Histopathological features of Eliza Jane Scovill's and Destiny Jacobo's lungs with analysis of the causes of death in both cases

Prepared by
Mohammed Ali Al-Bayati,
PhD, DABT, DABVT
Toxicologist & Pathologist

Toxi-Health International
150 Bloom Drive
Dixon, CA 95620
Phone: (707) 678-4484
Fax: (707) 678-8505
maalbayati@toxi-health.com

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Summary of the case and findings

Eliza Jane Scovill suffered from cardiac arrest and died following the administration of four doses of amoxicillin (400 mg twice a day) to treat an ear infection. She died in Los Angeles, California on May 16, 2005 at the age of 3.5 years. Eliza Jane is a white female child who did not suffer from unusual or serious acute or chronic health conditions prior to her upper respiratory tract infection on April 30, 2005. Dr. James K. Ribe, a supervising pathologist who oversaw the investigation in Eliza Jane's case, concluded that Eliza Jane suffered from *Pneumocystis carinii* pneumonia and died of AIDS.

I reviewed the medical evidence in Eliza Jane's case and examined five H&E stained sections of her lung microscopically. The histological features of her lungs show no evidence of inflammation and fibrosis (Figure 1). My investigation revealed that Eliza Jane died as a result of allergic reaction to amoxicillin and that she did not die of AIDS. Ribe's allegation that Eliza Jane suffered from *Pneumocystis carinii* pneumonia and died of AIDS is not supported by clinical data and medical facts.

Ribe performed the autopsy and also oversaw the investigation in the death of Destiny Jacobo, a 21-month-old Hispanic female toddler who died suddenly in December of 1995 in Los Angeles, California. Ribe listed the cause of death in Destiny's case as shaken baby syndrome with associated head trauma. He also alleged that there was forcible rectal insertion causing a retrorectal contusion. Destiny's parents were accused of abusing and killing their daughter. Both were convicted and sentenced to life in prison. The father was released on a technicality after five years; the mother remains incarcerated to this day.

I also reviewed the medical evidence in Destiny's case and examined four H & E stained sections of her lung. I found that Destiny suffered from severe acute hemorrhagic pneumonia (Figure 2) and that the bleeding in her case was caused by bacterial infection, septicemia, and vitamin K deficiency. The medical evidence also indicates that Ribe's diagnoses of shaken baby syndrome and sexual abuse made in Destiny's case are not supported by medical facts.

Furthermore, my investigation also revealed that Ribe did not perform the necessary medical tests or conduct a thorough investigation to identify the factual causes of death in

both cases as required professionally and by the law. My observations in Eliza Jane's case include:

1) The examination of five H&E stained lung sections of Eliza Jane's right and left lungs microscopically revealed **no evidence of inflammation or interstitial fibrosis.** The alveolar spaces are free of exudates and the alveolar walls are free of inflammation and fibrosis (Figure 1A and B). These findings contradict Ribe allegations that Eliza Jane died as a result of *Pneumocystis carinii* Pneumonia (PCP) due to Acquired Immunodeficiency Syndrome. Pneumonia is a term that refers to inflammation and consolidation of the pulmonary parenchyma. Furthermore, the PCP lesions usually comprise interstitial infiltrate of plasma cells and lymphocytes; an interstitial fibrosis; diffuse alveolar damage; hyperplasia of type II pneumocytes; and the alveoli are filled with characteristic foamy exudates.

2) It seems that Ribe interpreted the increase in the weight of Eliza Jane's lungs as evidence of inflammation. However, in doing so, he did not consider the accumulation of fluids in Eliza Jane's body cavities and in her heart, liver, and kidneys (Table 1) or the cause(s) of this accumulation. The pleural and peritoneal cavities contained 20 mL and 60 ml of serous fluid, respectively. My investigation revealed that the accumulation of the fluid in Eliza Jane's lungs and other organs and cavities was caused by the allergic reaction to amoxicillin. Prior to receiving amoxicillin on May 14, 2005, Eliza Jane's pediatrician examined her on May 14th and found her lungs were clear. Her lungs were also found to be clear on prior visits with other pediatricians on April 30th and May 5th.

3) Eliza Jane's blood analysis on May 16, 2005 shows that her lymphocyte count was 10,800 (cells/μL) which is higher than the normal range of normal 2000-8000 cells/μL. This indicates that Ribe's allegation Eliza Jane died as a result of an immune deficiency is not medically valid.

My observations in Destiny's case include the followings:

1) I examined four H & E stained sections of Destiny's lungs microscopically and found that she suffered from severe acute hemorrhagic pneumonia (Figure 2 and Table 2). This is a fatal illness, and septicemia causes bleeding in the lungs and other parts of the body. Ribe described Destiny's lungs as dark red and purple, airless and somewhat congested

with minimal edema fluid. The weight of her lungs was 142% of average normal for age but Ribe did not examine Destiny's lungs microscopically to find the nature and the cause of the problems in her lungs.

2) Ribe did not take Destiny's lung fluid to perform cytological and microbiological evaluation. These tests are usually conducted to identify the causes and nature of pneumonia in children as described in section III of this report.

3) Ribe did not take blood samples in Destiny's case to do a bacterial culture and standard hematology and serum tests to check for anemia, thrombocytopenia, infections, and liver and kidney problems. He also did not check for vitamin K deficiency. Prothrombin time (PT) and partial thromboplastin time (PTT) are usually prolonged in individuals suffering from vitamin K deficiency. Vitamin K deficiency leads to bleeding in the subdural region and other locations of the body as observed in Destiny's case.

4) Ribe's allegation of sexual abuse is not supported by medical facts. Ribe examined the retrorectal area in Destiny's case and found two small areas of hemorrhage in the anterior presacral fascia and behind the rectum at the approximately S5 level. He concluded that the bleeding was caused by forcible rectal insertion of an object.

I believe that inserting an object through the anal canal and the rectum of a child by force will cause injury and bleeding. Ribe's examination of Destiny's anal canal, the rectal mucosa and the wall revealed no injury or bleeding. The retrorectal space is one of the pelvic spaces located outside the rectum. I believe it is not medically possible to cause contusion in the reterorectal space with an object inserted by force through the anal canal and the rectum of a child with a clotting problem, without causing damage and bleeding in the anal and the rectal regions.

Medical examiners have a professional and legal responsibility to conduct all the necessary medical tests and to evaluate the medical evidence thoroughly prior to providing their opinions regarding the cause(s) of injury and death in any case. The medical evidence presented in this report and the cited references clearly indicates that Ribe did not meet these obligations in Eliza Jane's and Destiny's cases.

Section I. Histopathological features of Eliza Jane and Destiny Jacobo's lungs

I-A. Eliza Jane's lung shows no evidence of pneumonia

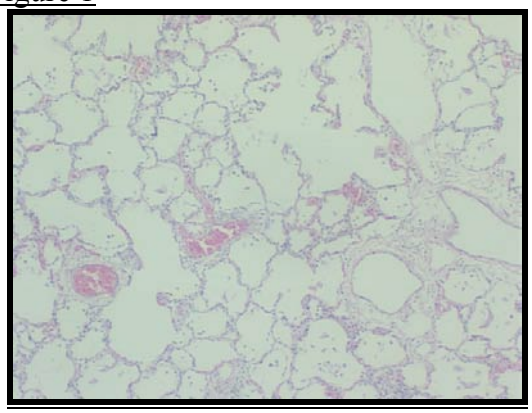
I examined five H&E stained lung sections of Eliza Jane's lungs microscopically and **I see no evidence of inflammation and fibrosis**. The alveolar spaces are free of exudates and the alveolar walls are free of inflammation and fibrosis (Figure 1A and B). These sections (# 05-3767: RML, RLL, RML, LLL, LUL) were obtained from the Los Angeles Medical Examiner's Office and represent three samples from the right lung and two samples from the left lung.

Furthermore, the Los Angeles County's medical examiners (ME's) also examined five similar H&E stained lung sections of Eliza Jane's right and left lungs microscopically and found **no evidence of inflammation or interstitial fibrosis** [1-3]. The ME's finding and my observation clearly indicate that Eliza Jane did not suffer from pneumonia. Pneumonia is a term that refers to inflammation and consolidation of the pulmonary parenchyma [4:566].

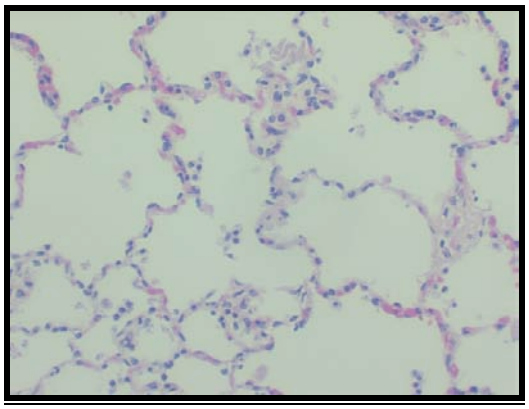
Grossly, the ME's reported that Eliza Jane's lungs were congested and edematous. The weight of Eliza Jane's lungs at autopsy was 306 g, which is about 184% of expected normal weight for age. The ME's also weighted Eliza Jane's heart, liver, and kidneys and the weights of these organs were also significantly higher than the expected normal weights for age (Table 1). Furthermore, the ME's found serous fluid in the pleural cavities (20 mL) and the peritoneal cavity (60 mL) with no evidence of inflammation and fibrosis present in these cavities [1, 2]. These data indicate that the accumulation of fluid in the lungs and other organs and body cavities resulted from cardiac problems and vasodilatation of blood vessels.

Eliza Jane was treated with four doses of amoxicillin (400 mg twice a day) to treat an ear infection during the 30 hours prior to her cardiac arrest and suffered from an allergic reaction to this antibiotic [2]. Histamines and other vasomediators were released from tissues and caused vasodilatation and cardiac problems that led to leakage of fluid from the blood vessels to tissues [4**].** I explained the causes of death in Eliza Jane's case in a report published in Medical Veritas in December of 2005 [2].

Figure 1



A. Photograph of Eliza Jane Scovill's H & E stained lung section shows normal structures of the lung. Note that the alveolar spaces are free of exudates and the alveolar walls are free of inflammation and fibrosis.



B. Photograph of Eliza Jane Scovill's H & E stained lung section at a higher magnification than in photograph A. This magnification shows the alveolar spaces are free of exudates and the alveolar walls and the interstitial tissues are free of inflammation and fibrosis.

Table 1. Significant increases in Eliza Jane's organ weights due to accumulation of fluid

Organs	Weight (g)	Expected average weight (g) for age	% of normal expected weight
Heart	77	59	131
Left lung	138	77	179
Right lung	168	89	189
Both lungs	306	166	184
Liver	500	413	121
Left kidney	75	49	153
Right kidney	67	48	140
Both kidneys	142	97	146

I-B. Destiny suffered from severe acute hemorrhagic pneumonia

The medical examiners at the Los Angeles County Coroner's Office described Destiny's lungs as dark red and purple, airless and somewhat congested with minimal edema fluid. The weight of Destiny's lungs was about 142% of average normal for age. Her lung weight was 190 g and the expected normal weight of lung for age is 134 g [3, 5].

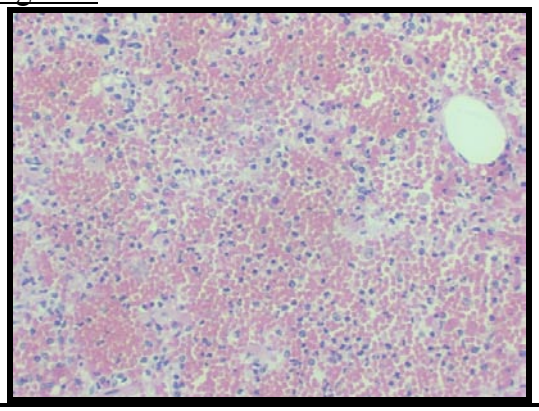
The ME's did not provide any information in their autopsy report about the nature of the microscopic structure of Destiny's lungs [5]. I examined four H & E stained sections of

Destiny's lungs microscopically and found bleeding and inflammation as described in Table 2 and shown in Figures 2A and B. This indicates that Destiny was suffering from severe acute hemorrhagic pneumonia. These sections (05-9550:2, 3, 4, 5) were obtained from the Los Angeles County Medical Examiner's Office.

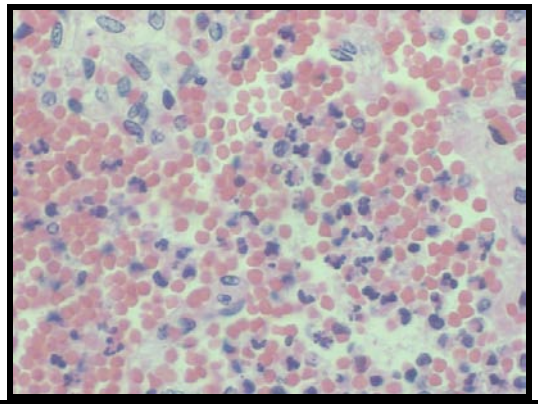
Table 2. Microscopical changes observed in Destiny's lungs and diagnosis

Slide #	**Lesions**	**Diagnosis**
05-9550 (2)	Alveoli contain red blood cells and neutrophils	Acute hemorrhagic pneumonia (diffuse), severe
05-9550 (4)	Alveoli contain red blood cells and neutrophils	Acute hemorrhagic pneumonia (diffuse), severe
05-9550 (5)	Alveoli contain red blood cells and neutrophils	Acute hemorrhagic pneumonia (multifocal), moderate
05-9550 (3)	Alveoli contain red blood cells and neutrophils	Acute hemorrhagic pneumonia (multifocal), mild

Figure 2



A. Photograph of Destiny Jacobo's H & E stained lung section shows the alveolar spaces are filled with red blood cells and inflammatory cells, mostly neutrophils. This indicates that Destiny suffered from an acute diffuse hemorrhagic pneumonia.



B. Photograph of Destiny Jacobo's H & E stained lung section at a higher magnification than in photograph A. This magnification shows the alveolar spaces are filled with intact red blood cells and neutrophils and indicates that Destiny suffered from an acute hemorrhagic pneumonia resulting from bacterial infection.

Section II. The likely causes of Destiny's pneumonia and bleeding

The bleeding and the inflammation observed in Destiny's lungs indicate that she suffered from severe acute hemorrhagic pneumonia (Figure 2A and B, Table 2). The red blood cells were intact and the bleeding probably occurred within a few hours prior to Destiny's death. The presence of neutrophils indicates that the inflammation of the lungs was caused by bacterial infections. The bleeding in the lungs probably resulted due to damage in the blood vessels caused by bacterial toxin and/or vitamin K deficiency.

Bacterial infections with group A Streptococcus, Staphyloccocus aureus, and Haemophilus influenzae have been known to cause pneumonia and bleeding in the lungs of immunodeficient and immunocompetent individuals [6]. Bamba et al. conducted microbial investigations including 141 children with community-acquired pneumonia. Their investigations included examination of postnasal swabs, cultures, polymerase chain reaction (PCR), and serology. Major bacterial pathogens detected in children under 4 years of age were Streptococcus pneumoniae in 34 children, Haemophilus influenzae in 60, Moraxella catarrhalis in 48, and multiple pathogens in 42 **[7].**

Destiny suffered from severe thymic atrophy and immune depression and her susceptibility to infection was increased. Her thymus weight was 6 g, which is about 25% of normal [3, 5]. Below are clinical studies that show bacterial infections of the lung, septicemia, and treatment with antibiotics cause bleeding in the lung and other parts of the body as observed in Destiny's case.

1) Lassalle et al. reported the clinical and histopathological features of four cases of pulmonary bacterial infections associated with extensive necrosis, which were fatal within a few hours. Histological findings included extensive hemorrhagic necrosis with very few or no inflammatory cell components, which was caused by toxins released by the bacteria. The infections were caused by group A Streptococcus (two cases), Staphyloccocus aureus (one case), and Hemophilus influenzae (one case). They stated that these types of infections can occur in immunodeficient and immunocompetent individuals and the diagnosis should be based on histological and microbiological results [6**].**

2) Corrigan conducted coagulation studies on 16 children with clinical signs of gram-negative septicemia (positive blood cultures for meningococcus in 7 cases and Hemophilus influenzae in 6 cases). The children's ages ranged from 4 months to 10 years. These children did not have complications of septic shock, liver disease, malnutrition, or laboratory evidence of classic disseminated intravascular coagulation (DIC). Ten (63%) of the 16 cases were found to have abnormal partial thromboplastin (46 seconds) and/or prothrombin times (20.6 seconds). The coagulopathy was caused by a reduction in the vitamin K-dependent coagulation factors. The evidence suggests that endotoxin may interfere with the vitamin K-carboxylation reaction [8].

3) Neame et al. evaluated the coagulation parameters in 31 individuals with septicemia. They found that 15 of 17 individuals with gram-negative septicemia and 8 of 14 individuals with gram-positive septicemia had thrombocytopenia. Platelet survival studied demonstrated a decreased platelet survival. In 11 of 12 individuals with severe thrombocytopenia (platelet count less than 50,000 per μL of blood), there was laboratory evidence of intravascular coagulation. In contrast, there was little evidence of intravascular coagulation in 8 of 11 individuals with moderate thrombocytopenia (platelet counts 50,000 to less than 150,000 per μL). They suggested that factors other than intravascular thrombin might also play a role in producing the thrombocytopenia of septicemia [9].

4) Levy et al. evaluated some hemostatic parameters in 65 individuals with septicemia. They found that coagulation and circulatory problems are more frequent in septicemia induced by gram-negative bacteria [10].

5) Izquierdo et al. conducted study including 28 infants with infectious gastroenteritis who had disturbances of coagulation. The most frequently seen abnormality was a combination of vitamin K dependent factors deficiency with thrombocytopenia. Some of these infants suffered from hypofibrinogenemia secondary to disseminated intravascular coagulation and lack of synthesis of this factor [11].

6) Kaplan et al. conducted a prospective randomized study included 41children with Hemophilus influenzae Type B meningitis who received moxalactam or ampicillin or chloramphenicol. They found that 6 children had prolonged bleeding times (greater than

6 min), and 7 of 9 tested had abnormal platelet aggregation at hospital admission. At the end of therapy, no children in the ampicillin-chloramphenicol group, compared with 5 of 22 moxalactam-treated children (23%) (P = 0.08), had prolonged bleeding times (6.5 to 7.5 min). They suggested that H. influenzae meningitis and treatment with moxalactam might each have an effect on platelet function in children [12].

7) Corrigan et al. performed coagulation analyses in 36 children (ages ranged from nine days to 12 years, and 48 per cent were one year old or less) who suffered from septicemia including 22 gram-negative and 8 gram-positive infections. Various changes in the clotting mechanism were encountered irrespective of the infectious agents. The most frequent single abnormality was thrombocytopenia in 61 per cent of all cases [13].

8) Sunakawa et al. evaluated infants infected with group B Streptococcus and treated with the antibacterial ceftriaxone (CTRX). Observed adverse reactions included diarrhea and vomiting. An examination for the vitamin K deficiency in 11 cases found a prolongation of prothrombin time (PT) in 3 cases and protein induced by vitamin K absence (PIVKA) II positive in 2 cases [14].

9) Bhat and Deshmukh conducted a prospective non-randomized study on children on antibiotic therapy at a tertiary care hospital. Children in the 1 month-1 year age group developed significant coagulopathy as compared to other age groups. Coagulation abnormalities were also seen to be more frequent in children with greater grades of malnutrition, on a more prolonged course of antibiotics and in children who were critically ill in intensive care [15].

10) Demiroren et al. evaluated the medical records of 19 children (13 male and 6 female) with a diagnosis of intracranial hemorrhage (ICH) due to vitamin K deficiency after the newborn period. The localizations of the ICHs were as follows: parenchymal (47%), subarachnoid (47%), subdural (42%), and intraventricular (26%). Mortality was observed in 6 (32%) infants. All babies were breast-fed, born at term to healthy mothers. Before the onset of the symptoms, four children had used antibiotics for respiratory infection, one child suffered from diarrhea, and one child had a mild hepatic dysfunction [16].

Section III. Medical data that argue against the validity of the medical examiner's opinions and allegations given in Destiny's and Eliza Jane's cases

III-A. Eliza Jane's case

Eliza Jane suffered from cardiac arrest and died following administration of four doses of amoxicillin (400 mg twice a day) to treat an ear infection. She died in Los Angeles, California on May 16, 2005 at the age of 3.5 years. She did not suffer from unusual or serious acute or chronic health conditions prior to her upper respiratory tract infection on April 30, 2005. Dr. Chanikarn Changsri conducted an autopsy and Dr. James K. Ribe oversaw the investigation in Eliza Jane's case (Case No 2005-03767) [1, 2].

These medical examiners (ME's) examined five H&E stained lung sections of Eliza Jane's right and left lungs microscopically and found **no evidence of inflammation or interstitial fibrosis.** However, they alleged that Eliza Jane died as a result of *Pneumocystis carinii* Pneumonia (PCP) due to Acquired Immunodeficiency Syndrome based of the findings of *Pneumocystis carinii* (PC) in her lungs [1, 2].

I examined Eliza Jane's H&E stained lung sections microscopically as described in section I of this report and investigated her case [2]. I found that the ME's methods of investigation and the diagnosis given in this case are medically invalid. Below is a list of medical facts that support my conclusions.

1) My examination and the ME's examination of the H & E stained sections of Eliza Jane's lungs revealed no evidence of inflammation and fibrosis (Section IA, Figure 1 A and B), therefore, the ME's diagnosis of *Pneumocystis carinii* Pneumonia (PCP) is medically unjustified. Pneumonia is a term that refers to inflammation and consolidation of the pulmonary parenchyma [4:566].

   Furthermore, the PCP lesions usually comprise interstitial infiltrate of plasma cells and lymphocytes; an interstitial fibrosis; diffuse alveolar damage; hyperplasia of type II pneumocytes; and the alveoli are filled with characteristic foamy exudates [4, 17]. For example, Chen et al. examined lung biopsies from twenty-three individuals who developed PCP using electron and light microscopes. Their examination showed alveolar exudate, inflammation in interstitium and alveolar space, interstitial fibrosis,

and alveolar epithelial damage in all patients [17]. None of these lesions were observed in Eliza Jane's case.

2) The presence of *Pneumocystis carinii* (PC) alone in the alveoli does not justify the diagnosis of pneumonia, AIDS, or HIV infection. PC was isolated from the lungs of HIV-negative immunocompetent individuals and individuals suffering from immune deficiency resulting from a variety of illnesses and/or treated with immunosuppressant agents. Eliza Jane suffered from viral upper respiratory system infection for about three weeks. She developed anemia and thymic and bone marrow atrophy as a result of her viral infection. Her hemoglobin level and hematocrit value were 6.3 g/dL and 21%, respectively [2]. Below are descriptions of two clinical studies that show PC was isolated from the lungs of HIV-negative individuals who suffered from variety of illnesses.

   a) Contini et al. evaluated the presence of *Pneumocystis carinii* (PC) in the respiratory tract in 36 specimens obtained from 28 HIV-negative immunocompetent children who suffered from chronic lung disorders (CLD). They used a nested polymerase chain reaction (PCR) assay. In addition, Gomori methenamine silver stain (GMS) and indirect immunofluorescence assay (IFA) were performed in parallel. Of the 36 specimens, 12 were PC PCR-positive compared to 10 positive by GMS-IFA. These results suggest an association between PC and exacerbations of CLD in childhood, in the absence of HIV infection or other immunodeficiency syndromes [18].

   b) Takahashi et al. analyzed bronchoalveolar lavage (BAL) specimens obtained from 45 non-HIV immunosuppressed individuals for the presence of *Pneumocystis carinii* (PC) by staining and by PC 5S rDNA determined by PCR. PC was observed by staining of BAL specimens in 20% of these patients. P. C 5S rDNA was also detected by PCR assay in four (8.9%) of these patients for whom staining was negative. None of these patients developed PCP within the follow-up period [19].

3) It seems that the ME's interpreted the increase in Eliza Jane's lung's weight as evidence of inflammation. However, the ME's failed to consider the cause(s) of the accumulation of fluids in Eliza Jane's heart, liver, kidneys and body cavities. The ME's reported that Eliza Jane's pleural cavities contained approximately 20 mL of

clear serous fluid. She also had pericardial effusion and her heart weight was increased by 31% of the expected average normal weight for age. In addition, she suffered from ascites. The ME's found about 60 mL of serous fluid in the peritoneal cavity and this fluid was not caused by inflammation. The peritoneal cavity was without evidence of peritonitis and there were no adhesions. Furthermore, the weights of her kidneys and liver were increased significantly (Table 1).

My investigation revealed that the accumulation of the fluid in Eliza Jane's lungs and other organs and cavities caused by the allergic reaction to amoxicillin. On May 14th, one of her physicians noted redness in addition to fluid in her right eardrum and prescribed amoxicillin (400 mg/twice a day). The physician who examined Eliza Jane on May 14th found her lungs were clear. Her lungs were also found to be clear on prior visits with other pediatricians on April 30th and May 5th [2].

4) Eliza Jane's blood analysis on May 16, 2005 shows that her lymphocyte count was higher than normal (Table 3). This indicates that the ME's diagnosis of AIDS is not medically justified.

Table 3. Eliza Jane's white blood cells count at 0240 on May 16, 2005

Measurements	Values	Normal range
White blood cell count (k/μL)	14.5	5.5-15.5
Neutrophil %	12	45-74
Lymphocyte %	75%*	16-45
Absolute Lymphocyte count (cell/μL)	10,800*	2000-8000
Monocyte %	8	4-10

* Value is higher than the normal range.

III-B. Destiny's case

Destiny Jacobo is a 21-month-old Hispanic female toddler. She died suddenly in December of 1995 in Los Angeles, California. The Los Angeles County Coroner, Dr. James K. Ribe conducted an autopsy on Destiny on December 9, 1995 (Case No 95-09550). He listed the cause of death in his report of February 13, 1996 as shaken baby syndrome with associated head trauma [5]. He also alleged that Destiny was sexually abused [3, 5].

My review of the medical evidence in Destiny's case clearly shows that the conclusions and allegations Ribe made in her case are medically invalid. The following is a list of medical data and studies that support my conclusions:

1) I examined four H & E stained sections of Destiny's lungs microscopically and found that she suffered from severe acute hemorrhagic pneumonia (Figure 2 and Table 2). This is a fatal illness, and septicemia causes bleeding in the lungs and other parts of the body as described in section II of this report. Ribe described Destiny's lungs as dark red and purple airless, somewhat congested with minimal edema fluid. The weight of her lungs was 142% of average normal for age. However, Ribe did not provide information in his autopsy report to show that he examined Destiny's lung microscopically.

2) Ribe did not take fluid from Destiny's lungs to perform cytological and microbiological evaluations. These tests are usually conducted to identify the causes and nature of pneumonia in children. For example, Gaidashev et al**.** performed cytological and microbiological evaluations on bronchoalveolar lavage fluids taken from 472 children who had pulmonary problems. They used the results to define the nature and the causes of lung problems in these children. They found that 125 children were suffering from acute pneumonia and 347 children had chronic nonspecific inflammatory pulmonary diseases [20**].** In addition**,** Lassalle et al. stated that the diagnosis of pneumonia should be based on histological and microbiological evaluation [6].

3) Ribe did not take blood samples in Destiny's case to do bacterial culture. The histopathological evaluation of the lungs indicates that she suffered from severe acute hemorrhagic pneumonia (Figure 2 and Table 2) and that her problem was caused by bacterial infections as explained in section II of this report. Ribe also did not order standard hematology and serum tests to check for anemia, thrombocytopenia, infections, and liver and kidney problems.

4) Furthermore, Ribe did not check for vitamin K deficiency. Prothrombin time (PT) and partial thromboplastin time (PTT) are usually prolonged in individuals suffering from vitamin K deficiency [8, 21**].** Vitamin K has coagulation activity and controls

the formation of coagulation factors II (prothrombin), VII (proconvertin), IX (Christmas factor), and X (Stuart factor) in the liver. Other coagulation factors that depend on vitamin K are protein C, protein S, and protein Z. All of these vitamin K-dependent proteins contain the amino acid γ-carboxyglutamic acid and the carboxyl groups of the glutamic acid residues provide the vitamin-K-dependent proteins with characteristic calcium and phospholipid binding properties [21, 22, 23, 24, 25].

Children who develop vitamin K deficiency usually suffer from bleeding in the subdural space, brain, spinal cord, body cavities, muscles, skin, and other locations as was observed in Destiny's case [23-27]. For example, in a study conducted in Japan, intracranial hemorrhage was observed in 353 cases out of 473 children who suffered from vitamin K deficiency [26]**.**

Additionally, bleeding in the brain was observed in eleven children who developed vitamin K deficiency. The localizations of the intracranial hemorrhage were as follows: intracerebral (91%), subarachnoid (46%), subdural (27%), and intraventricular (27%) [27]. Also, Bor at al. reported bleeding in the brain and other locations of children were found to be suffering from vitamin K deficiency. In nine children, cranial tomography (CT) was taken and showed intraparenchymal, intraventricular, and subarachnoid hemorrhage. In addition, two children had neurologic manifestations and hemorrhagic findings in the cerebrospinal fluid. Skin bleeding **(**ecchymosis) was also observed in three children [28].

5) I find Ribe's allegation of sexual abuse made in Destiny's case is not supported by medical facts. Ribe examined the retrorectal area in Destiny's case and found two small areas of hemorrhage. The first bleeding (1-1/2 inch) was located in the anterior presacral fascia and the second bleeding was observed behind the rectum at the approximately S5 level. He concluded that the bleeding was caused by forcible rectal insertion of an object [5].

I believe that inserting an object through the anal canal and the rectum of a child by force will cause injury and bleeding. Ribe's examination of Destiny's anal canal, the rectal mucosa and the wall revealed no injury or bleeding. He stated that a) no definite surface lesions were seen upon the buttocks; b) careful examination of the anal area revealed that the anus was not dilated and had no tears or bruising; c) the anal mucosa was

unremarkable; d) examination of rectum and the intestinal tract revealed no damage or bleeding in the rectum or any place in the GI tract.

Furthermore, the retrorectal space is one of the pelvic spaces. It is located outside the rectum. I believe that it is not medically possible to cause contusion in the reterorectal space with an object inserted by force through the anal canal and the rectum of a child, without causing damage and bleeding in the anal and the rectal regions. As stated above, these two regions were intact.

In addition, Destiny had a clotting problem and inserting a catheter in the urethra caused significant bleeding. Ribe stated that the vulva was unremarkable except that there was a ¼ inch zone of red mucosal hemorrhage surrounding the superior and lateral margins of the urethral meatus in the association with the catheter. Ribe also observed internal bleeding in other locations of Destiny's body but he did not attempt to explain the cause(s) for the bleeding. He found a 3 inch zone of hemorrhage in the parietal pleura (left of subclavian area). In addition, he observed perivescial hemorrhage on the anterior aspect of the urinary bladder and hemorrhage in the parametria area. It seems that Ribe selected the bleeding in the reterorectal space only to support his allegation of sexual abuse.

Section IV. Conclusions

My review of the medical evidence in Eliza Jane's case clearly indicates that 1) Eliza Jane died as a result of allergic reaction to amoxicillin and she did not die of AIDS; 2) the histological features of her lungs show no evidence of inflammation and fibrosis; 3) James Ribe's allegation that Eliza Jane suffered from *Pneumocystis carinii* pneumonia and died of AIDS is not supported by the clinical data and medical facts.

Furthermore, my review of the medical evidence in Destiny Jacobo's case revealed that she suffered from severe acute hemorrhagic pneumonia and septicemia. The bleeding in her case was caused by septicemia and vitamin K deficiency. It also shows that James Ribe's allegations of shaken baby syndrome and sexual abuse made in Destiny's case are not supported by medical facts.

Medical examiners have a professional and legal responsibility to conduct all the necessary medical tests and to evaluate the medical evidence thoroughly prior to providing their opinions regarding the cause(s) of injury and death in any case. My review of the medical evidence in Eliza Jane's case and Destiny's case shows that Ribe did not perform the necessary medical tests or conduct a thorough investigation to identify the factual causes of death in these cases as required professionally and by the law.

References

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