chest and stomach area a few times, and that he then put the baby back in his own bed and laid him face down [44].

Bryant claimed that he did not confess to the Detectives that he killed Jordan. He stated that the detectives altered the state-

ment he gave on 26 September describing the crisis and the way he did CPR to rescue the baby [45]. Bryant's alleged confession is an eight-page typed-document produced by Det. Solt with a computer and a word-processing program. There are no copies of Bryant's alleged confession handwritten by Bryant or the detectives to compare with the computer-generated document. Det. Solt discarded all interview notes [45,46,47].

I reviewed Bryant's alleged confession and the medical evidence, as well as the testimonies of the police officers, medical examiner, and other witnesses. I found that Bryant's alleged confession, described above, does not dovetail with the medical evidence and the sequence of events that led to the Jordan's cardiac arrest and death. Below is a list of medical facts that raises doubt about the claim of Detectives Methias and Solt that Bryant confessed to them that he killed Jordan.

1) Jordan's weight on 25 September was about 15 lb. The detectives alleged that Bryant picked Jordan up by one arm with a single hand and, using his other hand, punched the baby with a strong force in the chest and the abdomen. I do not believe it is possible to manhandle a healthy 15 lb baby in the manner described by the police, not to mention a baby with the bleeding problems Jordan had, without leaving bruises and marks. Jordan had serious neurological problems, aspiration pneumonia, inflammation of liver and other organs, sepsis, and thymus atrophy. Babies with these health problems also have thrombocytopenia and bleeding problems (see Section 3).

The paramedics, emergency physician, and the medical examiner did not see any evidence of trauma on Jordan's upper and lower extremities during their examinations on 25 September. The medical examiner reported that Jordan's upper arms, forearms, and hands were symmetrically developed and free of evidence of trauma, edema, and other significant natural diseases. In addition, prior to autopsy, full body x-rays were performed on Jordan and show no evidence of acute or healing trauma. The x-rays were also reviewed by Dr. Boal, a pediatric radiologist, who saw no significant abnormalities.

2) It was alleged that Bryant punched Jordan repeatedly in the upper abdomen. Repeatedly punching a baby that has bleeding problems with a strong force, as described in Bryant's alleged confession and by the medical examiner will certainly causes serious bruises. Dr. Ross examined the H & E stained tissue section from the marks on the skin in the abdominal area and observed only congested blood vessels with no obvious hemorrhage or inflammatory reactions. These findings do not support the allegations that the baby was hit with a strong force.

3) The size of the bruises on the lower chest area grew by 400% from the time the paramedics picked Jordan up from his house at 0305 until the time of autopsy at 0800 (Section 3). These data and the evidence described in Section 3 suggest that these minor bruises (1/8 inch) were the effects of CPR.

4) Jordan had spongiosis of the cerebrum and Purkinje cell dropout in the cerebellar cortex; aspiration pneumonia; thymus atrophy; inflammation of the liver, gallbladder, and mesentery; and weight loss. Babies with these health problems have thrombocytopenia and bleedings (Section 3). These are the factual

causes of Jordan's cardiac arrest and bleedings, not blunt trauma as alleged by the medical examiner and police.

5) Bryant's alleged confession about the manner of Jordan's death dovetail only with the medical examiner's theory that Jordan was killed by repeated blows to the chest and upper abdomen. The medical examiner's theory is not supported by the medical facts, and the medical examiner shared his theory with the police on 25 September before any microscopic examination had been done.

6) Bryant was interviewed three times by the police prior to 1600 on 26 September, and on all these occasions he stated that he did not hurt Jordan. He found him unconscious in his crib and performed CPR to revive the baby. Bryant's story was consistent on those three occasions, and it dovetails with the medical facts, whereas the allegation by police does not.

7) The police alleged that Bryant confessed to them that he had an anger problem and that he killed Jordan to let his anger out. They alleged that Bryant was angry with his cousin on 24 September. Bryant, his cousin, Jordan's mother (who had lived with Bryant for about four months), and seventeen other witnesses who knew Bryant well, contradicted the allegation that Bryant had an anger problem. These witnesses wrote affidavits on Bryant's behalf to state that Bryant is very calm person and they never saw him getting angry.

The medical evidence described above also clearly contradicts the statements made by Detectives Mathias and Solt that Bryant confessed to them that he killed Jordan and suffered from an anger problem. The medical data also indicate that Bryant did not provide the statements cited in his alleged confession describing the methods and manner of death. I believe that the State of Pennsylvania and the court should re-examine this case in light of these facts and ask Detectives Mathias and Solt to explain their positions and actions.

## 6. Conclusions and recommendations

The medical evidence described in this report clearly shows that the infant Jordan Anthony Shenk died as a result of serious acute and chronic illnesses that led to his cardiac arrest and bleedings on 25 September 1994. He had brain disease (spongiosis of the cerebral cortex and white matter, and focal Purkinje cell dropout in the cerebellum); aspiration pneumonitis; sepsis; inflammation of the liver, gallbladder, and the mesentery; thymus atrophy; and weight loss. These lesions and symptoms have been reported in children with propionic acidemia and other metabolic problems involving branched amino acids. Jordan's symptoms and lesions indicate that he probably suffered from a genetic illness that led to the development of propionic acidemia and his death.

The medical evidence dovetails with Bryant's claim that he found Jordan unconscious and lifeless in his crib and preformed CPR to revive him. It contradicts Dr. Wayne K. Ross's conclusions that Jordan died as a result of blunt trauma to the chest and abdomen and that the manner of death was homicide. It also raises many doubts about the claim of detectives Methias and Solt that Bryant confessed to them that he killed Jordan.

The objectives of Dr. Ross's investigation of this case were to find the cause and manner of death. My investigation of this case and the medical evidence presented in this report clearly show that Dr. Ross did not achieve his objectives as required by law, because (1) he did not follow standard medical procedure when investigating this case and interpreting the medical data; (2) he rushed to judgment by informing the police and the district attorney's office immediately after finishing his autopsy, and prior to doing the microscopic examinations, that Bryant killed Jordan.

Dr. Ross overlooked the following: (1) that Jordan lost about one pound during the six-weeks prior to his cardiac arrest; (2) that the baby had thymus atrophy; (3) that the number and size of the small bruises and marks observed on Jordan's chest and abdomen changed significantly between the time the paramedics picked the baby up from his house and the time of autopsy—the number and size increased by 400% and five, respectively; (4) that Jordan suffered from bacterial infection, because Dr. Ross did not take blood and fluid samples to do bacterial culture; (5) that Jordan had thrombocytopenia and blood clotting problems, because Dr. Ross did not do the proper blood tests.

Furthermore, Dr. Ross microscopically evaluated tissue sections taken from Jordan's organs and lesions. He found serious brain illness (spongiosis of the cerebral cortex and white matter and focal Purkinje cell dropout in the cerebellum) and aspiration pneumonitis. However, he did not take these important discoveries into account in his evaluation of the case. He also did not reveal his findings in court or inform the family that Jordan was suffering from serious illness.

The presence of these significant pathological abnormalities in Jordan's brain and lungs explain the sequence of events that led to Jordan's cardiac arrest, bleedings, and inflammation observed in Jordan's organs. The brain lesions led to neurological malfunction, which caused gastric and esophageal reflex problems, which brought on aspiration pneumonitis. These illnesses led to sepsis; thrombocytopenia; inflammation of the liver and other organs; deficiency of blood clotting factors and bleedings; bleedings and edema of the lungs/asphyxia; and cardiac arrest.

We made several attempts to get the histology slides of Jordan's organs, but Dr. Ross's office and the Coroner's Office at Lancaster County did not give them to us [48,49]. I wanted to examine the histology slides to evaluate the severity of the damage in the brain, lungs, and other organs. Dr. Ross reported that the spongiosis of the cerebrum involved both cortex and white matter [2]. This indicates an advanced stage of this serious disease.

My investigation of this case clearly shows that Bryant was falsely accused of killing Jordan. He was unjustly convicted because the factual causes of illness and death in this case were not revealed to the jury by the medical examiner and the police.

I hope that the government and medical authority in the State of Pennsylvania will evaluate the medical evidence presented in this report and open an investigation to correct the problems and injustice in this case. I believe that Bryant Arroyo should be released from prison immediately and should be compensated for his pain and suffering and time wrongly spent in prison.

In addition, the State of Pennsylvania should inform Jordan's family about the factual causes of his illness and death. The medical evidence clearly shows that he died from a genetically related metabolic problem. The gene involved in propionic acidemia is a recessive gene. Jordan's family should be alerted to test their other children and future children for this genetic defect to prevent tragedies similar to those occurring in this case.

## References

- [1] Jordan Anthony Shenk's medical record obtained from Dr. Bruce Silverstein, 2920 Marieta Avenue Lancaster Pennsylvania 17601.
- [2] Dr. Wayne K. Ross' autopsy report on Jordan Anthony Shenk. [Autopsy No: A94-347 (LC94-265)]. Conestogia View, Lancaster County, PA. September 25, 1994.
- [3] Detective Larry Mathias, Report No 94012979, 9/25/1994. Manheim Township Police Department. Lancaster County, PA.
- [4] Police's interview with Jordan's babysitter on 25 September 1994. Manheim Township Police Department. Lancaster County, PA.
- [5] The paramedics' report on Jordan Anthony Shenk, Lancaster County, PA. 25 September 1994.
- [6] Jordan Anthony Shenk's medical record from Lancaster General Hospital in Lancaster County, PA 25 September 1994.
- [7] Detective Shultz's Report No 94012979. Manheim Township Police Department. Lancaster County, PA. 25 September 1994.
- [8] Officer John Wettlafer's Report No 94012979. Manheim Township Police Department. Lancaster County, PA. 25 September 1994.
- [9] Arrest Warrant Affidavit. Commonwealth of Pennsylvania vs. Bryant Arroyo. Incident No 94012979. Lancaster County, PA. 26 September 1994.
- [10] Report of investigation. Lancaster County District Attorney's Office, Child Abuse Unit No 94-0305, 9/25/2004. Lancaster County, PA.
- [11] Hearing, Commonwealth of Pennsylvania vs. Bryant Arroyo. Hon. David P. Miller, District Justice, October 25, 1994, District 02-1-02.2205 Oregon Pike, Lancaster, Pennsylvania.
- [12] Commonwealth of Pennsylvania vs. Bryant Arroyo. Transcript of Trial Before: Hon. Paul K. Allison (Case # 3154-1994). May 1995, Lancaster county, PA.
- [13] Hamilton RL, Haas RH, Nyhan WL, Powell HC, Grafe MR. Neuropathology of propionic acidemia: a report of two patients with basal ganglia lesions. *J Child Neurol*, 1995; 10(1):25–30.
- [14] Feliz B, Witt DR, Harris BT. Propionic acidemia: a neuropathology case report and review of prior cases. *Arch Pathol Lab Med*, 2003; 127(8):e325–8.
- [15] Ozand PT, Rashed M, Gascon GG, Youssef NG, Harfi H, Rahbeeni Z, Al Garawi S, and Al Aqeel A. Unusual presentations of propionic acidemia. *Brain Dev*, 1994; 16(Suppl):46–57.
- [16] Wolf B, Hsia YE, Seetman L, Gravel R, Harris, DJ, Nyhan WL. Medical Progress. Propionic acidemia: A clinical update. *The Journal of Pediatrics*, 1981; 99(6):835–46.
- [17] Haas RH, Marsden DL, Capistrano-Estrada S, Hamilton R, Grafe MR, Wong W, and Nyhan WL. Acute basal ganglia infarction in propionic acidemia. *J Child Neurol*, 1995; 10(1):18–22.
- [18] Steinman L, Clancy RR, Cann H, Ulrich. The neuropathology of propionic acidemia. *H. Dev Med Child Neurol*, 1983; 25(1):87–94.
- [19] Asconape J, Challa VR, and Angelo JN. Spongy degeneration of the nervous system associated with propionic acidemia. *Acta Neurol Latinoam*, 1981; 27(1-2):91–8.
- [20] Nyhan WL, Bay C, Beyer EW, Mazi M. Neurologic nonmetabolic presentation of propionic acidemia. *Arch Neurol*, 1999; 56(9):1143–7.
- [21] Akman I, Imamoglu S, Demirkol M, Alpay H, Ozek E. Neonatal onset propionic acidemia without acidosis: a case report. *Turk J Pediatr*, 2002; 44(4):339–42.
- [22] Altman PL and Dittmer DS. Growth including reproduction and morphological development. Federation of American Societies for Experimental Biology, USA, 1962.
- [23] Pathophysiology: The Biologic Basis For Disease in Adults and Children. Edited by McCance KL and Hunther SE. Third Edition. Mosby-Year book Inc. New York, 1998.
- [24] Pathology. Second Edition. Edited by Rubin E and Farber JL. JB. Lippincott Company, Philadelphia, 1994.
- [25] Harrison's Principles of Internal Medicine. 15th ed. Editors: Braunwald E, Fauci AS, Kasper DL, Hauser SL, Longo DL, Jameson JL. McGraw-Hill, New York, 2001.
- [26] Schmidt H, and Manegold BC. Foreign body aspiration in children. *Surg Endosc*, 2000; 14(7):644–8.
- [27] Delmonte C, and Capelozzi VL. Morphologic determinants of asphyxia in lungs: a semi quantitative study in forensic autopsies. *Am J Forensic Med Pathol*, 2001; 22(2):139–49.
- [28] Rabl W, Baubin M, Broinger G, and Scheithauer R. Serious complications from active compression-decompression cardiopulmonary resuscitation. *Int J Legal Med*, 1996; 109(2):84–9.
- [29] Sokolove PE, Willis-Shore J, and Panacek EA. Exsanguination due to right ventricular rupture during closed-chest cardiopulmonary resuscitation. *J Emerg Med*, 2002; 23(2):161–4.
- [30] Samet JH, Flinn MS, Balady G, Skinner M. Costochondritis: a morbid complication in a survivor of cardiopulmonary resuscitation. *Am J Med*, 1987; 83(2):362–4.
- [31] Adler SN, Klein RA, Pellicchia C, and Lyon DT. Massive hepatic hemorrhage associated with cardiopulmonary resuscitation. *Arch Intern Med*, 1983; 143(4):813–4.
- [32] Druwe PM, Cools FJ, De Raedt HJ, Bossaert LL. Liver rupture after cardiopulmonary resuscitation in a patient receiving thrombolytic therapy. *Resuscitation*, 1995; 32(3):213–6.
- [33] Reinartz H. Blunt upper abdominal trauma as a complication of cardiac resuscitation. *Anasth Intensivther Notfallmed*, 1989; 24(2):111–4.
- [34] Aguilar JC. Fatal gastric hemorrhage: a complication of cardiorespiratory resuscitation. *J Trauma*, 1984; 21(7):573–5.
- [35] Waldman PJ, Walters BL, Grunau CF. Pancreatic injury associated with interposed abdominal compressions in pediatric cardiopulmonary resuscitation. *Am J Emerg Med*, 1984; 2(6):510–2.
- [36] McGrath RB. Gastroesophageal lacerations. A fatal complication of closed chest cardiopulmonary resuscitation. *Chest*, 1983; 83(3):571–2.
- [37] Hulewicz B. Gastric trauma following cardiopulmonary resuscitation. *Med Sci Law*, 1990; 30(2):149–520.
- [38] Reiger J, Eritscher C, Laubreyter K, Trattinig J, Sterz F, and Grimm G. Gastric rupture—an uncommon complication after successful cardiopulmonary resuscitation: report of two cases. *Resuscitation*, 1997; 35(2):175–8.
- [39] McDonnell PJ, Hutchins GM, Hruban RH, and Brown CG. Hemorrhage from gastric mucosal tears complicating cardiopulmonary resuscitation. *Ann Emerg Med*, 1984; 13(4):230–3.
- [40] Bedell SE, and Fulton EJ. Unexpected findings and complications at autopsy after cardiopulmonary resuscitation (CPR). *Arch Intern Med*, 1986; 46(9):1725–8.
- [41] Hood I, Ryan D and Spitz WU. Resuscitation and petechiae. *Am J Forensic Med Pathol*, 1988; 9(1):35–7.
- [42] Maxeiner H and Winklhofer A. Eyelid petechiae and conjunctival hemorrhage after cardiopulmonary resuscitation. *Arch Kriminol*, 1999; 204(1-2):42–51.
- [43] Kleemann WJ, Wiechern V, Schuck M, and Troger HD. Intrathoracic and subconjunctival petechiae in sudden infant death syndrome (SIDS). *Forensic Sci Int*. 21, 1995; 72(1):49–54.
- [44] Statement of Bryant (NMN) Arroyo, 26 September 1994 Manheim Township Police Department. Lancaster County, PA.
- [45] Bryant Arroyo, CU-1126, Petition filed In the United States District Court for the Eastern District of Pennsylvania. 11 February 2003.
- [46] Hon. Charles B Smith's report and recommendation on 27 January 2002. Bryant Arroyo, Petitioner vs. Supt. Robert D. Shannon and the District Attorney of the County of Lancaster, PA and the Attorney General of the State of Pennsylvania respondents No. 02-566). In the United States District Court for the Eastern District of Pennsylvania.
- [47] Bryant's letter to William K. Suter on 19 January 2004. Re: Bryant Arroyo vs. Commonwealth of Pennsylvania petition for Writ of Certiorari. Submitted to Office of the Clerk, Supreme Court of the United States, Washington, D.C. 20543
- [48] Nancy Garcia's letter to Dr. Wayne K. Ross, Gary Kirchner, and Howard Shaub on 29 October 2004. Requesting histology slides of the autopsy of Jordan Shenk A94-347 (LC94-265). Families United For Justice, Springfield, MA.
- [49] Nancy Garcia's letter to Dr. Mohammed Ali Al-Bayati on 8 December 2004. Telephone contact log with the Lancaster County's medical examiner's office and the Corner's office requesting histology slides of the autopsy of Jordan Shenk A94-347 (LC94-265). Families United For Justice, Springfield, MA.